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# The relationship between chronic hassles, cardiovascular responsivity and physical health symptomatology: an examination of the allostatic model of stress and health

Zachary Clay Wilcox

To the Graduate Council:

I am submitting herewith a dissertation written by Zachary Clay Wilcox entitled "The relationship between chronic hassles, cardiovascular responsivity and physical health symptomatology: an examination of the allostatic model of stress and health." I have examined the final electronic copy of this dissertation for form and content and recommend that it be accepted in partial fulfillment of the requirements for the degree of Doctor of Philosophy, with a major in Psychology.

Kathleen A. Lawler, Major Professor

We have read this dissertation and recommend its acceptance:

Warren Jones, Gary Klukken, Sandra Thomas

Accepted for the Council:

Carolyn R. Hodges

Vice Provost and Dean of the Graduate School

(Original signatures are on file with official student records.)

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# THE RELATIONSHIP BETWEEN CHRONIC HASSLES, CARDIOVASCULAR RESPONSIVITY AND PHYSICAL HEALTH SYMPTOMATOLOGY: AN EXAMINATION OF THE ALLOSTATIC MODEL OF STRESS AND HEALTH

A Dissertation Presented for
the Doctor of Philosophy Degree
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Zachary Clay Wilcox
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# DEDICATION

This dissertation is dedicated to my parents, Charles and Dorothy Wilcox, who have provided me with endless support.

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# ABSTRACT

Homeostasis has been the central model in physiology and therapeutics in modern medicine. Allostasis, a new model, has been put forth as a replacement for homeostasis. According to the allostatic model, health is defined in terms of the ability to respond to demands. Thus, in contrast to the homeostatic model, the allostatic model views responsiveness in a positive light. The allostatic model was examined by observing the relationships between chronic hassles, cardiovascular responsivity to an emotional interview and physical health symptomatology in a sample of 64 female undergraduates. As expected, chronic hassles scores were predictive of health symptomatology. In addition, cardiovascular responsivity variables were more strongly related to health symptomatology than cardiovascular baseline and recovery levels, providing general support for the allostatic model. Examination of the cross between chronic hassles and cardiovascular responsivity suggested that health may be more precisely viewed as appropriate responsivity for the environmental context, not just in terms of responsiveness *per se*.

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# CHAPTER I

# INTRODUCTION

## Homeostasis and the Physiological Concept of Stress

The term homeostasis, coined by Walter Cannon, has been one of the most important and enduring concepts in the scientific study of life processes. Cannon (1932) described homeostasis as the coordinated physiological processes that maintain steady states in organisms. He characterized it as the maintenance of physiological parameters within a range of values, not at a certain set point. For example, he noted that that blood sugar levels normally range between 70 and 180 mg/ml, which were considered the limits of the homeostatically defended range (Cannon, 1932). Cannon chose a combination of Greek root words to describe the physiological processes of homeostasis. "Homeo" means "like" or "similar" and admits some variation (Cannon, 1929/1973), and "stasis" means "state of standing." (Webster's New Universal Unabridged Dictionary, 1996). He emphasized that the condition of homeostasis is *relatively* constant, yet varies. In fact, Cannon explicitly warned that homeostasis does not imply something set and immobile. Moreover, he posited the existence of dynamic, self-regulating mechanisms (i.e., negative feedback) that serve to maintain equilibrium and acknowledged that the key to balance is not constancy, but flexibility (Cannon, 1932).

The concept of homeostasis has become so dominant that *all* physiological processes are referred to as homeostatic mechanisms since it can always be argued that a reaction is protecting the internal environment or ultimately bringing the parameters back to "normal" levels. However, it has also been recognized that excessive homeostatic responses can lead to chronic pathological states (e.g. hyper-immune disorders) and that

homeostatic processes do not always prevail (Richards, 1953). According to Richards (1960), modern physiology and medicine have focused too much on the purportedly, homeostatic "wisdom of the body" (See Cannon, 1932). Therefore, Richards (1960) proffered some additional terms, of ancient Greek origin, which were intended to balance out the importance of homeostatic processes in understanding health and disease. For example, the term "hyperexis" was suggested to refer to perturbations and uncontrollable changes in physiological processess that lead to disease. "Ellepsis" was suggested as a description of inadequate bodily responses that makes one more susceptible to disease. Additionally, "akairia" was submitted to refer to a general category of responses that are ill-timed or misguided. Richards (1960) also made it clear that "good" (i.e., homeostatic) and "bad" responses are found in both healthy and diseased individuals.

Homeostasis has been treated as an indispensable concept in understanding physiological processes like stress responses, which inherently disrupt steady states. According to Chrousos & Gold, 1992), our contemporary understanding of homeostasis and its inextricable link with the stress response has evolved over at least the past 2 ½ millennia. In the early history of medicine, Hippocrates considered health to be a harmonious balance and disease a disharmony of the elements of the body (Chrousos & Gold, 1992). Modern treatment of the concept of homeostasis is usually traced back to the notion of "milieu interieur" or "fluid matrix" of the body, introduced by Claude Bernard in the mid-1800s. Bernard made a distinction between a general environment, which surrounds the organism as a whole, and a "milieu interieu" (or internal environment) in which the living elements of complex animals find their optimal habitat. He believed in the radical teleological notion that all physiological mechanisms have only

one objective, that of preserving constancy in the internal environment (Bernard, 1878). Bernard was not only interested in understanding the internal milieu; he was equally intrigued by the reactivity between the internal and external environment. He viewed sickness and death as a dislocation or perturbation in the mechanisms that maintain fixity in the "milieu interieu" (Bernard, 1865/1949).

Contemporary treatments of the concept of homeostasis are often removed from the views of Cannon and Bernard, over-emphasizing its stagnant nature. For example, Langley (1973) maintained that, "One's body temperature, blood pressure, heart rate, respiratory rate, urine output, body weight, blood composition, total body composition, to name but a few, are static day after day, year after year" (p. 1). Likewise, Guyton and Hall's (1996) widely used Textbook of Medical Physiology, defines homeostasis as: "maintenance of static or constant conditions in the internal environment" (p. 4). Moreover, Bernard's teleological view of homeostasis is reiterated in this same text, "Essentially all of the organs and tissues of the body perform functions that help to maintain these constant conditions" (p.4). These "static or constant conditions" have been viewed as analogous to the set point on a thermostat that is maintained regardless of the ambient temperature. Fortunately, there are current definitions of homeostasis that underscore the involvement of continual, dynamic physiological mechanisms. For example, Taber's Cyclopedic Medical Dictionary (1997) emphasizes the fluctuating nature of homeostasis in defining it as "the state of dynamic equilibrium of the internal environment of the body that is maintained by the ever-changing processes of feedback and regulation in response to external or internal changes." (p. 909)

Physiological views of stress have usually included the concept of homeostasis as integral. For example, Chrousos and Gold (1992) define stress simply as "a state of disharmony, or threatened homeostasis" (p. 1245). Similarly, according to Sapolsky (1992), "A *stressor* can be defined in a narrow, physiological sense as any perturbation in the outside world that disrupts homeostasis, and the *stress-response* is the set of neural and endocrine adaptations that help reestablish homeostasis. By first defining the homeostatic value in an individual, researchers can then measure deviations from these values in response to stressors, and therefore quantify the stress response. Thus, researchers have a simple, convenient way of assessing the degree to which an organism is stressed. It should be noted that Hans Selye (1956), a major pioneer in the scientific study of stress, cautioned against viewing stress as merely a deviation from homeostasis. He opted for the now well-known definition of "the nonspecific response of the body to any demand" (1956, p. 55). By "response" he meant a deviation from homeostasis in certain systems of the body, but felt that an adequate definition of stress should include more than "threatened homeostasis." According to Selye, stress responses can precipitate illness via inherent physiological processes during the stage of resistance or through breakdown of systems during the stage of exhaustion. Illness was not viewed as a response to stressor, but a by-product of the stress response.

There have been few attempts to define stress physiologically without referring to homeostasis. One often-cited description was proposed by Fraser, Ritchie and Fraser (1975): "An animal is said to be in a state of stress if it is required to make abnormal or extreme adjustments in its physiology or behavior in order to cope with adverse aspects of its environment and management" (p.659). The authors admitted that there is

ambiguity in using such imprecise words as "abnormal" and "extreme" but deemed their formulation a significant improvement for animal welfare issues. Unfortunately, there are no straightforward ways of quantifying the stress response using this description, and thus it lacks the quantitative, scientific utility of homeostatic approaches.

## Allostasis: A Model for Understanding Stress and Health

Sterling and Eyer (1988) asserted that the concept of homeostasis has been useful in understanding animal physiology, particularly for describing cellular and subcellular processes. However, they proposed that the homeostatic model has not been as helpful for understanding the internal environments of intact, unanesthetized organisms. For example, with regard to a physiological parameter like blood pressure, there simply is no "normal," homeostatic value toward which automated mechanisms work to maintain pressure. They observed that organisms must occupy various behavioral (which covary with physiological) states in order to meet their demands, and that physiological levels are reset at each behavioral transition. Sterling and Eyer (1988) coined the term allostasis, which was described as "stability through change", to refer to the principle that an organism must *vary* all the parameters of its internal milieu and match them appropriately to environmental demands in order to maintain stability. (p. 636) "Allo" is of ancient Greek origin and means "other." (Webster's New Universal Unabridged Dictionary, 1996). The most straightforward interpretation of the description of allostasis provided by Sterling and Eyer (1988) is that an individual's overall integrity is maintained when challenges are easily met due to adequate variability in physiological systems.

Sterling and Eyer intended that allostasis supplant homeostasis as a central model in physiology and therapeutics. Indeed, allostasis may qualify as a new paradigm in science if it "attracts an enduring group of adherents away from competing modes of scientific activity", and it is "sufficiently open-ended to leave all sorts of problems for the redefined group of practitioners to resolve" (Kuhn, 1962, p. 10). Sterling and Eyer (1988) characterize allostasis as more complex than homeostasis in that it incorporates the activity of the entire body, with a particular recognition of the influence of the central nervous system, not just isolated organs and tissues and local feedback systems. In addition, it has several key advantages over homeostasis. It not only permits the fine matching of resources to needs but is also designed for anticipating altered need and achieving the necessary adjustments preceding a demand. Indeed, allostasis implies an active, dynamic organism that is constantly "in flux", whereas the homeostatic model views fluctuation in a physiological parameter as a potential problem.

According to the homeostatic model of stress and health, once a homeostatic level is breached, the "stabilizing factors of the organism" are "strained" to the breaking point (Cannon, 1935). In this model, it is clear that a wide range of variability is seen as potentially deleterious to the individual. In contrast, lack of fluctuation is seen as a problem in the allostatic model. For example, using a homeostatic model of medicine, treatment (usually pharmacological) is administered to change these "inappropriate" parameters. Unfortunately, according to Sterling and Eyer, (1988), the treatment may have the negative consequences of iatrogenic effects and polypharmacy. Drug treatment may reduce blood pressure, but by reducing blood pressure, other body systems are affected. In contrast, the allostatic model of health views the capacity to respond

behaviorally and physiologically to a variety of demands as the definition of health. For example, acutely elevated blood pressure may indeed be an appropriate response to the arousing conditions. However, chronically elevated levels of a physiological parameter like blood pressure leave little or no margin for responding to other demands, and may generate pathology within the system. According to the allostatic model, chronically elevated blood pressure would entail reducing physiological arousal by changing environmental demands at behavioral and societal levels (Sterling & Eyer, 1988).

Since the publication by Sterling and Eyer (1988), there has been little development in the application of allostasis in health research. Recently, however, a new-found interest in the allostatic model has emerged. If allostasis is to replace homeostasis as a central model in medicine and physiology, it must be shown that it improves our understanding of how the functioning of the body is related to health and disease. Indeed, there is current need for empirical validation of the allostatic model of health.

# CHAPTER II

# LITERATURE REVIEW

## Stress and Physical Health Symptomatology

There is a widely accepted belief in our culture that stress influences our susceptibility to disease. In addition, stress is also thought to be related to the course of recovery from disease, regardless of the etiology of the illness (Rabkin & Struening, 1976). According to Herbert & Cohen (1994), there are both direct and indirect pathways through which stress could precipitate physical illnesses. Direct influences include the physiological impact of bodily systems such as nervous, endocrine, immune and cardiovascular. Indirect influences of stress could involve changes in health habits such as diet, sleep and drug use, which are known to be associated with physical health.

Scientific investigations of the association between stress and health, beginning in the late 1950s and early 1960s, have provided some empirical support for their relationship (Brannon & Feist, 2000). Much of the early work was conducted using the well-known Social Readjustment Rating Scale (SRRS) by Holmes and Rahe (1967) as the measure of stress. Development of the scale was based on the assumption that life event changes, regardless of whether or not they are emotionally positive or negative, are the key ingredient in determining whether or not people experience stress. Holmes and Rahe began by noting which life events often preceded the onset of illness in a sample of 5,000 patients, and found 43 such events. The events were then weighted by an independent group of people with regard to the degree to which each event requires readjustment (Holmes & Masuda, 1974). There has been a continual debate about the

number and nature of items that should be included in life event scales. The scales have been criticized for not including any assessment of an event's subjective appraisal (Brannon & Feist, 2000).

Therefore, an approach to assessing the perceived severity as well as frequency of chronic stressors, called "daily hassles", was developed by Richard Lazarus and his associates. Lazarus (1984) defined daily hassles as "experiences and conditions of daily living that have been appraised as salient and harmful or threatening to the endorser's well-being" (p. 376). Kanner, Coyne, Schaefer and Lazarus (1981) developed the original Hassles Scale. A revision, called the Hassles and Uplifts Scale (HUS) was published by DeLongis, Folkman & Lazarus (1988) and, in addition to hassles, it included events that were perceived as "good." Research with the HUS revealed that perceived severity of hassles was a better predictor of the frequency and intensity of headaches than the SRRS (Fernandez & Sheffield, 1996).

Kohn, Lafreniere and Gurevich (1990) developed a hassles scale specifically for college students called the Inventory of College Students' Recent Life Experience (ICSRLE). Research was conducted in order to examine the relationship between the ICSRLE and minor physical ailments using 211 undergraduates. The results were consistent with previous research, as greater scores on the ICSRLE were significantly predictive of greater likelihood of minor physical ailments ($p < .001$), again supporting the notion that chronic stress is associated with physical health symptomatology (Kohn, Lafreniere and Gurevich, 1991).

These cross-sectional, retrospective reports are fraught with methodological concerns. For example, it is possible that individual differences in levels of stress and

physical symptoms are a reflection of a person's degree of somatization (tendency to endorse many symptoms). In addition, there is the problem of unreliability in retrospective self-reports, especially those of longer than three months (Whitehead, 1994). In addition, the term "retrospective contamination" has been used to refer to the act of exaggerating past events in order to justify the onset of illnesses (Rabkin & Struening, 1976). In other words, individuals who have experienced more illness may report greater life event stress due to a belief that stress causes physical disease.

However, prospective studies have also found statistically significant associations between life event change and physical illness (Rabkin and Struening, 1976). In perhaps one of the most convincing studies to date of the impact of stress on physical illness, Cohen, Tyrrell and Smith (1991) administered psychological stress questionnaires to 420 healthy volunteers prior to being nasally exposed to one of five viruses known to cause the common cold. Control participants received saline drops without a virus. After one week of quarantine participants were classified into one of three categories: not infected, infected but not ill, or infected and ill (clinical cold). No participants in the control group developed clinical colds. However, approximately one third of individuals in the experimental group developed clinical colds. For all three stress measures employed in the study (life events, perceived stress and negative affect) individuals reporting stress scores above the median were significantly more likely to develop clinical colds than participants reporting stress scores below the median. These relationships were found for all five viruses and could not be explained by stress-induced differences in health practices such as smoking, alcohol consumption, exercise, diet or sleep habits.

Even though there is mounting empirical evidence of a relationship between chronic stress and physical health, there still are not strong, conclusive data. Indeed, as Cohen and Manuck (1995) asserted, "convincing evidence that stress contributes to the pathophysiology of human disease is sparse, and, even where evidence exists, relatively small proportions of variance are explained" (p. 423). It is argued in this paper that a stronger effect for stress on physical health could be obtained by incorporating allostatic system (e.g., cardiovascular) responsivity indices in models of health prediction.

## Allostatic Load: Explaining the Consequences of Chronic Stress

The shift from a homeostatic to an allostatic model of health will involve alterations in the way research is conducted, and these changes are now being developed. Bruce McEwen has been the leader in the application of the construct of allostasis to research on stress and health. McEwen and Stellar (1993) extended the concept of allostasis over time in order to make it a useful research construct by proposing the term "allostatic load." The term was described as "the cost of chronic exposure to fluctuating or heightened neural or neuroendocrine response resulting from repeated or chronic environmental challenge that an individual reacts to as being particularly stressful" (p. 2093). Since the word "cost" is used, the concept of allostatic load seems to refer to negative health consequences, not just changes in the body associated with stress. However, McEwen (1998b) also asserts that allostatic load *can* result in disease over long periods of time, implying that allostatic load *per se* is not a disease state. McEwen (1998b) attempted to define the concept more specifically: "Allostatic load is the wear and tear on the body and brain resulting from chronic overactivity or inactivity of

physiological systems that are normally involved in adaptation to environmental challenge" (p. 37).

McEwen (1998a) particularly emphasizes the importance of being able to turn on and turn off allostatic system responses to a stressor. In fact, according to McEwen (1998a), "The core of the body's response to a challenge...is twofold, turning on an allostatic response that initiates a complex adaptive pathway, and then shutting off this response when the threat is past"(p.172). McEwen (1998a) asserts that the sympathetic nervous system and the HPA axis are involved in the most common allostatic responses to challenges. Activation of both systems results in an increased release of stress hormones, which can have damaging effects over a long time period. McEwen (1998b) specifies three stress-related situations that can have pathophysiological consequences: 1) frequent stress; 2) failed shut down (i.e., recovery) of allostatic response to a demand; and 3) inadequate allostatic response to a demand, which leads other systems to overreact.

Using the concept of allostatic load, one would predict that individuals exhibiting physiological hypo-reactivity and/or prolonged recovery are at increased risk for physical illnesses. In addition, individuals who have experienced repeated allostatic system activation (e.g., chronic stress) would also be at increased risk for health problems, regardless of their reactivity or recovery abilities. Interestingly, a similar view of physiological responsivity and health was put forth by Dienstbier (1989), although he did not connect his proposal to the notion of allostasis. He suggested that there is little evidence for viewing general, peripheral, physiological arousal in a negative way, as has often been done since the pioneering works of Cannon and Selye. Dienstbier (1989) also

suggested that in individuals who are best at tolerating stress, "SNS-adrenal onset is fast and strong...arousal decline is fast with stressor offset; and across repeated similar episodes, arousal levels...decline more quickly than in less fit controls" (p. 88). Sapolsky (1992) shares a similar view in summarizing the two most important features characteristics of stress physiology:

> (1) if an organism cannot appropriately initiate a stress-response during an acute physical stressor, the consequences can be extremely deleterious; (2) if an organism cannot appropriately terminate a stress-response at the end of stress, or if it activates the system too much because of repeated or chronic stressors, numerous stress-related diseases can emerge. (p. 6)

The first published study using the concept of allostatic load was reported by Seeman, Burton, Singer, Rowe, Horwitz and McEwen (1997). The researchers presented an operational definition of allostatic load using a multi-systems view. They examined a range of ten regulatory physiological parameters including systolic and diastolic blood pressure, waist-hip ratio, serum high-density lipoprotein (HDL) levels, total cholesterol levels, blood plasma levels of total glycosylated hemoglobin, serum dehydroepiandrosterone sulfate (DHEA-S), 12-hour urinary cortisol excretion, 12-hour urinary norepinephrine and epinephrine excretion levels. It was reasoned that these parameters reflect the activity of regulatory systems that produce wear and tear on the body. Cutoff points (based on being in the highest quartile) were determined for each physiological parameter. Allostatic load was operationally defined as the frequency with which individuals were classified in the highest-risk quartile. The highest quartile was used for each parameter except HDL cholesterol and DHEA-S since greater risk is

associated with low levels of these parameters. In initially high-functioning older (age range: 70 – 79) men and women, higher allostatic load scores were significantly associated with poorer performance on cognitive tests of memory, spatial ability and abstract reasoning. Moreover, higher allostatic load scores were related to significant decreases in memory performance at 2.5 years follow-up assessment. Greater allostatic load was also associated with poorer baseline physical performance, as assessed by five tasks involving timed measures of balance, gait, chair stands, foot taps, and manual ability. Additionally, higher allostatic load was related to greater decline in physical performance at 2.5 years follow-up. Comprehensive allostatic load scores were also predictive of incident cardiovascular disease, whereas individual parameters and various other parameter combinations were not.

The researchers concluded that their initial operational definition of allostatic load was quite successful in predicting health outcomes, but admitted that there are several potential limitations with their formulation. They critiqued it for not including any immune system parameters, which should be closely linked to disease end points. In addition, they used physiological "snapshots", which do not capture an individual's typical profile of activity, and certainly do not reflect dynamic variability in physiological systems (Seeman, et al, 1997). In other words, staying closer to the original concept of allostasis by Sterling and Eyer (1988), it is important to examine an individual's ability to respond to challenges. The health of an individual is best assessed by examining patterns of responsivity in a range of physiological systems; it is viewed as "unhealthy" if systems do not exhibit an adequate magnitude of response, or if they respond but don't recover in a timely manner (McEwen, 1988b).

A quite different approach to measuring allostatic load was taken by Johnston-Brooks, Lewis, Evans and Whalen (1998). These researchers studied the relationship between household density, an enironmental (overcrowding) stressor, and the number of days missed from school, an index of children's health. They used cardiovascular reactivity as a marker for allostatic load and reasoned that allostatic load should be an explanatory variable linking chronic stress and health. Cardiovascular reactivity (CVR) was assessed by measuring the difference between mean systolic blood pressure, diastolic blood pressure and heart rate levels during baseline and mean values during a computer game and an arithmetic test. It is unclear why larger reactivity scores were viewed as an indication of greater allostatic load. Recall that Sterling and Eyer (1988), the originators of the concept of allostasis, view responsiveness as a sign of a healthy system. Thus, adequate cardiovascular reactivity is viewed in a positive light. However, their data showed household density was positively associated with CVR, indicating that greater chronic stress was predictive of increased reactivity. It was also found that CVR was positively associated with number of days ill, which seems to support a homeostatic model of stress and health.

## Cardiovascular Responsivity and the Allostatic Model

One way of assessing the ability of individuals to meet challenges is to examine the magnitude of responsivity in allostatic systems. (In this paper, responsivity refers to reactivity to a demand as well as recovery from it once the stressor has discontinued). According to the allostatic model, individuals exhibiting a relatively wide range of physiological fluctuation while being challenged should be better suited to meet

challenges, and therefore should suffer fewer negative health consequences. By examining the magnitude of responsivity in physiological systems and relating them to health outcomes, the validity of the allostatic model of health can be evaluated.

Cardiovascular Reactivity and Physical Health

There has already been a plethora of research examining cardiovascular responsivity in relation to stressors and personality characteristics (Krantz and Manuck, 1986). The most common approach involves assessing an individual's cardiovascular *reactivity* to laboratory stressors. According to Buell, Alpert & McCrory (1986), "reactivity refers to the physiologic cardiovascular measurable or measurables under investigation in response to a specified stimulus." (p. 128). Manuck, Kasprowicz and Muldoon (1990) suggested a more informative, yet admittedly, much less concise definition: "psychophysiological reactivity refers to a portion of the variability among individuals that is seen on measurement of a physiologic parameter during subjects' exposure to a common behavioral or psychological stimulus (e.g. a psychomotor, cognitive or interpersonal challenge) which cannot be predicted from a knowledge of the variability that exists in that same parameter and among the same individuals in the absence of notable behavioral stimulation (e.g. at rest)." (p. 17)
The authors noted that the value of reactivity assessment is that it provides information that cannot be obtained from resting subjects.

The definition provided by Buell, et al (1986) is more closely linked with the most common approach to measuring cardiovascular reactivity. Researchers have typically quantified cardiovascular reactivity by calculating range or modified range

scores using baseline and task periods. For example, peak heart rate (or an average of heart rates during a peak period) during a stressful challenge is subtracted from the lowest heart rate (or an average of heart rates) during a baseline (rest) period resulting in change (delta) scores. Greater change (delta) scores are obtained and viewed as potentially being associated with greater health risk due to a view that hyperdynamic reactivity is an individual difference that may reflect an underlying diathesis to stress-related diseases (Boyce, et al, 1995). Implicit in the view of cardiovascular reactivity as potentially deleterious is a homeostatic model of stress and health. In contrast, in the allostatic model of stress and health, adequate responsivity is seen as an indication of a healthy system. Potential health problems are predicted to arise when allostatic systems do not respond with a great enough magnitude in order for demands to be met. In addition, problems may arise when there is inadequate responsivity in a particular allostatic system. Other allostatic systems, such as the immune system must compensate, which could put an individual at risk for pathology (McEwen 1998b). Even though there have been few attempts to incorporate the allostatic model in research examining the relationship between cardiovascular reactivity and health, it does not does not preclude one from interpreting previous findings from studies on reactivity and health from the allostatic perspective.

*General Physical Health Symptoms*

There have been few studies examining the relationship between cardiovascular reactivity and general physical health. The paucity of research is probably due to a lack of rationale for predicting such a link, as researchers have considered cardiovascular

reactivity a potential risk factor for cardiovascular disorders, but there has been little theoretical framework for extending the predictive utility of cardiovascular responses to general health symptoms. Using the allostatic model, it now makes sense to explore the link between cardiovascular reactivity and general health, since allostatic system responsivity is viewed as inextricably linked with overall health. From this perspective, it would be predicted that individuals with insufficient cardiovascular reactivity would be at greater risk for general health symptomatology. However, the research findings are mixed.

For example, Rose, Jenkins and Hearst (1978, as cited in Dembroski, MacDougall, Slatts, Eliot & Buell, 1981), in a study of air traffic controllers, found that reduced reactivity levels were associated with increased frequency of illnesses. In a subsequent research, Dembroski, et al (1981) studied 64 male college students who participated in a competitive, laboratory "TV tennis game" while heart rate and blood pressure were monitored. Health records were inspected to determine general health, indexed by dividing the total frequency of illnesses by the number of semesters enrolled in college. Cardiovascular reactivity scores were calculated by subtracting baseline levels from the average values exhibited during the task. In this case, there were significant positive correlations between diastolic blood pressure reactivity and frequency of illness ($r = .32$) as well heart rate reactivity and illness frequency ($r = .25$). Interestingly, baseline values of all cardiovascular parameters were not associated with general health. Participants were divided into four groups based on the amount of cardiovascular reactivity they exhibited. It was revealed that extremely high diastolic

blood pressure and heart rate reactivity responders exhibited significantly more illnesses than their less reactive counterparts.

Goldstein, Trancik, Bensadoun, Boyce and Adler (1999) examined the relationship between heart rate (HR) reactivity, health and socioeconomic status (SES) (as indexed by family income which was treated as a continuous variable), in seventy 3 – 5 year-olds (43 boys; 27 girls). In order to assess HR reactivity, the children underwent a 30-minute protocol involving developmentally challenging tasks while HR and blood pressure were monitored. Parents reported the health data in response to a 6-item, global physical health symptoms questionnaire created by the researchers. Regression analyses revealed an interesting interaction between HR reactivity and SES in predicting externalizing symtpoms, but not global physical health. Unfortunately, the researchers gave no description of what was meant by "externalizing symptoms." For children from high-income families, heightened HR reactivity was significantly related to greater externalizing symptomatology ($p<.01$).

Due to the small number of studies that have been published, it is not currently possible to specify the nature of the relationship between cardiovascular reactivity and global physical health. It is likely that other factors impact the relationship between cardiovascular reactivity and general physical health, such as stress levels and SES, as found by Goldstein et al (1997).

*Heart Disease*

There have been an enormous number of studies driven by the belief that cardiovascular reactivity to stressors may play a specific etiological role in hypertension

and heart disease, and it is often referred to as the "reactivity hypothesis" (Light, Dolan, Davis & Sherwood, 1992). However, despite persistent inquiry into this topic, a recent review of the literature concluded that reactivity *per se* is *not* an established risk factor for cardiovascular disorders (Manuck, 1994). However, there is some empirical support, as a longitudinal study indicated that high heart rate reactivity is predictive of atherosclerotic pathology in monkeys (Manuck, Kaplan & Clarkson, 1983). In addition, cardiovascular reactivity has been found to be significantly predictive of follow-up blood pressure in a longitudinal study on humans (e.g., Light, et al, 1992).

The most often-cited support for the "reactivity hypothesis" is the longitudinal study by Keys, Taylor, Blackburn, Brozek, Anderson and Simonson (1971). These researchers followed 279 men, initially without heart disease, over a twenty-year time period. In order to assess cardiovascular reactivity, a cold pressor task was administered by immersing the participant's hand in ice water for one minute, while blood pressure changes were recorded. Sixty of the 279 men developed heart disease during the follow-up period. Diastolic blood pressure change scores were the best single predictor for cardiovascular disease in the study. In fact, the men classified into the hyper-reactor group showed a relative risk for CHD death or myocardial infarction that 2.4 times that of their less reactive counterparts.

*Respiratory Illness*

Boyce, Chesney, Alkon, Tschann, Adams, Chesterman, Cohen, Kaiser, Folkman and Wara (1995), in a 6-month longitudinal study, examined the relationship between stress levels, CVR and respiratory illness in 137 three to five-year olds. Stress

measurements were computed every two weeks, and CVR testing was conducted within the first four weeks of the study period. Respiratory illness was surveyed weekly. Stress measurement involved an assessment of childcare stressors via a 20-item "daily hassles" type questionnaire filled out by the primary teacher for each child as well as environmental stress ratings based on the perceived quality of the childcare environment. In order to assess cardiovascular reactivity, the researchers developed a 20-minute, 7-task protocol which included an interview of the child, a hidden block construction test, a recall task involving recitation of a series of digits, a gestalt closure task, a social problem-solving task, a blinded object identification task and a verbal description of an emotional event. A "neutral", calming story was read to each child before and after the presentation of the series of tasks. Three measurements of heart rate (HR) and mean arterial pressure (MAP) were obtained before the children began the 7-task protocol and two measurements were taken afterwards. In addition, a HR and MAP measurement were taken at a standard point in the presentation or completion of each of the seven tasks. Respiratory illnesses were assessed and recorded by a pediatric nurse practitioner and a physician who were unaware of the stress and CVR data. A hierarchical multiple regression revealed a significant interaction between environmental stress and MAP reactivity, as indexed by standardized residual scores. Indeed, greater frequency of respiratory illnesses were experienced by children with high MAP reactivity and high numbers of environmental stressors. Interestingly, low MAP reactivity children in high stress conditions experienced no increases in rates of illness. Perhaps most intriguing from the perspective of the allostatic model, the lowest rates of illness were observed in high MAP reactivity children in low stress environments. The findings from this study

support the notion that high cardiovascular responsivity to demands may have a beneficial effect on disease risk unless the individual is repeatedly exposed to these demands (i.e., chronic stress).

Cardiovascular Recovery and Physical Health

Inadequate recovery in allostatic systems, once a demand has been met, may also result in wear and tear on an organism. Indeed, a prolonged response in an allosatic system (e.g., cardiovascular) is one of three types of allostatic load that individuals experience (McEwen, 1998b). Therefore, in order to investigate stress and health from the allostatic model, one must assess recovery in the various physiological systems of the body. Vitaliano, Russo, Paulsen and Bailey (1995) defined recovery as "the physiological processes that follow reactivity—the degree to which one's physiological responses return to prestress levels" (p. 363). Cardiovascular recovery has been measured in a variety of ways and there is not yet a consensus on an appropriate methodology.

The study of the relationship between physical health and cardiovascular recovery from a demand seems to be a fruitful avenue of investigation for understanding how stress is related to physical health. To date, there has been little research on etiologic role of recovery from psychological stressor. However, there is a developing literature suggesting that delayed cardiovascular recovery from stressors is associated with other risk factors for hypertension (Schuler, & O'Brien, 1997). In addition, there is evidence that delayed recovery from exercise stressors is predictive of mortality. Cole, Foody, Blackstone and Lauer (2000) conducted a longitudinal study in which participants

engaged in a treadmill exercise protocol that involved reaching at least 85% to 90% of their age and fitness-predicted maximum heart rate for one minute or until medical contradictions precluded them from continuing. Heart rate and blood pressure were monitored before and after testing. Heart rate recovery scores were obtained by subtracting the value two minutes into the recovery period from the level observed at the peak of exercise testing. Participants were divided into one of two groups based on their heart rate recovery values: normal (43 beats per minute or more) or abnormal heart rate recovery (42 beats per minute or less). Out of the 5,234 participants who met all criteria for inclusion in the study, 325 patients died during the 12-year follow-up period. Abnormal group members were significantly more likely than normal group individuals to die during the study, with a relative risk of 3.20 as compared to 2.58 in the normal group. The results from this study strongly suggest that cardiovascular recovery from a demand is predictive of health outcomes and support the need for further inquiry using various types of challenges.

# CHAPTER III

## RATIONALE AND HYPOTHESES

The allostatic model of stress and health, advocated by Sterling and Eyer (1988), is in need of empirical support if it is to replace the homeostatic paradigm. To date, there have been few attempts to test the allostatic model. Central to the notion of allostasis is the assumption that multi-system, physiological responsivity to demands is the hallmark of health. Hence, physiological indices of responsivity should be more strongly related to health than levels of physiological parameters.

The primary purpose of the present study was to begin development of a comprehensive method for assessing the allostatic model of stress and health. Thus, it was necessary to examine measures of the three stress-related situations that are proposed by the allostatic model to have negative health consequences: a) frequent stress; b) failed shut down (i.e., recovery) of allostatic system to a demand; and c) inadequate allostatic response to a demand, which leads other systems to overreact (McEwen, 1998b). The "demand" used in this study was an interview about an emotionally intense topic. There is ample evidence that such conversations consistently produce responses in cardiovascular parameters (Lynch, 1985). The research hypotheses for this study were as follows:

1) CV responsivity values would be a better predictor of physical health symptomatology than CV baseline and recovery levels.

2) A model which includes measures of CV reactivity, CV recovery and chronic stress/hasses would predict physical health symptomatology better than any of these variables alone.

# CHAPTER IV

# METHODS

## Participants

The data for this research were obtained from a study on the physiology of forgiveness, which included sixty-four female college students who were undergraduates at a large, southeastern U.S. university.

## Apparatus

The laboratory in which data were collected consists of a five-room suite, containing a main room for greeting participants and filling out questionnaires and smaller rooms opening onto the suite, which were used for the experiment. The participant room contains a recliner chair, a desk with TV/VCR, a chair for the interviewer and a second desk for electrodes, supplies and intercom. A wooden lap table was placed across the recliner for writing ease. The equipment room is adjacent and contains the equipment described below as well as tables, chairs and a computer.

Heart rate (HR) and mean arterial blood pressure (MAP) were measured non-invasively with a Critikon Dinamap Vital Signs Monitor, Model SX/P (Johnson & Johnson, Tampa FL 33634). An automated blood pressure cuff was placed on the participants' non-dominant upper arm, and measurement was activated from the adjacent room. At the conclusion of cuff deflation, HR and MAP were printed on a hard-copy output.

## Self–report Measures

### Demographic Information

Each participant was asked to complete a Research Participant Information Sheet. The sheet contains questions pertaining to participant's sex, age, height, weight, race, relationship status, medical conditions, medications, parental history of hypertension, smoking status, use of contraceptives and parental situation.

### Inventory of College Students' Recent Life Experiences (ICSRLE)

The perceived severity of "hassles" over the past month was assessed by the ICSRLE (Kohn, Lafreniere & Gurevich, 1990). The scale lists 49 common, stressful experiences and asks individuals to "Please indicate for each experience how much it has been a part of your life *over the past month.*" Each experience is rated using a 1 – 4 scale. ICSRLE scores are obtained by summing up the responses to all 49 items. The scale was designed to only include items that measure "hassles" *per se* and not items which imply distressed physical and mental reactions to stress. The internal reliability (Cronbach's alpha) of the scale for a sample of 50 male undergraduates was .88, and the reliability for a sample of 156 female undergraduates was .89. In addition, the ICSRLE correlated .67 and .59 with the Perceived Stress Scale, a well-established scale.

Cohen-Hoberman Inventory of Physical Symptoms (CHIPS)

The perceived severity of physical symptoms during the past month was assessed by the CHIPS (Cohen & Hoberman, 1983). The scale lists 33 physical symptoms and asks individuals to indicate "how much, if any, it has been a part of your life *over the past month.* Each statement is rated using a 1 – 4 scale. Scores on the CHIPS are calculated by summing up the responses to all 33-items. The internal reliability (Cronbach's alpha) of the measure was .88 for a sample of 70 college students.

## Procedure

Student volunteers for this study received extra credit in their introductory psychology course. They were signed up for a time and day when they could meet individually in the Health Psychology Laboratory. When participants arrived, they were greeted and given an Informed Consent Form to read and sign. The experiment was described as a study of relationship and bodily responses. After signing the Informed Consent Form, participants generally worked on their questionnaire packet for 10 – 15 minutes, and then completed them at the conclusion of testing. Next, participants were given an Interview Recall Sheet, which informed them that "During the interview, you will be asked to recall a time when you were deeply hurt by someone close to you (close friend, relative, romantic partner). Think of a time now when you were deeply hurt or betrayed by someone close to you. On the following couple of lines, jot down a few words about the incident to remind you during the interview which event you picked to share." The same paragraph followed, with the change of offender to "parents or primary caregivers." After completion of these items, participants were escorted into the

participant room, where transducers for the physiological recording were applied. Next, the blood pressure cuff was applied and a sample reading was taken. In addition, alcohol was used to prepare the skin for heart rate electrodes, which were placed on the abdomen.

After transducer application and assessment for signal clarity, the participant was asked to rest for 10 minutes. During this time, they viewed a relaxing video of tropical fish (Piferi, Kline, Younger & Lawler, 2000) to elicit a baseline level of physiological responding before the interview. The video was accompanied by quiet, instrumental music. The interview was videotaped, and the recorder was turned off during the state assessment periods. Participants were alerted that they could turn off the video camera at any time, or request audiotaping instead of videotaping. After the interview, participants completed their questionnaire packets. After completion of the questionnaires, participants were thanked for their participation in the study.

## Physiological Data Quantification

HR and mean arterial blood pressure (MAP) readings were obtained at minutes 5, 7 and 9 of the rest period. Minute 9 measurements were used as the baseline values since, at this time point, participants were better adjusted to the setting and blood pressure cuff inflation and deflation. MAP is an index of the average of all arterial pressure measured over a period of time. It is determined approximately 60% by diastolic and 40% systolic blood pressure (Guyton & Hall, 1996). During the interviews, blood pressure readings were taken from all participants at minutes 0'15" and 2'15" for all participants. Measurements were also taken at 4'15" and 6'15" if interviews were of longer duration. The length of the interviews varied somewhat from participant to

participant in order to complete discussions, but all were at least three minutes and none were longer than seven minutes. For HR and MAP, reactivity (change) scores were calculated by subtracting minute 9 baseline values from the time point in the interview that exhibited the greatest group mean values. Beginning with participant number 9, HR and MAP readings were also obtained after the interview, during the resting period at 0'15" and 2'15", while the participants were completing questionnaires. HR and MAP recovery scores were calculated by subtracting 0'15" levels from peak interview levels.

## Statistical Analyses

Descriptive data for demographics, CHIPS and the ICSRLE are presented. CV levels during baseline, interview and resting periods are also reported along with CV reactivity and recovery change values. Correlation matrices including CHIPS, ICSRLE, CV levels during rest, and recovery periods as well as peak interview CV level, and CV reactivity and CV recovery change values were constructed. Each CV variable was entered individually, after ICSRLE scores, in hierarchical multiple regressions, in order to examine its unique contribution in predicting the CHIPS. Cross-product terms (ICSRLE x CV variable) were additionally entered in each hierarchical regression analysis in order to assess interaction effects between chronic hassles and CV measures. Lastly, all CV variables exhibiting an additional contribution to ICSRLE scores in predicting the CHIPS were individually entered in a stepwise multiple regression (forward inclusion) in order to examine the predictive utility of combining a combination of CV variables and ICSRLE scores.

# CHAPTER V

## RESULTS

The research sample consisted of sixty-four female undergraduates, with a mean age of 20.2(S.D., 2.9) years, ranging from 18 – 35 years. Fifty-six of the participants were Caucasian, five African American, and three other. Fifty-nine women in the sample were single, with seventeen not involved, and forty-two involved with a romantic partner. There were only four married women and one divorced woman. Only three out of sixty-four women reported any serious health conditions and only six participants reported any serious health conditions in the past. Eighteen individuals reported that they currently smoke cigarettes. There were forty-two women who reported parental history of hypertension, six participants had a parent who had suffered from a heart attack, and two women reported a parental history of stroke. Demographic characteristics of the sample are presented in Table A. (All tables can be found in the appendices)

Descriptive statistics for both HR and MAP, at each time point, are reported in Table B-1. Peak HR during the interview occurred at the first data collection point (0'15") whereas peak interview MAP was exhibited at the second time point (2'15"). Examination of the HR and MAP data revealed that 0'15" values during the recovery period were lower that 2'15" levels. Therefore, 0'15" values were subtracted from peak interview levels for HR (0'15") and MAP (2'15") in order to calculate recovery change scores. HR and MAP reactivity and recovery change values are presented in Table B-2.

In order to examine the interrelationships between chronic hassles (ICSRLE), physical symptomatology and baseline and recovery cardiovascular levels, a correlation matrix was constructed. As expected, a significant positive relationship between chronic

hassles and physical symptomatology was revealed ($r = .41$, $p < .01$). Baseline levels of HR and MAP were not related to physical symptomatology or chronic hassles. However, there was a non-significant, positive association between HR recovery level and symptomatology ($r = .17$, $p = .23$) as well as MAP recovery level and symptomatology ($r = .20$, $p = .14$). In addition, there was a marginal association between chronic hassles and HR recovery level ($r = .22$, $p < .10$). As expected, baseline and recovery HR levels were highly, positively associated ($r = .83$, $p < .01$), as were baseline and recovery MAP levels ($r = .67$, $p < .01$). In addition, HR and MAP values at baseline were significantly related ($r = .44$, $p < .01$), as were HR and MAP measures at recovery ($r = .29$, $p < .05$). The results of the correlation analyses are presented in Table C-1.

A correlation matrix was also constructed to examine the associations between chronic hassles, physical symptomatology and CV responsivity values. Chronic hassles scores were significantly negatively related to HR recovery values ($r = -.34$, $p < .05$), but no other CV responsivity measures. HR reactivity and recovery scores were not related to physical symptomatology. However, peak MAP levels during the interview ($r = .25$, $p < .05$) and MAP reactivity ($r = .29$, $p < .05$) were positively associated with symptomatology, but MAP recovery level showed no such association. A significant positive correlation between HR and MAP was found for peak interview levels ($r = .37$, $p < .01$) but not reactivity or recovery values. As expected, peak HR levels during the interview were highly correlated with HR reactivity ($r = .61$, $p < 01$) and HR recovery ($r = .71$, $p < .01$). HR reactivity was also highly associated with HR recovery ($r = .85$, $p < .01$). Likewise, peak MAP levels were highly related to MAP reactivity ($r = .72$, $p < .01$)

and MAP recovery ($r = .68$, $p < .01$). In addition, MAP reactivity was highly associated with MAP recovery ($r = .75$, $p < .01$). These data are presented in Table C-2.

In order to examine the potential for developing a comprehensive way of assessing the allostatic model, physical symptomatology was predicted using hierarchical multiple regression analyses. Chronic hassles scores were entered in Step 1, followed by an individual CV variable in Step 2, and their cross-product (interaction) term in Step 3. As mentioned previously, chronic hassles scores were predictive of physical symptomatology [Rsq. = .17, Fch. (1, 52) = 12.4, $p < .05$]. First, CV baseline and recovery levels were examined. MAP recovery level added only marginally to chronic hassles scores, accounting for 5% more variance in symptomatology [Rsq. = .22, Fch. (1, 51) = 3.2, $p < .10$]. The regression summary is presented in Table C-3. No other baseline or recovery level CV variables added significantly to chronic hassles scores.

Next, CV responsivity measures were examined using the same procedure. HR recovery values added marginally to chronic hassles, acounting for 5% more variance [Rsq. = .22, Fch. (1, 52) = 3.0, $p < .10$]. Their interaction was not significant [Rsq. = .22, Fch. (1, 51) = .16, $p > .05$]; the regression summary is presented in Table C-4. Peak interview HR and HR reactivity did not significantly add to the predictions of symptomatology. However, Peak MAP interview level added uniquely to chronic hassles, accounting for 6% more variance in symptomatology [Rsq. = .23, Fch.(1, 61) = 5.1, $p < .05$]. Their interaction was also not significant [Rsq. = .23, Fch. (1, 60) = .05, $p > .05$]; Table C-5 presents the regression summary. MAP reactivity also added uniquely to the prediction of physical symptomatology, accounting for 9% more variance [Rsq. = .26, Fch. (1, 61) = 8.0, $p < .05$]. As before, the interaction was not significant, although

3% of variance was added [Rsq. = .29, Fch. (1, 60) = 2.1 p < .17]. The regression summary is presented in Table C-6. MAP recovery change values did not add to the prediction of physical symptomatology.

Lastly, a step-wise multiple regression (forward inclusion) was performed in order to assess the utility of adding a combination of CV predictor variables to chronic hassles scores. CV variables chosen for the analyses were those that had previously accounted for additional variance in symptomatology, when added to chronic hassles. These included MAP recovery levels, HR recovery change values, peak MAP interview levels and MAP reactivity values. As expected from previous analyses, chronic hassles made the strongest contribution to the model [Rsq. = .17, Fsq. (1, 53) = 11, p < .05]. The only CV variable that added significantly to chronic hassles was MAP reactivity, accounting for 9% more variance in symptomatology [Rsq. = .25, Fch.(1, 52) = 5.6, p < .05]. These results are presented in Table C-7.

Since chronic hassles and MAP reactivity were the variables most strongly associated with physical symptomatology, each variable was divided into low, medium and high groups, and a 3 X 3 matrix was constructed in order to gain a better understanding of their individual contributions to physical symptomatology. As shown in Table C-8, the total mean for physical symptomatology was 53.6(12.0). There is a stair-step increase in physical symptoms with each level of stress. The total mean for the high chronic hassles group was 59.6(15.0), whereas low chronic hassles was only 48.4(8.6), and the medium chronic hassles group displayed a mean of 52.5(8.8). For MAP reactivity, as with chronic hassles, the high group exhibited the greatest mean symptomatology scores (58.0, SD = 15.5). However, the medium reactivity group had

the lowest CHIPS scores [medium = 49.7(8.8); low = 53.4(9.8)]. The greatest individual cell value is the cross between high chronic hassles and high MAP reactivity, where a mean of 66.4(19.4) is exhibited. Interestingly, the lowest cell value is between low chronic hassles and medium MAP reactivity, with a mean of 43.7(4.4).

# CHAPTER VI

# DISCUSSION

This study assessed the application of the concept of allostasis to stress research. Allostasis is a model explaining global health that was intended to replace the enduring concept of homeostasis (Sterling & Eyer, 1988). Support for the allostatic model of health was examined by assessing cardiovascular measures before, during, and after an interpersonal demand, which is a particularly relevant and effective stressor for women. From the standpoint of allostasis, health is viewed as a state of physiological and behavioral responsiveness (Sterling & Eyer, 1988). Therefore, it was predicted that CV responsivity values would show a stronger association with symptomatology than baseline and recovery levels of CV measures. The allostatic model, like the homeostatic model, also predicts an associaton between frequency of chronic stress and general health. Thus, chronic hassles scores were included in the model predicting health symptomatology.

These hypotheses were tested with correlational and multiple regression analyses using baseline, interview and recovery CV levels as well as CV reactivity and recovery change values. HR levels or responsivity values were not significantly associated with physical symptomatology. However, an interesting pattern emerged for MAP, responsivity values predicted symptomatology significantly better than baseline and recovery levels. Peak interview MAP measures and MAP reactivity change values were significantly related to symptomatology whereas baseline and recovery levels were not. The strongest model predicting health was an additive one including chronic hassles and MAP reactivity. High stress levels, coupled with an over-reactive CV response to the

recall of emotional stress, were most associated with increased levels of physical symptomatology. Thus, the findings from this study suggest that health is more precisely defined as appropriate responsivity for the environmental context, not just in terms of responsivity *per se*. Indeed, if health is defined strictly as a condition of responsiveness, one would predict greater responsiveness to always be associated with reduced symptomatology. The data from this study suggest the possibility for interactive relationship between chronic stress and physiological responsivity. For example, in Table C-8, the lowest cell value is between medium MAP reactivity and low chronic hassles (i.e., low allostatic system activation), supporting the notion that adequate (i.e., moderate) responsiveness, in the context of low stress levels, is the best scenario in terms of general health. In addition, the cross between high hassles and high MAP reactivity suggests a high degree of responsiveness amplifies the detrimental consequences of high chronic stress. Clearly, the usefulness of combining CV responsivity measures with chronic stress is supported by these data.

A chief limitation of the current study can be understood by recalling that allostasis refers to maintenance of stability by matching physiological parameters to demands (Sterling & Eyer, 1988). Thus, the concept of allostasis was actually put forth as a way of understanding positive well-being, not the detrimental impact of chronic stress. This critical aspect of allostasis has been disregarded in the literature. The current research would have been a more valid examination of allostasis if it had related responsivity to positive health outcomes. There are two likely reasons that positive well-being has not been the focus of research on allostasis. First, pathology remains the focus of health research despite persistent attempts to define health as more than the absence of

disease. Second, Sterling and Eyer (1988) chose a misleading title and focus for their landmark paper: "Allostasis: A New Paradigm to Explain Arousal Pathology." It is difficult to understand why the originators of the concept of allostasis focused on its application to explaining the detrimental consequences of stress. If allostasis had been more clearly presented as a physiological concept that directly relates to positive health, and perhaps indirectly to arousal pathology, subsequent researchers would have more easily incorporated the allostatic model.

The current study is also limited in that it only examined data from healthy, college women, and thus the findings cannot be extended to other populations. In addition, physiological responsivity was done very crudely, simply subtracting CV baseline measures from peak CV interview values. This approach does not lead to reliable "responsivity" assessment. Future studies need to assess responsivity in multi-systems, using many time points, allowing for the examination of reliable patterns of variability. The use of "variability" assessment may be a promising approach to understanding how the allostatic model relates to health. Kleiger, Miller, Bigger and Moss (1987) studied the relationship between heart rate variability HRV and all-cause mortality in post-infarction patients. Twenty-four hour ambulatory ECG recording were made just before the patients were discharged from nine different hospitals. Standard deviations of normal R-R intervals were used as an index of HRV. Of the 808 participants in the study, 127 died during the follow-up period (mean = 30 months). HRV was shown to be significantly independently associated with all-cause mortality. Participants were categorized into HRV groups and it was revealed that 34.4% of individuals with standard deviations of less than 50 died during follow-up. Only 13.8%

of patients with standard deviations between 50 and 100 died, whereas 9% of participants with standard deviations of greater than 100 died during the same time period.

In summary, the current study examined the allostatic model of health by observing the relationship between chronic hassles, CV levels, CV responsivity values and health symptomatology. As predicted by the allostatic model, chronic hassles scores and CV responsivity values were strongly related to physical symptoms. Future research is needed in order to more precisely incorporate the concept of allostasis our understanding of health and disease.

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# APPENDICES

# APPENDIX A

## Demographic Information

Table A. Demographic characteristics, chronic hassles (ICSRLE) scores and physical symptomatology (CHIPS) values for research participants. n = 64.

| Variable | |
|---|---|
| Age (yrs.) | 20.2(2.9) |
| Height (in.) | 64.7(3.4) |
| Weight (lbs.) | 139.7(33.2) |
| Race | |
| Caucasian | 56 |
| African American | 5 |
| Other | 3 |
| Relationship Status | |
| Single and not involved | 17 |
| Single and involved | 42 |
| Married | 4 |
| Separated | 0 |
| Divorced | 1 |
| Widowed | 0 |
| Health Conditions | |
| Medications | |
| Yes | 3 |
| No | 61 |
| Past Serious Illness | |
| Yes | 6 |
| No | 58 |
| Parental History of CVD | |
| Hypertension | 22 |
| Heart Attack | 6 |
| Stroke | 2 |
| Smoke | |
| Yes | 18 |
| No | 46 |
| ICSRLE | 108.6(21.2) |
| CHIPS | 53.6(12.0) |

# APPENDIX B

Descriptive Statistics for Cardiovascular Measures.

Table B-1. Means and standard deviations for cardiovascular measures during baseline(B), interview (I) and resting periods (Q). HR (beats per minute) = heart rate. MAP (mmHg) = mean arterial pressure.

| Variable | B1 | B 2 | B3 | I1 | I2 | Q1 | Q2 |
|---|---|---|---|---|---|---|---|
| HR | 74.8(11.8) | 75.4(11.4) | 74.9(11.6) | 91.4(13.7) | 82.5(12.2) | 78.3(9.9) | 80.2(10.1) |
| MAP | 78.8(7.5) | 77.7(6.8) | 77.3(7.4) | 91.6(11.3) | 92.1(9.9) | 83.4(7.2) | 84.0(8.2) |

n = 64 for B and I variables, n = 55 for Q variables.

Table B-2. Means and standard deviations for cardiovascular reactivity and recovery change scores. HR = heart rate (bpm), MAP = mean arterial pressure (mmHg). n = 64 for Reactivity, n = 55 for Recovery.

| Variable | Reactivity | Recovery |
|---|---|---|
| HR | 16.5(12.3) | 14.5(11.8) |
| MAP | 14.8(8.7) | 8.6(7.2) |

# APPENDIX C

## Correlation Matrices and Regression Summary Tables

Table C-1. Correlation matrix for chronic hassles (ICSRLE), physical symptomatology (CHIPS), heart rate (HR) and mean arterial pressure (MAP) levels.***

| | CHIPS | ICSRLE | HRB | MAPB | HRQL | MAPQL |
|---|---|---|---|---|---|---|
| CHIPS | - | .41** | .03 | .00 | .17 | -.03 |
| ICSRLE | | - | .07 | .07 | .22 | -.06 |
| HRB | | | - | .44** | .83** | .34* |
| MAPB | | | | - | .23 | .67** |
| HRQL | | | | | - | .29* |
| MAPQL | | | | | | - |

*** B = baseline, QL = recovery levels.

n = 64 for B, and n = 55 for QL.

** Correlation is significant at $p < .01$ level (two-tailed).

* Correlation is significant at $p < .05$ level (two-tailed).

Table C-2. Correlation matrix for chronic hassles (ICSRLE), physical symptomatology (CHIPS), heart rate (HR) and mean arterial pressure (MAP) responsivity values.***

| | CHIPS | ICSRLE | HRI | MAPI | HRR | MAPR | HRQ | MAPQ |
|---|---|---|---|---|---|---|---|---|
| CHIPS | - | .41** | .17 | .25* | .03 | .29* | .06 | .07 |
| ICSRLE | | - | -.13 | .00 | -.21 | -.06 | -.34* | -.02 |
| HRI | | | - | .37** | .61** | .72** | .71** | .06 |
| MAPI | | | | - | -.02 | .72** | .12 | .68** |
| HRR | | | | | - | .19 | .85** | .08 |
| MAPR | | | | | | - | .20 | .75** |
| HRQ | | | | | | | - | .19 |
| MAPQ | | | | | | | | - |

*** I = peak interview levels, R = reactivity change values, Q = recovery change values. n = 64 for I and R values, and n = 55 for Q values.

** Correlation is significant at $p < .01$ level (two-tailed).

*Correlation is significant at $p < .05$ level (two-tailed).

Table C-3. Results of hierarchical multiple regression for chronic hassles (ICSRLE), mean arterial pressure recovery level (MAPQL) and their interaction. Criterion variable = physical symptomatology (CHIPS). n = 55.

| Variable | R | Rsq. | Adj.Rsq. | Beta | Fch. | Sig. Fch. |
|---|---|---|---|---|---|---|
| Set 1: ICSRLE | .41 | .17 | .16 | .41 | 11.0 | .002 |
| Set 2: ICSRLE | .47 | .22 | .19 | .42 | 3.2 | .070 |
| MAPQL | | | | .23 | | |
| Set 3: ICSRLE | .47 | .22 | .18 | .86 | .34 | .777 |
| MAPQL | | | | .42 | | |
| ICSRLE X MAPQL | | | | -.47 | | |

Table C-4. Results of hierarchical regression for chronic hassles (ICSRLE), heart rate recovery change values (HRQ) and their interaction. Criterion variable = physical symptomatology (CHIPS).

| Variable | R | Rsq. | Adj.Rsq. | Beta | Fch. | Sig. Fch. |
|---|---|---|---|---|---|---|
| Set 1: ICSRLE | .41 | .17 | .16 | .41 | 11.0 | .002 |
| Set 2: ICSRLE | .47 | .22 | .19 | .49 | 3.0 | .087 |
| HRQ | | | | .23 | | |
| Set 3: ICSRLE | .47 | .22 | .17 | .44 | .16 | .69 |
| HRQ | | | | -.03 | | |
| ICSRLE x HRQ | | | | .25 | | |

Table C-5. Results of hierarchical multiple regression for chronic hassles (ICSRLE), mean arterial pressure peak interview level (MAPI) and their interaction. Criterion variable = physical symptomatology (CHIPS). n = 64.

| Variable | R | Rsq. | Adj.Rsq. | Beta | Fch. | Sig. Fch. |
|---|---|---|---|---|---|---|
| Set 1: ICSRLE | .41 | .17 | .15 | .41 | 12.4 | .001 |
| Set 2: ICSRLE | .48 | .23 | .21 | .41 | 5.1 | .03 |
| MAPI | | | | .25 | | |
| Set 3: ICSRLE | .48 | .23 | .19 | .14 | .05 | .82 |
| MAPI | | | | .10 | | |
| ICSRLE X MAPI | | | | .31 | | |

Table C-6. Results of hierarchical multiple regression for chronic hassles (ICSRLE), mean arterial pressure reactivity (MAPR) and their interaction. Criterion variable = physical symptomatology (CHIPS). n = 64.

| Variable | R | Rsq. | Adj.Rsq. | Beta | Fch. | Sig. Fch. |
|---|---|---|---|---|---|---|
| Set 1: ICSRLE | .41 | .17 | .15 | .41 | 12.4 | .001 |
| Set 2: ICSRLE | .51 | .26 | .24 | .43 | 8.0 | .006 |
| MAPR | | | | .31 | | |
| Set 3: ICSRLE | .54 | .29 | .25 | .18 | 2.1 | .166 |
| MAPR | | | | -.61 | | |
| ICSRLE X MAPR | | | | .95 | | |

Table C-7. Results of stepwise multiple regression for chronic hassles (ICSRLE), mean arterial pressure recovery levels (MAPQL), HR recovery change values HRQ, peak mean arterial pressure peak interview level (MAPI) and arterial pressure reactivity (MAPR). Criterion variable = physical symptomatology (CHIPS). n = 55.

| Variable | R | Rsq. | Adj.Rsq. | Beta | Fch. | Sig. Fch. |
|---|---|---|---|---|---|---|
| Set 1: ICSRLE | .41 | .17 | .16 | .41 | 11.0 | .002 |
| Set 2: ICSRLE | .50 | .25 | .22 | .44 | 5.6 | .022 |
| MAPR | | | | .28 | | |

Table C-8. Physical Symptoms (CHIPS) means and standard deviations for the cross between chronic hassles (ICSRLE) and mean arterial pressure reactivity (MAPR).

| Variable | Low MAPR | Medium MAPR | High MAPR | Total |
|---|---|---|---|---|
| Low ICSRLE | 49.0(6.6) | 43.7(4.4) | 52.4(11.4) | 48.4(8.6) |
| Medium ICSRLE | 57.8(11.0) | 49.6(6.2) | 53.3(10.0) | 52.5(8.8) |
| High ICSRLE | 53.9(10.5) | 60.5(11.5) | 66.4(19.4) | 59.6(15.0) |
| Total | 53.4(9.8) | 49.7(8.8) | 58.0(15.5) | 53.6(12.0) |

# VITA

Zachary C. Wilcox was born in Killeen, Texas on June 16, 1968. He lived in nearby Belton, Texas through the third grade, where he attended public school. He moved to Tucumacari, New Mexico before the beginning of his fourth grade year. Zachary attended public school in Tucumcari through high school and received his diploma in May, 1987. He entered Eastern New Mexico University in fall, 1987, and graduated with a Bachelor of Science degree in May, 1991 with a major in Psychology. In fall, 1992, He enrolled in at the University of Tennessee to pursue a doctoral degree in Psychology. En route to his Ph.D., He completed the Master of Arts degree in August, 1997. Zachary was hired as a psychology instructor at Tennessee Technological University in January, 1999, and has continued being a full-time instructor to the present date. He completed his Doctor of Philosophy degree in Psychology in December, 2000.