Multiple Psychosocial Factors as Predictors in the Progression of HIV-1 to AIDS

Robert Louis Dutile

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Multiple Psychosocial Factors as Predictors in the Progression of HIV-1 to AIDS

by

Robert Louis Dutile

A Dissertation submitted in partial satisfaction of the requirements for the degree of Doctor of Philosophy in Psychology

September 2003
Each person whose signature appears below certifies that this dissertation in their opinion is adequate, in scope and quality, as a dissertation for the degree Doctor of Philosophy.

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ABSTRACT OF THE DISSERTATION

Multiple Psychosocial Factors as Predictors in the Progression of HIV-1 to AIDS

by

Robert Louis Dutile

Doctor of Philosophy, Graduate Program in Psychology
Loma Linda University, September 2003
Dr. Kiti Freier, Chairperson

Nearly 3.1 million individuals have died of AIDS as of December 2002 and more than 38.6 million individuals were identified as being infected with HIV (Joint United Nations Programme on HIV/AIDS (UNAIDS) and World Health Organization (WHO) 2002). Psychoneuroimmunology is a discipline that increases our understanding of the relationships between psychosocial factors, the central nervous system, the immune system, and disease. From this perspective, this study reviews the biopsychosocial medical model and components of the stress-immunity-health (SIH) model. The constructs of stress and social support are discussed and defined referencing current theory and research. In addition, physiological mechanisms of stress and the immune system are discussed to provide the foundations of the SIH model. This study specifically examines six types of perceived social support as buffers of three types stress and their effects on immune functioning in the progression of HIV to AIDS. The sample consisted of 97 males diagnosed with HIV/AIDS receiving comprehensive care at a center for immunology. In general, results indicated that the sample perceived higher than average levels of social support, average levels of stress, and exhibited compromised
immune functioning. Applications to the SIH model can be understood in relation to these positive social indicators.
Introduction

With the pandemic reports from the World Health Organization of HIV and AIDS continuing to rise (Joint UNAIDS and WHO, 2002), the future for treatment may be found in fairly recent paradigm shifts within the medical community with respect to examining psychosocial variables in the treatment of HIV and AIDS. The predominant view of health and illness over the last few centuries has been the biomedical paradigm (Taylor, 1999). From this perspective, the etiologies of illness are viewed as biological abnormalities that can be compared to physical norms, reduced to observable anomalies. Some inadequacies of the biomedical model exist in that biological variables are solely considered as contributing to health (Engel, 1977). Another deficiency arises from solely observing the lower level of processes, such as molecular changes, that eliminate alternative explanations such as psychological and social factors. Further, the model erroneously suggests that the mind and body are not interdependent, highlighting abnormalities and dismissing social and psychological influences.

Engel challenged this traditional paradigm in 1977 by proposing a paradigmatic shift to what is currently known as the biopsychosocial model. From this perspective, the etiology of an illness is understood through the reciprocal triadic determinism between biological, psychological and social variables. “Disease is seen as multifaceted and multi-determined. Not only do the behavior, cognitions, and emotions of the individual interact with and influence the physiological, neurological, and immunological systems of the body, but the individual also interacts with and is affected by interpersonal, social, and environmental contexts” (Todd & Bohart, 1994, p. 315). Major benefits that arise
from this model are that the mind and body are viewed as an interactive entity and multiple factors exhibit multiple effects (Taylor, 1999). This approach to illness allows both the micro- and macro-level of processing to be viewed as influences in health and illness.

From this vantage point, practitioners understand the psychosocial meaning attributed to an illness, individualized response style to illness and desired course of action to an illness. Thus, an illness is no longer viewed as a specific set of symptoms with a generalized approach in treatment. For example, cultural beliefs, cognitions, values, roles and socioeconomic status are considered in developing a treatment plan. The biopsychosocial model also implies that treatment compliance may be enhanced with this line of tailored intervention.

As a result of the growing support for the biopsychosocial model, research is fueling a fairly new subdivision of medicine that is specifically aimed at investigating the etiologies of health and illness from this ecological perspective—psychoneuroimmunology (PNI). This paradigm shift has broadened our understanding of how the interaction of human factors and environmental factors affect health. As with most new paradigms, the shift is slow and requires much hard work. For example, as late as the 1980’s, the New England Journal of Medicine published studies that disputed psychosocial factors as having any influence on the course of illness. The editor's harsh analysis, which were loosely based on methodological problems, also attempted to invalidate mind-body models (Keller, Shiflett, Schleifer, & Bartlett, 1994). Ultimately,
the evidence was undeniable and research began to demonstrate interactions between psychosocial factors and immune functioning (O’Leary, 1990).

For example psychological research on emotion demonstrated that an inpatient population diagnosed with major depression exhibited higher cortisol concentrations, and lower absolute counts of T and B cells within blood samples (Schleifer, Keller, Meyerson, Raskin, Davis, & Stein, 1984). In a follow-up study, major depressive and schizophrenic inpatients were compared in an attempt to control for the hospital environment (Schleifer, Keller, Siris, Davis, & Stein, 1985). Results demonstrated similar findings from the first study in that the major depressive group exhibited lower absolute T cell counts (results did not support differences in B cells). In addition, research investigating social support has illustrated that an inverse relationship exists between social support and cardiovascular disease (House, Robbins, & Metzer, 1982). Similarly, Snydersmith and Cacioppo (1992) found that activation of the sympathetic nervous system in cardiovascular responses was greater during challenging tasks in the presence of strangers, as compared to lower reactivity in the presence of friends. In another study, husbands who exhibited higher levels of hostility and anger exhibited elevations in blood pressure, cortisol levels, natural killer cells and cytotoxicity than their controls who exhibited lower levels anger (Miller, Dopp, Myers, Stevens, & Fahey, 1999).

With regard to health behaviors that decrease the experience of stress, exercise has been a major focus in enhancing and maintaining good mental and physical health (DiMatteo, 1991; Taylor, 1999). Specific attention concerning aerobic exercise
demonstrates its relations with better cardiovascular functioning (Alpert, Field, Goldstein, & Perry, 1990; Dishman, 1982). Similarly, Wright (1988) examined the influence of Type A personality characteristics and found that stress was related to heart lesions, while exercise was related to better cardiovascular functioning. Another health behavior variable that has been widely studied is primary prevention behavior (DiMatteo, 1991; Taylor, 1999). The risk of contracting many illnesses has been reduced through educational programs promoting preventive behaviors. Yet, the application of these efforts appears to be circumvented through mediation factors that involve personal beliefs (Taylor, 1999). For example, adherence to breast self-examination that is geared toward early detection is practiced by few and often incorrectly (Stevens, Hatcher, & Bruce, 1994). Internal beliefs such as low efficacy in technique have been associated with irregular practice and not applying the self-exam at all (Kegeles, 1985). Additionally, early detection and treatment of an illness has been examined through health seeking behavior. Such variables as making appointments, keeping appointments and follow-up care, and compliance have been found to be intimately related to the patient’s perception of care (Taylor, 1999). DiMatteo (1991) provides an overview of Cassell’s work in 1985, in which he formulated four premises involved in promoting physician-patient communication: (1) doctors treat patients, not illness; (2) the body makes the ultimate decision; (3) medical care is provided through the interpersonal relationship between the physician and client; and (4) the verbal communication expressed between the physician and client is the most influential aspect of treatment.
The preceding are just a few examples of how the biopsychosocial field has gained respect and fostered growth in understanding the individual approach to illness. Moreover, this once controversial method of analyses has continued to grow and subdivide, opening the door to research investigating how stress can help produce health or illness and influence the functions of the immune system (Bandura, 1997; Taylor, 1999).

The purpose of this paper is to focus specifically on a stress-immunity-health model (SIH) that examines how various types of stress interact and may lead to neurological changes effecting immunological functions that lead to health outcomes. It has been proposed that stress is an important factor in the etiology of illness behavior and the prognosis over the course of illness, including infectious and chronic diseases (Ader, Felton, & Cohen, 1991; Bandura, 1997; DiMatteo, 1991; Glaser & Kiecolt-Glaser, 1994; Gottlieb, 1997; Locke, Ader, Besedovsky, Hall, Solomon, & Strom, 1985; and Taylor, 1999). For example, subjects were exposed to rhinoviruses via nasal mucosa in a controlled experiment, which resulted in level of stress being correlated with rate of infection and colds (Cohen & Williamson, 1991). In addition, Glaser & Kiecolt-Glaser (1994) provided a comprehensive illustration of research supporting the stress-immunity-health model, relating stress to numerous variables (e.g., HIV, cancer, heart disease, herpes, and endocrine function).

The following sections will examine the various dimensions of the SIH model. The review of literature provide a composite view of stress and social support, and discuss how these variables relate to immune functioning and health. Further, the
benefits of understanding how various types of stress interact and social support affect the immune functioning in an HIV/AIDS population are discussed.
Review of the Literature

Stress Defined

There are varying models of stress that emphasize different processes involved in the experience of stress (Rice, 1999). For example, the diathesis-stress-model does not provide a definition of stress, but believes stress arises from a mismatch between biological endowment and environmental stressors. While this model promotes the interaction between internal and external factors, it does not acknowledge cognitive-social factors in stress. Learning theory defines stress as faulty conditioning of a conditional or reinforcing stimulus that results in an emotional response. Though learning theory clearly defines basic terms and procedures, it is limiting in that it minimizes biological and social-context factors. Control theory defines stress as a disturbance between reference value and comparator value in a feedback loop, resulting from any information that results in disequilibrium in the system. While control theory presents a systemic approach by examining all potential systems that influence stress reactions, its downfall stems from an all-inclusive theory that is challenging to operationalize and test.

Stress often occurs as a result of an individual’s attempt to reconcile circumstances that threaten to obstruct an individual’s physical or psychological functioning. This would include environmental variables and individual physiological events and reactions (DiMatteo, 1991; Keller, Shiflett, Schleifer, & Bartlett, 1994; Gottlieb, 1997). Taylor (1999) further defined the construct of stress as “a negative emotional-experience accompanied by predictable biochemical, physiological, cognitive,
and behavioral changes that are directed either toward altering the stressful event or accommodating to its effects” (p. 168).

DiMatteo (1991) noted that stress research frequently has offered three main conceptualizations of the meaning of stress (external constructs such as stressors, internal constructs such as physiological measures, and outcome constructs such as ecological measures). Similarly, Mason (1975) provided three categorical definitions in which to organize the literature when referring to stress, which include stressors, internal processes, and person-environment-fit. More recently, the literature has increasingly distinguished between acute and chronic stress, suggesting another category that describes the temporal quality of stress.

**Stressors.** Many studies have investigated stress as stimuli, or more commonly referred to as stressors (pressures or strains), that cause tension or anxiety in an individual. Frequently, this definition places stress within the context of the environment (Rice, 1999). Thus, stressors may be viewed as external demands placed on the individual. Stressors can arise from basically any aspect of an individual's life and may include such variables as insufficient funds to pay monthly bills, crowding, noise, and role expectations from others. A variety of stressors have been defined as to how the stressor relates to the family system (McCubbin & Patterson, 1983). This systemic definition suggests that an individual within a family may experience a stressor directly or indirectly such as a family member losing employment, becoming ill, dying, being relocated, or any change in the family system.
Stressors that are more frequently encountered on a daily basis are referred to as hassles in the stress literature. Numerous measures of daily hassles have exhibited predictive abilities in psychological and physical health outcomes (Aldwin, 1994). Hassles scales typically tap into a variety of stressors in a single measure by drawing from a large pool of items such as waiting in lines, traffic, flat tire, or unexpected change in daily routines (Delongis, Folkman, & Lazarus, 1988).

**Internal processes.** While challenges in life have been defined as stressors, the literature has often focused on an individual’s response to stressors in specific situations such as internal feelings, physical and psychological (Coyne & Holroyd, 1982; in DiMatteo 1991). From this perspective stress may be examined as a psychological or cognitive process with outcomes being influenced by an individual’s evaluation of whether or not the necessary resources are available to overcome or accommodate the stressor. For example, those who maintain higher levels of efficacious beliefs will experience less stress, while those who feel less efficacious in being able to meet the demands of a situation will experience more stress (Bandura, 1997). Similarly, emotions and perceptions appear to be central in much of the literature that discusses modifiers of stress (DiMatteo, 1991; Taylor, 1999). For example, individuals who exhibit a pervasive negative affect report higher levels of distress, discomfort and dissatisfaction in varying conditions (Brett, Brief, Burke, George, & Webster, 1990). In contrast, those who have a sense of coherence and view stressors as inevitable, yet not necessarily bad, experience less stress (DiMatteo, 1991). Furthermore, individuals who are committed, view conditions as controllable and challenging, and are characterized as hardy also experience
less stress (DiMatteo, 1991). Additionally, induced positive and negative affect has a specific relationship with the autonomic nervous system. For example, negative affect was associated with the activation of the sympathetic nervous system and positive affect induced homeostasis by activating the parasympathetic nervous system (Futterman, Kemeny, Shapiro, Polonsky, & Fahey, 1992).

*Person-environment fit.* Stress has also been investigated as an outcome between the interaction of an individual and the environment—person-environment fit (Lazarus & Folkman, 1984; Rabin, 1999; Rice, 1999). Accordingly, stress is viewed as a very individualized experience influenced from emotional reactions, various physiological patterns, and personality characteristics (O’Leary, 1990). Clearly, an interaction between internal processes and external events appears to influence the severity of stress experienced by an individual. For instance, van Eck, Berkhof, Nicolson, & Sulon (1996) found that individuals who possessed negative affectivity were more likely to exhibit higher levels of saliva cortisol. Similarly, older adults with a pessimistic explanatory style in their worldview tend to attribute negative circumstances to pervasive internal and stable qualities (Kamen-Siegel, Rodin, Seligman, & Dwyer, 1991). These styles of viewing personal experiences can eventually lead to a generalized interpretation of the world known as catastrophizing, which may lead to a sense of learned helplessness.

In a critical study by Tomaka, Blascovich, Kelsey, and Leitten (1993), three experiments provided support for a stress coping model involving primary and secondary appraisals of stress. Stressful tasks were manipulated either by employing arithmetic tasks that involve active coping, or by showing films of accident victims that involve
passive coping. Subjective measures of stress included whether subjects perceived tasks as challenging or threatening. Physiological measures of stress included cardiac reactivity, skin conductance reactivity, and vascular resistance. Results indicated that cognitive appraisals concerning threatening and challenging situations were able to predict subjective, physiological, and behavioral responses to the stressors. Subjective stress scores during active coping tasks were higher in those who appraised the situation as threatening than those who appraised the tasks as challenging. Physiological measures indicate that the challenge appraisal group exhibited higher cardiac reactivity and lower vascular resistance than those in the threat appraisal group. In addition, those with high primary appraisals during passive coping tasks exhibited higher cardiac and skin conductance reactivity. Overall, Tomaka, Blascovich, Kelsey, and Leitten (1993) provided strong support for a systemic approach to examining the process of stress as it relates to the interaction between psychological, physiological, and environmental variables.

**Temporal quality of stress.** Another classification of stress in the literature falls under a temporal definition in which studies have distinguished between acute and chronic stress, as well as their effects on the human condition (Gottlieb, 1994). Chronic stressors typically involve long-term or recurring life events such as a physical or emotional stress related to an illness, work environment, discord between family or friends, or environmental noise and crowding (Evans, Hucklebridge, & Clow, 2000).

The distinction between acute and chronic stress is less delineated, and an interaction between the two appears to exist. The vague boundaries in definitions appear
to stem from research suggesting that acute stressors may result in experiencing long-
term threat and chronic stress (e.g., loss of a job, parenthood, and natural disaster).
Therefore, it would be beneficial to examine stress comparatively (acute vs. chronic) to
understand the overlap in response and coping. Moreover, hassles in daily living may not
fit into such categories as the traumatic, acute stress or enduring, chronic stress. As a
result, daily frustrations, annoyances, and hassles appear to be in a category by
themselves, suggesting more of an intermittent temporal pattern.

These definitions of stress that have been presented provide an understanding as
to what may qualify as stress and where it may arise. While some interventions attempt to
target specific types of stress, it is difficult to eliminate stress in an individual’s life.
Therefore, it is necessary to further investigate and understand how individuals internally
experience stress and how this process may impact adaptation and health outcomes. The
current study will examine the effects of acute, chronic, and intermittent stress.

**Process of Stress**

Clearly, stress is a phenomenon that occurs in everyday life. Different models
have attempted to link the effects of stress to internal processes. Knowing how the body
responds to various types of stress offers progressive implications for the prevention of
an illness or improving the prognosis. This study discusses the benefits and shortcomings
of General Adaptation Syndrome, Transactional theory, and the Holistic health theory
(Rice, 1999). While each of these models are supported by the literature, they fall short of
being able to provide a complete understanding of the process of stress in that they
address specific components. The Stress-Immunity-Health model is then discussed as
building upon these theoretical foundations and offers the most comprehensive approach in examining the process of stress.

*General adaptation syndrome.* Selye (1956) proposed a biological model of the body’s response to stress that incorporate stages that involve both the sympathetic and parasympathetic nervous system. According to his model, environmental demands are viewed by the individual as being stressful and produce a common internal stress response. His *General Adaptation Syndrome* involves three stages in response to stress. When a stressor is initially encountered, the alarm stage is activated. The individual will experience physiological responses to the stressor that involve increases in adrenal activity, cardiovascular and respiratory functioning (sympathetic nervous system). As time passes, the resistance stage becomes activated. During this second stage, the body attempts to overcome the stressor or at least adjust to the stressor by decreasing the activation of various organisms that were formerly elevated during the alarm stage (parasympathetic nervous system). Finally, the third stage represents conditions in which the individual is unable to terminate or escape the stressor. During the exhaustion stage, Selye found that chronic stress resulted in a hormonal production pattern. An example taken from an animal model showed that rats undergoing chronic stress produced structural changes correlated with physiological responses. The structural changes that Selye noted were enlarged adrenal cortices, atrophied thymus glands and lymphatic structures following exposure to repeated stress (DiMatteo, 1991).

A large body of evidence has evolved that provides support for this General Adaptation Syndrome, and has expanded its pragmatic application (e.g., Frankenhaeuser,
1975; Levi, 1965; Mason, 1975; Theorell, 1974). These findings begin to argue the relevance of stress in immunological functioning. However, Selye’s model of stress is narrow in its scope; it does not account for internal cognitive processes or coping strategies, which currently has much support from the literature in the ability to generally explain the physiological responses to stress (Rice, 1999).

*Transactional theory.* The shortcomings noted in the previous model are addressed by Lazarus and Launier (1978) who developed a cognitive transactional model of stress. This approach does not define stress as an external or internal construct or outcome, but rather, a relational interaction between environmental demands and personal resources (Aldwin, 1994). Basically, the cognitive transactional model proposes that stress is contextually defined and experienced through cognitive appraisals. Demands arising from the environment may pose as threatening in one situation but non-threatening in another due to available resources influencing current appraisals of the situation. Lazarus and Folkman (1984) further developed the theory of stress and coping, which involves two phases in the cognitive appraisal of stress and identifying five types of appraisals (harm, threat, loss, challenge, or benign). Initially, an individual employs a primary appraisal in assessing potential negative outcomes in a situation. Next, secondary appraisal involves the individual assessing their personal abilities in being able to master potential threats. These components parallel the construct of perceived-efficacy, which concerns the belief of possessing the internal skills or resources necessary to complete a specific task. Therefore, stress is viewed as a function of mismatch between the individual’s available resources in the face of environmental demands.
While efficacy theoretically accounts for all internal processes including cognition and affect, the transactional theory has been critiqued for placing too much emphasis on cognition. In addition, there is some debate as to whether the cognitive-transactional model accurately describes the internal process of stress. This is reflected in the ongoing James-Lange versus Cannon debate (Aldwin, 1994). As early as 1922, the James-Lange hypothesis proposed that emotional (visceral) reactions to stressors not only occurred before conscious responses, but also helped develop them. Opposing this view, Cannon reported that neural processing originating in the thalamus was primary and occurs prior to the slow processing involved in visceral reaction times. Consequently, either perspective would tend to attribute the experience of stress either to thoughts or feelings rather than an interaction.

The debate continues within the stress literature between cognitive (Lazarus) and emotional (Zajonc) influences in the perception of stress (Aldwin, 1994). The cognitive transactional model argued that cognitive appraisal determines the perception of whether a situation exhibits potential harm and subsequent emotional reactions. In contrast, Zajonc argued that simple awareness should not be mistaken for cognition and that emotional reactions to stressors occur prior to cognitive reactions. However, there is common acknowledgment and acceptance within the scientific community that the brain exhibits parallel processing (Demasio, 1994; Ganzzaniga, 1989; LeDoux, 1996). Moreover, "if emotional processing is mediated more by the right hemisphere, and rational processing by the left hemisphere, then it should not be surprising that both
mechanisms are involved and that one can inform the other in a noncausal sequence” (Aldwin, 1994, p. 41).

The transaction theory defines stress and health as a bi-directional interaction between the individual and the environment and is compatible with the General Adaptation model. The shortcomings of this model include its minimization of the role that emotions play in stress and the theory’s poor attempt in explaining how the mind influences bodily functions (Rice, 1999).

*Holistic health theory.* Although the previous models each have deficits, they have served as the foundation for much of the stress research today. Traditionally, these approaches provide a more linear model, rather than a holistic and multifaceted understanding of stress. The holistic health theory is more of a health movement rather than a formalized theory (Rice, 1999). Its ideals arose from the humanistic philosophy in reaction to the dominant biological reductionism and medical specialization commonly practiced in westernized medicine. Holistic health attempts to reestablish the utility and value of mental processes in health and healing. This holistic model is “the concept underlying an approach to controlling stress and tension that deals with the complete lifestyle of the individual, incorporating interventions at several levels—physical, psychological, and social—simultaneously” (Girdano & Everly, 1979, p. 20). Therefore, any study examining multiple sources of stress will fall under the framework of this perspective (e.g., General Adaptation Syndrome, Transactional theory, as well as incorporating the influence of other psychological processes). Despite the fact that
Holistic health does not exhibit formal properties of a scientific theory, there is much research supporting its premises (Rice, 1999).

**Stress-Immunity-Health (SIH) model.** Psychoneuroimmunology examines the interaction between the functions of behavior, the nervous system, immune system, and health. Implicit in most psychoneuroimmunology research is the SIH model. Within this model, “S” represents psychosocial stress factors, “I” represents immunologic change, and “H” represents health outcome. From this perspective, the construct of stress is broken into two broad categories, which include stressors that represent stressful events and stress reaction that represents the distress experienced by the individual following a stressor. Stress reaction represents diverse experiences such as anxiety, anger, depression, fear, or a composite reaction of “feeling distressed” (Keller, Shiflett, Schleifer, & Bartlett, 1994). Ultimately, the goal is to evaluate how stress alters immune functioning, which consequently leads to adverse health effects.

“While the psychoneuroimmunology model is intuitively sensible and appealing and is supported by substantial research indicating that ‘stress’ leads to changes in immune and health outcomes, nearly all of the psychoneuroimmunology research examines only part of the overall model. There is precious little evidence which supports or even tests the entire S-I-H model in humans” (Keller, Shiflett, Schleifer, & Bartlett, 1994, p. 218). There are many methodological limitations when investigating evidence for the SIH model in human studies such as ethical issues regarding noninvasive measures (e.g., obtaining blood samples). Most studies attempting to provide support for
the SIH model are not able to test the completed model due to the constraints of funding, ethics, and practicality.

The search for evidence in understanding the process of stress needs to incorporate the ideals of the holistic approach, while providing an understanding of the physiological and cognitive components of stress that has been identified by research employing the General Adaptation model and the Transactional model. The answer lies in the Stress-Immunity-Health model and psychoneuroimmunology. This approach to research views the individual contextually and builds upon previously supported models. The current study was conducted to provide further examination of some of the components of the Stress-Immunity-Health model.

*Physiological Mechanisms of Stress*

The following presents an overview of various anatomical structures, their functions, and chemical reactions within the body as they relate to stress. As the body receives information from sensory input, whether it is real or imagined, the information travels various pathways to, from, and within the brain. For example, the hypothalamus assimilates somatic and visceral motor responses and provides the brain with responses necessary for orchestrating many of the body’s functions (Bear, Connors, & Paradiso, 1996; Glaser & Kiecolt-Glaser, 1994).
Hypothalamo-pituitary-adrenal axis (HPA). The immune system and the HPA exhibit a functional interaction that results in health outcomes (Ader, Felton, Cohen, 2001). The hypothalamus maintains many crucial tasks that are necessary for internal homeostasis and for sustaining life such as regulating temperature, blood volume, pressure, salinity, acidity, and oxygen and glucose levels in the blood. The following examples illustrate how the hypothalamus plays an integral role in understanding the physiological effects of stress in that it assimilates information from various stimulus inputs and thoughts from the cerebral cortex and the resultant affects on internal organs (Taylor, 1999). When embarrassment is experienced, the hypothalamus receives input from the cerebral cortex and then activates blood vessels via the vasomotor center in the medulla, which results in blushing behavior. Another illustration of how the hypothalamus integrates thoughts and behavior can be observed in the increase secretions of hydrochloric acid as result of the hypothalamus receiving information concerning anxiety being experienced.

The hypothalamus has been subdivided into three major zones in both hemispheres (lateral, medial, and periventricular). The lateral and medial zones have circuitry that is intimately involved in the limbic system (Bear, Connors, & Paradiso, 1996). As a result, these areas of the hypothalamus primarily deal with emotional stress and behavioral responses directed from inputs in the amygdala. The amygdala receives information from sensory and motor input regions and sends information to the lateral and medial zones of the hypothalamus. If a threat is perceived, the hypothalamus helps to regulate fear responses via the lateral zone, and sends information to the ventral
tegmental area of the midbrain via the medial forebrain bundle and elicits predatory aggressive behaviors. In addition, the medial zone sends information to the periaqueductal gray matter of the midbrain via the dorsal longitudinal fasciculus and elicits aggressive emotions such as anger. Conversely, the corticomedial nuclei of the amygdala send information to the hypothalamus via the stria terminalis and aid in decreasing aggressive behavior. The reciprocal transfer of information to and from the lateral and medial zones is communicated with the periventricular zone, which results in coordinating other organs and systems throughout the body in response to perceived stress in hopes of achieving homeostasis.

The role of the periventricular zone of the hypothalamus and its relevance in responding to stress becomes apparent when examining the following two functions. One group of cells interacts with the pituitary gland that controls the endocrine system, while another group of cells controls the autonomic nervous system (ANS) (Bear, Connors, & Paradiso, 1996). The endocrine system includes a variety of hormone releasing glands that activate changes in target organs (Ader, Felton, Cohen, 2001). In combination, the hypothalamus and pituitary gland work together in releasing various hormones in the regulation of the endocrine system, which in turn, mediates the larger system of internal organ functioning (Taylor, 1999). In the case of physical stress, the periventricular zone sends information to the posterior region of the pituitary and a variety of changes can take place within the body. For example, the release of antidiuretic hormone (ADH or vasopressin) responds to control depleting blood volume and salt concentrations by altering the functions of the kidneys to retain water and
decrease urine production. Similarly, blood pressure can be increased in a response to threatening stimuli by the kidneys secreting the enzyme, renin, which through various biochemical transformations becomes angiotensin II that directly effect blood vessels and the kidneys in raising the pressure. ADH eventually has an affect on the lateral zone of the hypothalamus via the subfornical organ, which stimulates drinking behaviors as a result of thirst.

In addition, when situations of physiological, emotional, or psychological stress (real or imagined) arises, the periventricular zone of the hypothalamus produces the corticotropin-releasing hormone (CRH), which is released into the bloodstream and reaches the anterior pituitary via the infundibulum (Bear, Connors, & Paradiso, 1996; Ader, Felton, Cohen, 2001). The pituitary reacts to CRH by releasing corticotropin or andrenocorticotropic hormone (ACTH). As ACTH travels through the bloodstream and reaches the adrenal cortex, it serves as a catalyst for the release of the steroid hormone cortisol. As a reaction to various types of stress, the release of cortisol works to engage numerous energy reserves, while simultaneously suppressing the immune system (energy consumer). This chain of physiological events serves to provide the individual with the energy necessary to effectively cope in the face of adversity or stressful condition. The beginning effects of cortisol being released into the blood stream takes approximately 30 minutes (Rabin, Kusnecov, Shurin, Zhou, & Rasnick, 1994). From a physiological observation, cortisol increases the Ca$^{2+}$ intake through voltage-gated ion channels in the brain and has been presumed to equip the brain to better deal with stress (Bear, Connors, & Paradiso, 1996). Though cortisol release is beneficial in times of distress, chronic
stress and release of cortisol can be dangerous. McEwen and Schmeck (1994) found that rats that received daily injections of corticosterone over many weeks developed corticosterone receptors on specific dendrites. In turn, these dendrites deteriorated and died in less than a month. Correspondingly, Sapolsky (1994) found that baboons that were exposed to chronic stress died prematurely and exhibited gastric ulcers, enlarged adrenal glands, and neuron degeneration in the brain.

The periventricular zone of the hypothalamus also controls the ANS, which automatically coordinates and controls the visceral functions (Bear, Connors, & Paradiso, 1996). This unconscious fine-tuned comprehensive system activates the moment-to-moment responses of visceral organs to internal and external stimuli (e.g., regulating rates of breathing, digestion, and heart rate). The ANS can be subdivided into two meaningful systems, the sympathetic nervous system and the parasympathetic nervous system. These two subsystems work together in creating an interdependent system and a rise in one system relates to fall in the other system’s activation.

The sympathetic nervous system prompts the body for a course of action in the presence of threats, intense emotions, and arduous activities (Taylor, 1999). When the body experiences stress, the sympathetic nervous system becomes activated and dominates functioning—increasing heart rate and blood supply to muscles, releasing glucose, and dilating bronchi for higher levels of oxygen consumption (DiMatteo, 1991). Correspondingly, as early as 1932 Cannon described stress as the body’s response to a threatening stimuli, which involved the activation process of the sympathetic nervous system (Taylor, 1999). This activation response to a threat has been called the flight or
fight response. As heart rate, blood pressure, and respiration increase, the individual’s body prepares to cope with the current threat by either fighting or fleeing from the immediate environment.

The parasympathetic nervous system focuses on safeguarding energy and works to return the body to a homeostatic balance once the stressor is no longer perceived as threatening (Taylor, 1999). Working in opposition to calm the body and decrease the arousal response, the parasympathetic nervous system dominates functioning—slowing heart rate, and increasing blood supply to the digestive system (DiMatteo, 1991).

The following two studies are examples of how the ANS responds to stress. Barr and Keirnan’s (1983) research illustrates how the hypothalamus helps regulate body temperature during times of stress by stimulating the parasympathetic system. The results indicated the anterior region of the hypothalamus activates heat loss behavior (e.g., cutaneous vasodilatation and sweating) in response to a rising internal temperature. These findings suggest that hyperthermia may arise in hot environments or under states of high metabolic rate (Reitan & Wolfson, 1992). In contrast, during the threat of internal temperatures dropping, the posterior region of the hypothalamus activates the sympathetic system, resulting in increased heat retention behaviors such as vasoconstriction and shivering (Reitan & Wolfson, 1992).

Various studies have examined the involvement of the nervous system and endocrine pathways as an explanation for distress affecting immune functioning. Affective states that are frequently related with stressors have led to links with decrements in immune functioning such as increase levels of depression and decline in
NK cell cytotoxicity (Ironson, et. al., 2000). While the literature suggests that stress is correlated with immune functioning and health outcomes, it has also been suggested that social support serves as a buffer to stress. Such research implicates that the construct of social support improves the functioning of the immune system, thus leading to better prognoses. The following reviews health related research with respect to social support.

Social Support as a Buffer – Health Outcomes

From a comparative and evolutionary perspective, interactions in social environments have been observed in many species and impact physiological and health functioning (Cohen & Herbert, 1996; Francis, Kiran, & Fernald, 1993; Papini, 2002; Sapolsky, 1989). From birth, attachment and dependency can be observed at varying degrees across species during early development, which may influence negative health outcomes (Boccia, Scanlan, Laudenslager, Berger, Hijazi, & Reite, 1997). Moreover, social connections and interactions are believed to be important throughout lifespan. According to some research, individuals who exhibit more isolation from or lack of support networks suggests interference with adjustment as a whole (Rice, 1999).

Links between mortality rates and absence of social support exist; with some research reporting as much as twice as high mortality rates as compared with those who exhibit a more active social support system (Berkman & Syme, 1979; House, Landis, & Umberson, 1988; Lynch, 1977; Tucker, Friedman, Wingard, & Schwartz, 1996). Additional research links positive effects of social support which has been observed in the reduction of morbidity and mortality (Berkman, 1995; Seeman, Berkman, Kohout, Lacroix, Glynn, & Blazer, 1993; Smith, Fernengel, Holcroft, Gerald, & Marien, 1994);
perceptions of emotional support on reducing mortality (Penninx, van Tilburg, Kriegsman, Deeg, Boeke, & van Eijk, 1997), and sustained low levels of social connections correlated with higher mortality rates (Cerhan & Wallace, 1997).

A great deal of research has indicated that social interactions impacts immune functioning and health outcomes (Goodkin & Visser, 2000; Rabin, 1999; and Schedlowski & Tewes, 1999). A disruption in social support has also been shown to have a negative bearing on immune functioning as shown with marital conflict (Kiecolt-Glaser, Fisher, Ogrocki, Stout, Speicher, & Glaser, 1991) and loss of a spouse (Zisook, Shuchter, Irwin, Darko, Sledge, & Resovsky, 1994). Likewise, higher levels of social support have been associated with increased positive immune functioning as observed with natural killer cytotoxicity (Baron, Cutrona, Hicklin, Russell, et al, 1990; Levy, Herberman, Lippman, D'Angelo, & Lee, 1991). Moreover, various types of social support have been found to be related to immune functions. For example, the Social Provisions Scales (Weiss, 1974) was developed to assess the six domains of relational provisions, which was positively correlated with immune function as measured by lymphocyte mitogen response, natural killer assay, and total lymphocyte and t-cell populations (Baron, Cutrona, Hicklin, Russell, & Lubaroff, 1990). Social support has also been indirectly linked with immune function by providing emotional support, influencing prosocial behaviors, and enhancing stress appraisals (Wills, 1998).

When reviewing the research on social support, there appears to be some dissension in how the construct is defined and measured. Some researchers have focused their efforts on understanding how social support structure impacts health such as the
properties of a social support network or group (Hirsch, 1981; Taylor, Sylvestre, & Botschner, 1998; Wellman, 1981). A much larger body of research has studied how social support functions impact health through instrumental or emotional support. For example, if an individual turns to a friend to vent about a specific problem, this may be defined as emotional support if the friend provides the individual with empathy. However, if the friend provides advice or material assistance in solving the problem, it now may be defined as instrumental support. In general, social support as a dynamic function has been identified as the availability of esteem, love, help, and mutual obligation via significant relationships such as family and friends (Cobb, 1976; Kaplan, Cassel, & Gore, 1977; Taylor, 1999). Research has indicated “social support has several major components, including the expression of positive affect; the expression of agreement with or acknowledgment of the appropriateness of a person’s beliefs, interpretations, or feelings; the provision of material aid; and the provision of information that the distressed person belongs to a network of mutual help and obligation” (Rodin, 1985).

Particular to this study, social support functions may be viewed as a coping mechanism in order to buffer stress, whether it is direct or indirect (Cohen & Wills, 1985). There are three theoretical perspectives that attempt to account for the buffering effects of functional support: direct effects hypothesis, buffering hypothesis, and matching hypothesis (Taylor, 1999). The direct effects hypothesis postulates that social support can prove beneficial to physical and mental health even when stress is not present. Daily interactions with family and friends are believed to provide practical
support that improves the overall quality of life. The buffering hypothesis assumes that benefits of social support are evident during times of greater distress, serving as a cache to diminish the negative effects of stress on physical and mental health. Daily interactions are not viewed as significant as opposed to support provided during stressful situations from this theory. The matching hypothesis is similar to person-environment-fit theory. This theory assumes buffer effects of social support are contextually influenced by possible incongruencies of the phenomenological needs of the individual and whether the contextual needs are received by the support system. In reality, these theories are supported by research and appear to work in a multifaceted system of social support (Rodriquez & Cohen, 1998).

Clearly, social support can be viewed as the provision of social comparison, a method for contextual interpretation of situations and emotions, models for behavior, aid, and a sense of identify or belonging (Taylor, 1999). However, there exists some ambiguity in the literature as to the benefits of social support, which strongly implies that the type of support needs to be taken into consideration (Rodin, 1985). Weiss (1974) proposes six unique interpersonal dimensions of social support that includes instrumental and emotional forms of support. Instrumental social supports include reliable alliance (awareness of trustworthy tangible aid) and guidance (information and advice from trustworthy resource). Emotional social supports include reassurance of worth (appreciation of one's abilities or competence), attachment (security cultivated by intimacy), social integration (sense of belonging to a group with shared beliefs and values), and the opportunity to provide nurturance (sense of feeling needed by others).
The present study employs the six dimensions of social support identified by Weiss (1974).

Examining various forms of social support as buffers to stress in an HIV/AIDS population would be beneficial. In order to understand better the relationship between psychosocial factors and nervous, endocrine, and immune system function, we need to examine immune functioning in general terms. The following section provides a brief overview of the human immune system. In addition, areas within the immune system that are related to the stress-immunity-health model will be underscored.

*Human Immune System*

The primary function of the immune system is to prevent or limit the negative effects in the body from microorganisms such as bacteria, viruses, and parasites that cause infection, allergies, cancer, and autoimmune disorders (Ader, Felton, & Cohen, 1991; Glaser & Kiecolt-Glaser, 1994; Levinson & Jawetz, 1998). The primary organs that make up the immune system are known as the lymphoid organs and consist of the thymus, lymph nodes, spleen, tonsils, appendix, bone marrow, and Peyer’s patches, which are areas of the small intestine that are made of lymphoid tissue (Taylor, 1999). The following overview of the immune system is derived from Ader, Felton, and Cohen (1991; 2001), Glaser and Kiecolt-Glaser (1994), and Levinson and Jawetz (1998). The literature states that there is a distinction between natural and acquired immunity.

Natural immunity consists of defensive systems that are not acquired, such as, barriers to infectious agents (e.g., skin and mucous membranes), specific cells (e.g., natural killer cells), specific proteins (e.g., interferons), and specific processes such as phagocytosis.
and inflammation. The efficacy of natural immunity does not include cells with memory or improve over time. Therefore, previous or extended exposure to the microorganism does not increase the efficiency of the natural immunity system.

In contrast, acquired immunity deals with specialized processes that are activated after exposure to specific microorganisms. In addition, acquired immunity cells increase their effectiveness after repeated exposure to an agent, as well as possessing memories to attenuate its effectiveness. Either cell-mediated or antibody-mediated (hormonal) divisions of the immune system provide acquired immunity. The response of both these divisions exhibit three important characteristics: (1) they are diverse in that they respond to millions of varying antigens; (2) long-term memory effects allow the system to respond to antigens many years after their initial exposure; and (3) the responses demonstrate discriminating specificity toward the initial antigen.

The immune process in which these host defenses originate takes place in three general ways. Initially, specific immune cells recognize the foreign organism, which is followed by an activation of specialized immune cells that bring about a specific response to the organism. Finally, the specialized cells target the identified antigen for eradication. For reasons of simplification, most immune related cells work through specific receptors designed to bind to specific microorganisms, which results in the extinction or immobilization of the intruder.

The cell-mediated division of the immune system generally begins with leukocytes or white blood cells that originate in bone marrow and are then release into the blood. The leukocytes migrate to the various organs of the immune system and are
stored until maturation. Leukocytes are then re-released and circulate in the blood, searching for cells that exhibit unfamiliar antigens. When leukocytes encounter a foreign antigen, they produce an antibody that attacks and terminates the foreign cell. There exist a variety of leukocytes within the cell-mediated division of the immune system such as T cells and B cells.

T lymphocytes (e.g., helper T cells and cytotoxic T cells) produce a variety of interleukins (e.g., IL2, IL4, and IL5) and provide defenses against infections (e.g., viruses and fungi), allergies (e.g., poison oak), graft and tumor rejection, and help augment and suppress the regulation of antibody responses. T lymphocyte-CD4 cells activate helper cells and macrophages that work to inhibit intracellular bacteria and fungi. T lymphocyte-CD8 cells activate cytotoxic cells that work to kill cells that are infected with viruses.

The antibody-mediated division of the immune system typically consists of B-lymphocytes and plasma cells. B cells function to neutralize toxins and viruses, opsonize bacteria so it is susceptible to phagozytize, along with working as a defense against allergies (e.g., hay fever), and aiding the autoimmune system.

Returning to natural immunity, natural killer cells belong in a category by themselves in that they specialize in killing virus-infected and tumor cells through the secretion of cytotoxins. They belong to the natural immunity division because they are non-specific moving throughout the body and extinguish their targets without prior exposure. In addition, they do not possess memory and their effectiveness is not
increased with exposure. However, it has been discovered that antibodies can enhance their effectiveness.

The immune system is complex and comprehensive; it does not work in isolation. The immune system is susceptible to adaptations by the brain, and therefore, is subject to psychosocial influences (Ader, Felton, & Cohen, 2001; DiMatteo, 1991). In addition, research is confounded by the ethical limitation when examining the interaction between stress, immune functioning, and health outcomes. Kemeny (1994) suggested examining the progression of HIV-1 due to the following benefits. First, there exists a wide variety in the progression of HIV-1 following infection that implies a variety of factors influence the prognosis. Second, immune functioning is vital to the progression of HIV-1 and can exhibit positive or negative influences. Third, essential immunological processes have been identified with the course of HIV and can be further tested. This study will investigate the stress-immunity-health model in the naturally occurring population of those who have been diagnosed with HIV-1 or AIDS.

**Stress-Immunity-Health Model in HIV-1 and AIDS**

The human immunodeficiency virus, Type 1 (HIV-1) is a human retroactive virus involving T-cell leukemia/lymphoma line that has been identified as the causative agent in acquired immunodeficiency syndrome (AIDS) (Schneiderman, Antoni, Ironson, Klimas, LaPerriere, Kumar, Esterling, &Fletcher, 1994). The prognosis for individuals with HIV-1 varies a great deal in that some appear to remain asymptomatic for years while others progress to AIDS within a short period of time (Ader, Felton, Cohen, 2001). The pathogenesis of HIV has been categorized into four different phases or group by the
Center for Disease Control: acute infection, asymptomatic infection, persistent generalized lymphadenopathy, and AIDS (Balbin, Ironson, & Solomon, in press).

Pathogenesis of HIV-1 and immune function. The HIV infection is usually transmitted through free virions or infected lymphocytes in blood or genital fluid. Once the infection enters a new host, “HIV virion bind to cells expressing the CD4 surface protein (mainly monocytes/macrophages and CD4+ T lymphocytes) via gp120, a glycoprotein expressed on the HIV viral envelope” (Cole & Kemeny, 2001). The HIV viral envelope fuses in the host cell’s membrane via CCR5 and CXCR4 coreceptors. When fusion has taken place, viral RNA is released into the cell cytoplasm initiating the process of reverse transcription and alters the makeup into HIV proviral DNA. Upon completion of the reverse transcription, the provirus moves to the nucleus where it is integrated into the host’s chromosome. During this acute infection phase, the infection targets organs containing lymphatic tissues or CD4+ cells such as the lymph nodes, spleen, tonsils, intestines or resident macrophage cells. The progression of the infection continues to reproduce itself with a rapid regularity, which is countered primarily by a proliferation of CD8+ T lymphocyte cells, which ultimately diminishes the immune response and leads to the asymptomatic phase. This process involves the acute spread of the infection and acute response by the immune system, which is frequently accompanied by a syndrome that mimics mononucleosis that include fever, malaise, head and muscle aches, sore throat, skin rash, swollen lymph nodes, nausea, and diarrhea (Ader, Felton, Cohen, 2001).
The second category in the progression of HIV to AIDS is marked by the absence of symptoms following the immune reaction to acute infection. While some of the initial symptoms may remain, they typically disappear by themselves or with symptom specific treatments. The asymptomatic phase has been noted to last 10 years on average with most individuals free of symptoms. In addition, this phase is marked with the presence of seroconversion (antibodies to HIV). The second and third category in the progression of HIV to AIDS appears to be less delineated and noted by the proliferation of cell conversion in lymphoid tissues, and generally, a relentless decline in CD4+ cells. Persistent lymphadenopathy is believed to involve the process of follicular dendritic cells confining much of the free HIV virions before they have a chance to leave the lymphoid tissues, thus, resulting in an increased number of infected cells within these organs.

Numerous studies have investigated other symptoms that are correlated with outset of the HIV infection such as cognitive decline, and disruptions in the HPA and autonomic nervous system (see Ader, Felton, Cohen, 2001). The final category being AIDS in the progression of HIV is marked by a substantial drop in immune functioning, particularly when CD4+ cell counts drop below 200. Consequently, the immune system becomes defenseless against a variety of opportunistic infections, which are often the cause of death. Numerous studies have added to our knowledge in HIV disease progression and the following list indicates the risk of AIDS onset or HIV related deaths: increased viral load, decline in CD4+ T cells level, increased expression of cellular activation markers (e.g., HLA-DR and CD38), increased serum activation markers (neopterin, $\beta_2$ microglobulin) and the appearance of syncytium-inducing (T-cell tropic)
viral strains (Cole & Kemeny, 1997). In addition, various correlates have been identified with HIV progression such as reduced cellular proliferation to recall antigens and mitogens, altered cytokine production (e.g., decreased IL-2 and IL-12, and increased IL-6 and IL-10), increased serum levels of IgA and IgG, reduced NK cell cytotoxicity, and a decline in serum dehydroepiandosterone concentration. While immunologic markers such as level of CD4+ T cells and cellular activation markers can provide information regarding disease prognosis, viral reproduction rates and plasma viral load have been found to be the most convincing biological predictors of HIV onset (Cole & Kemeny, 2001).

Interaction between stress and HIV. Psychological events can influence physiologic functions through the central nervous system’s control of the autonomic nervous system and its innervation of various secondary tissue systems. Psychological events also influence physiological functioning through the central nervous system’s control of circulating hormone and neuropeptides such as cortisol, \( \beta \)-endorphin, growth hormone, and prolactin (Cole & Kemeny, 2001). The release of these hormones by the limbic-hypothalamic-pituitary-adrenal axis in response to stress is being increasingly identified for their immunologic effects. For example, cortisol has been found to act as an immunosuppressant (Markham, Salahuddin, Veren, et. al., 1986), while glucocorticoids have been shown to augment HIV-1 reproduction and suppress immune functioning (Cupps & Fauci, 1982).

Moreover, psychological distress and affective distress (e.g., depression and anxiety) are believed to involve a neuroendocrine mediation through the activation of the...
limbic-hypothalamic-pituitary-adrenal axis or the sympathetic adrenomedullary system (Ironson, et. al., 2000). These authors reviewed studies that have implicated psychosocial factors, which have influenced the course of HIV infection and may account for some of the variability observed. For example, bereavement, depression, social support, and coping response to HIV infection have been indicated in altering the effectiveness of the immune system via the nervous and endocrine system as stated above, which has resulted in a poorer prognosis in HIV+ individuals. Similarly, Cole and Kemeny (2000) reviewed studies examining the linkage between psychological variables and HIV infection. The authors indicate that passive coping, negative HIV-specific expectancies, and self-blame have been found to be predictors in the acceleration of HIV progression. In addition, some studies were reviewed that identified subjective social support and interpersonal attachment as correlated with declines in immune functioning (e.g., decreased level of CD4+ and T-cells). However, many studies provide contradictory results, which may be a byproduct of methodological issues. “Understanding the biological mechanism of such relationships remains an important topic for future research—one that would be greatly aided by more comprehensive analyses of the network of reciprocal relationships among psychosocial characteristics, central and peripheral nervous system function, and immunologic and virologic bases of HIV pathogenesis” (Cole & Kemeny, 2000, p. 605).

General measures of psychosocial variables have typically failed to exhibit a relationship with HIV-1 and its progression, yet many studies have identified specific stressors and distress reactions such as chronic depression and maladaptive expectations. While subjective stress can be linked to coping styles that have been found to support the stress-
immunity-health model, there is little known regarding the interaction between different types of stress. It would be advantageous to examine the effects of various stresses so we may better understand the possible influence they have in the progression of HIV-1 infection. Ultimately, this would enable controlled studies to examine health outcomes under the stress-immunity-model by tailoring treatment plans according to the interaction effects of stress.

Statement of the Problem

Clearly, there is a vast amount of the literature supporting psychoneuroimmunology. In addition, the stress-immunity-health model provides an important avenue in the development of treatment plans when examining illness. The construct of stress has been found to interact with various endocrine and immunological functions within humans, which generally can affect health outcomes via physiological mechanisms of stress and internal processes. While many studies have investigated the relationship of stress in health, little is known about the interaction between types of stress in health outcomes. This study specifically examines psychosocial experiences of acute stress, chronic stress, daily hassles, and social support as predictors of the immune system functioning in the progression of HIV to AIDS. It is hoped that understanding how various types of stress may interact and suppress the immune system will lead to valuable information in developing individualized treatment plans that will augment pharmacological interventions according to stressful experiences.

Thus, in light of the above literature review, the following hypotheses are offered:
1. It is hypothesized that a relationship will exist between types of Stress (Acute Stress, Chronic Stress, and Daily Hassles) as well as between each type of stress and each immune marker (T-Cell Count, T-Cell Ratio, and Viral Load).

2. It is also hypothesized that a relationship will exist between Social Support variables (Attachment, Guidance, Nurturance, Reassurance of Worth, Reliable Alliance, Social Integration, and Total Support) and between each Social Support Variable with each immune marker (T-Cell Count, T-Cell Ratio, and Viral Load).

3. It is also hypothesized that social support measures will moderate the effects of stress on immunological functions as measured by T-Cell Count, T-cell Ratio, and Viral Load. This hypothesis will be examining several different relationships between the psychosocial and immunological measures.
Method

Participants

This study involved an investigation of archival data obtained from the Orange County Center for Special Immunology. All patients in that database who had completed a psychosocial assessment battery were included in this study. Due to the extremely low number (n=4) of female patients, the current study only utilized data from male patients. This resulted in a total of 97 subjects with the necessary patient data. In addition, no identifying data was a part of this study's database and only minimal demographic information was collected to ensure anonymity (e.g., age and gender).

Measures

Millon Behavioral Health Inventory (MBHI). The MBHI is a self-report inventory consisting of 150 true-false items aimed at assessing psychological characteristics that have been demonstrated to exacerbate medical conditions (Millon, Green, & Meagher, Jr., 1982). Two of the twenty scales were employed in this study in order to measure stress: Chronic Tension and Recent Stress. All of the scales have established reliability and validity.

Daily hassles scale. This is a self-report measure that taps into a wide variety of stressors that typically occur in one's daily life. This scale was also used as a measure of stress in this study. The scale has established reliability and validity (Kanner, Coyne, Schaefer & Lazarus, 1981).

Social provisions scale. This is a self-report instrument that reflects how an individual perceives what support is received from relationships with other people (Weiss, 1974). The measure consists of six scales that tap into guidance (e.g., advice or
information), reliable alliance (e.g., assurance that others may be counted on during stressful times), reassurance of worth (e.g., recognition of own competence), attachment (e.g., emotional closeness), social integration (e.g., sense of belonging to a support system), and opportunity for nurturance (e.g., providing nurturance to others). All scales have established reliability and validity.

*Immunologic measures.* Previous research validates the CD4 cell count, cell percent, and viral load as reliable predictors of many clinical outcomes in HIV-infected individuals (Bing, Hays, Jacobsen, Chen, Gange & Kass, 2000; Kemppainen, 2001). Thus, T-cell percent, viral load, and Beta-2 microglobulin were employed as indicators of immune function in the current HIV-infected sample.

*Procedures*

Orange County Center for Special Immunology provided access to data to identify those patients who had completed the psychosocial assessment battery. The necessary data for this study was gathered from the identified patient records. This included the age of the subject and data from the psychosocial measures. In addition, lab results were reviewed in order to obtain immunologic measures such as viral loads. Mean counts for viral load, T-cell counts and ratios were assessed from the data gathered from the patients’ charts in three-month intervals. All data collected were input and analyzed using SPSS.

*Analyses*

Analyses included descriptive statistics describing the sample and assessing the statistical assumptions as well as to assess the stepwise regression within the stress-immunity-health model. Predictor variables included were acute stress, chronic stress,
and daily hassles to test hypothesis 1; and guidance, reliable alliance, reassurance of worth, social integration, and attachment were included to test hypothesis 2. Criterion variables included were T-cell percent, viral load, and T-cell CD4+ independently to test hypotheses 3. Bivariate correlations among the predictor variables and each criterion variable were calculated and entered into the hierarchical equation in SPSS.
Results

Data Screening

There were six subjects whose data exhibited a missing value in the predictor variables. The missing value was replaced with group means for that variable. This method was chosen as a conservative method for estimating missing data as suggested by Tabachnick and Fidel (1996).

Univariate outlier values were identified as 3 standard deviations from the mean upon converting raw scores into standardized scores in SPSS. The converted scores revealed three cases exhibiting outlier scores in the predictor variables of Acute Stress (n=3), Chronic Stress (n=1), or Daily Hassles (n=0). As these were omnibus measures in which no other indicators were available it was decided to delete these subjects from the database; leaving a total of 94 subjects in subsequent analyses. There were no outliers reflected in the standardized scores for the social support variables.

In addition, standardized scores were computed to assess for outliers for the three criterion variables of T-Cell Count, T-Cell Ratio, and Viral Load. The converted scores reflected no outlier scores for T-Cell Count, 1 outlier score for T-Cell Ratio, and 9 outlier scores for Viral Load. The outlier for T-Cell Ratio was deleted based on the assumption that it was a measurement or data input error when compared with other scores. In addition, the outlier scores for Viral Load were deleted due to the severe violation of the assumptions and possibly skewing the data to produce unreliable results. Thus, due to overlap of extreme scores on different variables, an additional 9 subjects were deleted, leaving 85 subjects in the resultant analyses.
In addition, multivariate outliers were assessed by using Mahalanobis statistics, which identified one case, related T-cell Count, T-cell Ratio, and Viral Load. This subject was also deleted from the data in an attempt to control for unreliable skewness in the data. The final sample for analyses resulted in a total of 84 subjects from the original 97.

To test the assumption of normality, histograms were employed as suggested by Tabachnick and Fidel (1996). According to the histograms, the stress variables generally meet the assumptions of normality with the exception of Daily Hassles being slightly positively skewed and peaked (kurtosis).

Statistical Analyses

Descriptive statistics were run in SPSS on the three stress variables (Acute, Chronic, and Daily Hassles), six social support variables (Attachment, Guidance, Nurturance, Reliable Alliance, Reassurance of Worth, and Social Integration), and three immune functioning variables (T-Cell Count, T-Cell Ratio, and Viral Load). The number of subjects, minimum and maximum values, means, and standard deviations for each variable are provided in Table 1.
Table 1

Descriptive Statistics for Predictor and Criterion Variables.

<table>
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<th></th>
<th>N</th>
<th>Minimum</th>
<th>Maximum</th>
<th>Mean</th>
<th>Std. Deviation</th>
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</thead>
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<td>1</td>
<td>16</td>
<td>8.37</td>
<td>3.43</td>
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<tr>
<td>Daily Hassles</td>
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<td>3</td>
<td>173</td>
<td>55.54</td>
<td>41.42</td>
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<tr>
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<td>8</td>
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<tr>
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<td>16</td>
<td>13.30</td>
<td>2.09</td>
</tr>
<tr>
<td>Reliable Alliance</td>
<td>84</td>
<td>9</td>
<td>16</td>
<td>14.23</td>
<td>1.92</td>
</tr>
<tr>
<td>Social Integration</td>
<td>84</td>
<td>8</td>
<td>16</td>
<td>13.33</td>
<td>2.06</td>
</tr>
<tr>
<td>T Cell Count</td>
<td>84</td>
<td>7.89</td>
<td>1525.38</td>
<td>431.4880</td>
<td>296.1807</td>
</tr>
<tr>
<td>T Cell Ratio</td>
<td>84</td>
<td>.8</td>
<td>51.4</td>
<td>22.165</td>
<td>12.160</td>
</tr>
<tr>
<td>Viral Load</td>
<td>84</td>
<td>50</td>
<td>158437</td>
<td>23603.16</td>
<td>43871.65</td>
</tr>
</tbody>
</table>

Hypothesis 1. In order to assess the hypothesis that a relationship will exist between types of Stress (Acute Stress, Chronic Stress, and Daily Hassles) as well as between types of stress and Immune Functioning (T-Cell Count, T-Cell Ratio, and Viral Load) bivariate correlations (Spearman) were performed. Spearman’s Rho correlations were indicated due to the ordinal scales and non-normal distributions. Results indicated that all three types of stress were significantly correlated (Table 2). In addition, bivariate Spearman’s Rho correlations were run to assess the

Table 2

Spearman’s Rho Correlations Between Stress Variables.

<table>
<thead>
<tr>
<th></th>
<th>Acute Stress</th>
<th>Chronic Stress</th>
<th>Daily Hassles</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute Stress</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chronic Stress</td>
<td>.48**</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Daily Hassles</td>
<td>.56**</td>
<td>.40**</td>
<td>1.00</td>
</tr>
</tbody>
</table>

** Correlation is significant at the 0.01 level (2-tailed).
relationship between the three stress variables and the three immune function variables. There was one significant statistical relationship observed between T-Cell Ratio and Chronic Stress. Table 3 below displays a summary of these bivariate correlations between stress variables and immune function variables. A scatterplot exhibits the weak relationship between Chronic Stress and T-Cell Ratio in Figure 1. In sum, the hypothesis of stress variables being significantly correlated was supported, however, only one of the stress variables was correlated with one of the immune function variables.

Table 3

*Spearman’s Rho Correlations Between Stress Variables and Immune Function Variables.*

<table>
<thead>
<tr>
<th></th>
<th>T-Cell Count</th>
<th>T-Cell Ratio</th>
<th>Viral Load</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute Stress</td>
<td>.08</td>
<td>.09</td>
<td>-.16</td>
</tr>
<tr>
<td>Chronic Stress</td>
<td>.12</td>
<td>.22*</td>
<td>-.15</td>
</tr>
<tr>
<td>Daily Hassles</td>
<td>.21</td>
<td>.21</td>
<td>-.25</td>
</tr>
</tbody>
</table>

**Correlation is significant at the 0.05 level (2-tailed).**

_Hypothesis 2._ In order to assess the hypothesis that a relationship will exist between Social Support variables (Attachment, Guidance, Nurturance, Reassurance of Worth, Reliable Alliance, Social Integration, and Total Support) and between Social Support Variables with Immune Functioning Variables (T-Cell Count, T-Cell Ratio, and Viral Load) bivariate correlations (Spearman) were run. Spearman’s Rho correlations were indicated due to the ordinal scales, non-normal distributions, and the violations of normality. Results indicated that all six types of social support were significantly correlated with each other (Table 4). In addition, bivariate Spearman’s Rho correlations were run to assess the relationship between the six social support variables
Figure 1. Scatterplot of Correlation Between Chronic Stress and T Cell Ratio.

Table 4

*Bivariate Spearman's Rho Correlations Between Social Support Variables.*

<table>
<thead>
<tr>
<th></th>
<th>Attachment</th>
<th>Nurturance</th>
<th>Guidance</th>
<th>Reassurance Worth</th>
<th>Reliable Alliance</th>
<th>Social Integration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Attachment</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nurturance</td>
<td>.48**</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Guidance</td>
<td>.59**</td>
<td>.58**</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reassurance Worth</td>
<td>.43**</td>
<td>.65**</td>
<td>.72**</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reliable Alliance</td>
<td>.43**</td>
<td>.53**</td>
<td>.67**</td>
<td>.61**</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Social Integration</td>
<td>.45**</td>
<td>.45**</td>
<td>.57**</td>
<td>.65**</td>
<td>.53**</td>
<td>1.00</td>
</tr>
</tbody>
</table>

**Correlation is significant at the 0.01 level (2-tailed).
and three immune functioning variables. T-cell Count exhibited significant negative relationships with Attachment, Guidance, Nurturance, Reliable Alliance that ranged from -.23 to -.41, p<.05. T-Cell Ratio exhibited significant negative relationships with Nurturance and Reliable Alliance, -.38 and -.23 at p<.05 respectively. Viral Load exhibited a significant relationships with Nurturance resulting with .23, p<.05. Table 5 displays a summary of these results. While there is statistical significance in these observed relationships, these correlations are displayed in scatterplots in Figures 2 to 8 below. These visual presentations are indicative of a truncated sample. From Hypothesis 2, the prediction of social support variables being significantly correlated was supported, however, the social support variables being correlated with the immune function variables was generally not supported.

Table 5

**Bivariate Spearman’s Rho Correlations Between Social Support and Immune Functioning Variables.**

<table>
<thead>
<tr>
<th></th>
<th>T-Cell Count</th>
<th>T-Cell Ratio</th>
<th>Viral Load</th>
</tr>
</thead>
<tbody>
<tr>
<td>Attachment</td>
<td>-.23*</td>
<td>-.17</td>
<td>.06</td>
</tr>
<tr>
<td>Nurturance</td>
<td>-.41*</td>
<td>-.38**</td>
<td>.23*</td>
</tr>
<tr>
<td>Guidance</td>
<td>-.27*</td>
<td>-.20</td>
<td>.08</td>
</tr>
<tr>
<td>Reassurance Worth</td>
<td>-.15</td>
<td>-.13</td>
<td>-.09</td>
</tr>
<tr>
<td>Reliable Alliance</td>
<td>-.30**</td>
<td>-.23**</td>
<td>.01</td>
</tr>
<tr>
<td>Social Integration</td>
<td>-.21</td>
<td>-.18</td>
<td>.04</td>
</tr>
</tbody>
</table>

* Correlation is significant at the 0.05 level (2-tailed).
** Correlation is significant at the 0.01 level (2-tailed).
Figure 2. Scatterplot of Negative Correlation Between Attachment and T-Cell Count.
Figure 3. Scatterplot of Negative Correlation Between Guidance and T Cell Count.
Figure 4. Scatterplot of Negative Correlation Between Nurturance and T-Cell Count.
Figure 5. Scatterplot of Negative Correlation Between Reliable Alliance and T Cell Count.
Figure 6. Scatterplot of Negative Correlation between Nurturance and T-Cell Ratio.
Figure 7. Scatterplot of Negative Correlation between Reliable Alliance and T-Cell Ratio.
Figure 8. Scatterplot of Correlation between Nurturance and Viral Load.

Hypothesis 3. It was hypothesized that social support variables will moderate the effects of stress on immunological functions as measured by T-Cell Count, T-Cell Ratio, and Viral Load. However, due to the strong correlations exhibited between the predictor variables of social support and between the stress variables, a factor analyses was done to assess whether these sub-domains of social support and stress effectively differentiated. Likewise, a reduction in the variables run in the multiple regressions would also decrease chances for alpha error. With respect to stress variables, principal components analysis was conducted utilizing a varimax rotation with Acute Stress, Daily Hassles, and Chronic Stress. Eigenvalue, variance, and screeplot indicated a single component as appropriate, accounting for 66% of the variance. The component loadings for stress are shown in Table 6.
Table 6

*Component Loading for Stress.*

<table>
<thead>
<tr>
<th></th>
<th>Loading</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute Stress</td>
<td>.75</td>
</tr>
<tr>
<td>Daily Hassles</td>
<td>.62</td>
</tr>
<tr>
<td>Chronic Stress</td>
<td>.59</td>
</tr>
</tbody>
</table>

With respect to social support variables, principal components analysis was conducted utilizing a varimax rotation with Attachment, Guidance, Nurturance, Reassurance of Worth, Reliable Alliance, and Social Integration. Eigenvalue, variance, and screeplot indicated a single component as appropriate, accounting for 66% of the variance. The component loadings for social support are shown in Table 7.

Table 7

*Component Loading for Social Support.*

<table>
<thead>
<tr>
<th></th>
<th>Loading</th>
</tr>
</thead>
<tbody>
<tr>
<td>Attachment</td>
<td>.75</td>
</tr>
<tr>
<td>Guidance</td>
<td>.78</td>
</tr>
<tr>
<td>Nurturance</td>
<td>.48</td>
</tr>
<tr>
<td>Reassurance of Worth</td>
<td>.67</td>
</tr>
<tr>
<td>Reliable Alliance</td>
<td>.68</td>
</tr>
<tr>
<td>Social Integration</td>
<td>.59</td>
</tr>
</tbody>
</table>

Based on these observations, an omnibus measure of stress and social support were created by adding each of the sub-scales that loaded on the respective domain. The hypothesis that social support measures will moderate the effects of stress on immunological functions as measured by T-Cell Count, T-cell Ratio, and Viral Load was
assessed via a series of hierarchical multiple regressions. Multiple regressions were run for each of the dependent variables of immune functioning. The omnibus measure of social support was entered into the first step, the omnibus measure of stress was entered into the second step, and the cross product of social support and stress was entered into the third step for each immune measure (T-Cell Count, T-Cell Ratio, and Viral Load). Regression results indicate that the overall model did not predict the progression of HIV to AIDS. A summary of regression coefficients is presented in Table 8. While a trend for Viral Load may have presented with, $p = .086$, the change in $R^2$ was 3.5% and thus meaningless within the model.
Table 8

Model Summary of Coefficients for T Cell Count, T Cell Ratio, and Viral Load.

<table>
<thead>
<tr>
<th></th>
<th>B</th>
<th>β</th>
<th>t</th>
<th>p</th>
<th>Bivariate r</th>
<th>Partial r</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>T-Cell Count</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Social</td>
<td>-107.178</td>
<td>-0.289</td>
<td>-2.735</td>
<td>0.008</td>
<td>-0.29</td>
<td>-0.29</td>
</tr>
<tr>
<td>Stress</td>
<td>22.514</td>
<td>0.059</td>
<td>0.510</td>
<td>0.611</td>
<td>0.167</td>
<td>0.057</td>
</tr>
<tr>
<td>Social x Stress</td>
<td>-36.273</td>
<td>-0.082</td>
<td>-0.740</td>
<td>0.462</td>
<td>-0.156</td>
<td>-0.082</td>
</tr>
<tr>
<td><strong>T-Cell Ratio</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Social</td>
<td>-4.082</td>
<td>-0.268</td>
<td>-2.522</td>
<td>0.014</td>
<td>-0.268</td>
<td>-0.268</td>
</tr>
<tr>
<td>Stress</td>
<td>3.121</td>
<td>0.200</td>
<td>1.741</td>
<td>0.085</td>
<td>0.276</td>
<td>0.190</td>
</tr>
<tr>
<td>Social x Stress</td>
<td>-1.246</td>
<td>-0.068</td>
<td>-0.625</td>
<td>5.34</td>
<td>-1.48</td>
<td>-0.070</td>
</tr>
<tr>
<td><strong>Viral Load</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Social</td>
<td>2353.238</td>
<td>0.043</td>
<td>0.389</td>
<td>0.699</td>
<td>0.043</td>
<td>0.043</td>
</tr>
<tr>
<td>Stress</td>
<td>-14104.456</td>
<td>-0.251</td>
<td>-2.121</td>
<td>0.037</td>
<td>-0.227</td>
<td>-0.229</td>
</tr>
<tr>
<td>Social x Stress</td>
<td>-12664.362</td>
<td>-0.193</td>
<td>-1.740</td>
<td>0.086</td>
<td>-0.149</td>
<td>-0.191</td>
</tr>
</tbody>
</table>
Discussion

The literature has shown in a substantial number of previous investigations that there exists support for the stress-immunity-health model (Ader, Felton, & Cohen, 2001; DiMatteo, 1991; Goodkin & Visser, 2000; Rabin, 1999; Rice, 1999; Schedlowski & Tewes, 1999). While this model has had many promising results, those findings were not evidenced in the present study. In specific, this study examined the influence of various types of social support as moderators of stress with a population of persons with HIV-1. This is the first study of this type to research stress as a temporal construct looking at acute, chronic, and daily hassles in the progression of HIV to AIDS. The data in this study indicated that chronic and acute stress resembled a normal distribution suggesting that this HIV/AIDS affected sample experienced a typical amount of acute stress and chronic stress and counters would be expected due to the chronic problems and treatment associated with this population. In addition, the data reflects that acute stress was experienced at a slightly lower level than would be anticipated based on the normative data. It was expected that compliance to medical treatment and possible side effects of medication would present a higher level of daily hassles than observed. Further, this male only sample appeared to experience a low level of daily hassles as a whole. The data also indicated that this male sample experienced a relatively high level of social support on all domains, with the exception of Nurturance which reflected average range scores. As for the immune markers, the sample appears to represent what would be expected in an HIV/AIDS affected population in that their T-cell counts were extremely low and T-cell ratios were somewhat high. In addition, the data showed that a considerable number of the males had a viral load that was being maintained in the mid to
lower levels of the continuum. Thus, overall the majority of the subjects in this study had significant immune system compromise but perceived adequate levels of support and experienced average levels of stress.

The first hypothesis in this study was to assess whether a relationship existed between types of Stress (Acute Stress, Chronic Stress, and Daily Hassles) as well as between types of stress and Immune Functioning (T-Cell Count, T-Cell Ratio, and Viral Load). With respect to the first hypothesis, the three temporal measures of stress were significantly correlated with one another, however, unexpectedly there was only one fairly low, significant relationship between the stress measures and the immune functioning measures (Chronic Stress and T-Cell Ratio). This finding would appear to counter the theory of General Adaptation model and its supporting research of ongoing physiological changes in response to stressors. Thus it may be interesting to look at any potential structural changes that were observed in some of the comparative studies examining rats with chronic stress to assess whether it impacts the immune system. While the cognitive relationships may be difficult to assess, the physiological and structural relationships may be present. It is proposed here that this well documented relationship may not be easily detected in this sample due to the possible moderating effects of the extremely high level of support reported by participants in this study.

The Transactional Theory may provide some explanation for the minimal support observed for this hypothesis in that the relational interaction of the individuals in this sample between the environmental demands (stressors) and personal resources (support) are better balanced than what is reported by the average male, the average individual with HIV/AIDS, or other subpopulations with illnesses. It is important to point out that the
specialized clinic where the males in this study received treatment were effectively provided with interventions (social support) that is emphasized in the SIH model such as support groups, counseling, and psychoeducation about proactive changes in all aspects of their life. Thus already negating the effect of stress on immune functioning. It is also possible that with the technological advances in HIV/AIDS research that most individuals who are diagnosed with HIV will be offered a number of different resources including psychoeducation as well as very promising pharmacological interventions that greatly reduce and suspend the progression of HIV to AIDS. Therefore, these individuals may have learned not to appraise HIV as threatening as once believed. Thus, based upon their cognitive appraisals including support and effective treatment today this may mask any relationship between stress and immune functioning. Another possible explanation may be provided by the characteristics of the specific clinic where treatment was received by those who participated in this study such as their reputation and destigmatizing approach to treatment. The Center for Special Immunology provides primary medical care, psychosocial services, nutritional services, and case management that is tailored to meet the ongoing needs of those who are diagnosed with HIV/AIDS. Medical care incorporates specialized integrated treatment elements that target the disease, including subspecialties such as ophthalmology, gastroenterology, and infectious disease. Psychosocial services provided include individual and group counseling that target specific needs within the HIV/AIDS population such as adjustment to a chronic illness as well as other general concerns. Nutritional services are provided to promote healthy diets and dietary supplements to augment treatment and to target malnutrition, which is frequently accompanied early on in immune disorders. Case management represents
advocates for the patients’ needs by continually assessing, planning, implementing, coordinating, monitoring, and evaluating options and services to improve overall health outcomes.

Related to the integrated and comprehensive care obtained, the males that volunteered for this study may represent a subgroup that possesses a more optimistic understanding and outlook of their disease. Thus, addressing perception of the disease and care availability may be important in future studies. Moreover, while the transactional model may be embedded in the Stress-Immunity-Health model, this study’s measures may not have been sensitive to the specific areas identified in this model and thus the intended relationships were undetectable due to the measures employed. However, it is still important to note that the results indicated small and mostly insignificant relationships between stress (i.e., acute, chronic, and daily hassles) and the immune function variables. These findings do not reflect the body of research that suggests that stress appears to work as an immunosuppressant (e.g., Bear, Connors, & Paradiso, 1996; McEwen and Schmeck, 1994; Rabin, Kusnecov, Shurin, Zhou, & Rasnick, 1994; Sapolsky, 1994). Further, findings in several studies have been replicated with respect to stress exhibiting a negative effect on immune functioning in a HIV/AIDS population (e.g., Cole & Kemeny, 2001; Cupps & Fauci, 1982; Ironson, et. al., 2000; Markham, Salahuddin, Veren, Orndorff, & Gallo, 1986). It is suggested here that the low and unpredictable relationships between stress and immune function may have been influenced by the methodologies employed in the current study. Thus, these concerns are presented below in a discussion of the psychosocial measures utilized. The males in this study also exhibited an average amount of stress and the redundancy of constructs
observed in each type of stress may have also contributed these insignificant relationships between stress and immune functioning.

The second hypothesis in this study was to examine whether a relationship existed between Social Support variables (Attachment, Guidance, Nurturance, Reassurance of Worth, Reliable Alliance, and Social Integration) and between Social Support variables with Immune Functioning variables (T-Cell Count, T-Cell Ratio, and Viral Load). With respect to the second hypothesis, the six types of social support were significantly correlated with each other as predicted.

The findings reflect truncated distributions within the sample for each type of social support measured. There are a couple of possible explanations regarding the general uniformly high levels of social support perceived by the sample. First, the correlations between the six types of social support were fairly high. However, it is interesting that Nurturance was perceived as lower in support than Attachment, Guidance, Reassurance of Worth, Reliable Alliance, and Social Integration. Though Nurturance was significantly related with the other five types of support, it did not exhibit the high relationship observed between other types of social support. A possible explanation may be that individuals with HIV/AIDS have more opportunities for social support except when it involves nurturing others. This may be due to a protective factor of friends and family in that they do not want to place any demands on the individual who has HIV/AIDS. Another issue regarding high correlations of the different domains of social support may be that the social support measure did not adequately differentiate between these domains as measured within this HIV/AIDS sample.
Second, it is likely that this study sample maintains a unique social support makeup, which may be perceived to be greater than the average person due to their diagnostic condition and the environment they have chosen (i.e., treatment at this clinic). This sample’s high ratings of social support may also be indicative of the last decade’s change in stigmatization and treatment for those affected with HIV/AIDS. It is not uncommon for an individual who is diagnosed with HIV to receive counseling and referrals to support groups as a regular component of the treatment protocol. While these individuals may be experiencing some relational disruptions due to phobic responses, various alternative support is available and encouraged as part of a treatment package; increasing ones resources to aid their coping process. This provides further support for the Transactional model within a HIV/AIDS population in that the cognitive processes and perceptions shape stress. In addition, individuals diagnosed with HIV may present a unique group of men who are more apt to seek out support than the average male, generally resulting in a higher than average support network. Likewise, these males are not representative of the larger HIV population in that they received treatment from a clinic that specializes in immunology, and is by reputation more supportive than the typical medical clinic.

The observations in the current study exhibited various relationships between four of the six social support variables (i.e., Attachment, Guidance, Nurturance, and Reliable Alliance) and immune functions in the progression of HIV to AIDS as measure by t-cell count, t-cell ratio, and viral load. Both, Attachment and Guidance exhibited a small, inverse relationship that was significant. Similarly, Reliable Alliance exhibited a small, inverse, significant relationship with T-Cell Count and T-Cell Ratio. Furthermore,
Nurturance exhibited a small, inverse, and significant relationship with T-Cell Count and T-Cell Ratio as well as a small and significant relationship with Viral Load. These inverse relationships observed between social support variables and T-Cell variables, and between Nurturance and Viral Load appear to counter the theoretical implications of the SIH model at first glance (Keller, Shiflett, Schleifer, & Bartlett, 1994).

These findings also counter what would be predicted by previous research showing a positive relationship between social support and immune functions (e.g., Goodkin & Visser, 2000; Rabin, 1999; and Schedlowski & Tewes, 1999). What’s more, the current findings were unable to replicate previous research that utilized the Social Provisions Scale (Weiss, 1974) and indicated that social support interacts with immune functions (Baron, Cutrona, Hicklin, Russell, & Lubaroff, 1990). Once again, however, it is important to highlight the truncated scores in the data, the perception of high support in the subjects, and the type of clinic that these subjects obtained services. While the truncated observations in the data may present a dilemma of a skewed or biased sample, it is consistent across all predictor variables and suggests a phenomena within the sample, which may be explained by the unique makeup and wide-ranging services provided by the Center for Special Immunology.

Nevertheless and according to the SIH model, one would expect positive relationships between social support and immune markers, and a negative relationship between social support and viral load. In spite of this, a likely alternative explanation of the inconsistent results may be derived from an impetus within the individual based on the status of the individual’s health and HIV/AIDS. For example, individuals may be
more likely to reach out and incorporate increased support when they perceive their stress or poorer health greater than before. Consequently, these periods of increased strain on their system would produce stronger needs to attain the support necessary to cope with the stress. At the same time as their perceived support being high, their immune functioning may also low, creating an inverse relationship. Furthermore, the small relationships observed within this sample may be explained by the variable and unpredictable course of HIV/AIDS in that the observed relationships between social support and immune markers are greatly determined by intermittent fluctuation in the status of HIV/AIDS as well as in psychosocial conditions.

Unfortunately, the third hypothesis that examined the social support variables as moderators of stress in the progression of HIV to AIDS resulted in insignificant relationships between the three constructs being investigated. The lack of findings between social support variables, stress variables, and immune function variables are disappointing, but as stated above, the intricate and more subtle relationships in this sample may be difficult to assess. Correspondingly, it is possible that the constructs under investigation would be difficult to assess if the components of the SIH model were already addressed effectively in the treatment process. For example, efforts concentrating on the different stressful (S) aspects related to acquiring HIV such as stigma, comorbid health risks, medication side effects, and treatment compliance would moderate stress effects on immune functioning (I). Further, advances in pharmacological interventions targeting immune functioning (I) would moderate the effects on health outcomes (H).
Importantly, it may also be proposed that these findings supporting the null hypotheses are possibly due to the methodologies employed by the current study. As discussed above the sample studied was unique as being only male, perceiving both average stress and high support and attending a supportive and specialized clinic. Further, how the measures were attained and utilized may have also affected the results. The social support measures and the stress measures were highly correlated within each construct and suggest that the validity and reliability of the instruments were retained in the current study as well as retaining its multidimensionality. Therefore, the psychometric properties are not as easily questioned as the methodology.

One possible methodological explanation for the insignificant findings as related to measures may be found in the gathering of immune function data. For example, the immune data represented the mean for each individual taken from blood tests in three-month intervals. The actual number of blood samples for each subject was eight. However, one subject may have a mean representing 8 blood samplings across a three-year period, while another subject may have a mean representing 8 blood samplings across two-year period. Moreover, there was no control in the blood sampling, and therefore, some subjects were not treatment compliant in obtaining blood samplings every three-month interval (i.e., some maintained regular appointments while others did not). Consequently, the lack of control in obtaining reliable and regular immune function measures from each subject may have skewed the data.

Another central issue to the present study is raised when examining the time sequence in gathering the data. As previously stated, the immune function data was gathered over a varied amount of time; in some cases as long as three years. In contrast,
the psychosocial data was collected on a single day for participants and did not correlate with any point in time with respect to their treatment or course of disease. For example, data collection examining a participant's psychological and social phenomenological perspective may have been obtained at the outset, the middle, or the end of treatment. This poses a threat to possible relationships between psychosocial variables and the immune function variables in that one is more representative of longitudinal data set (immune function) and the other is more representative of cross-sectional data set (psychosocial). However, it is surprising that chronic stress which is taken at a single point in time did not exhibit a stronger relationship to the more enduring mean immune measures. More specifically, chronic stress and immune measures in this study represent more longitudinal measures and one may suspect that chronic stress exhibit a more resilient effect on the immune functioning.

As implied earlier, another concern about the methodology is that the psychosocial data was collected at different times in treatment for different subjects, according to their office visits. Associated with these problems was the inconsistency in collecting the immune functioning measures. It is proposed that the inherent problems in the current methodology may have compromised the findings and resulted in contraindicated outcomes when compared to SIH literature.

Limitations

Limitations in this study related to the sample and measurement methodologies, and are inherent in HIV/AIDS research. One limitation deals with the sample comprising only males. Males represent a specific phenomenology that may have biased the results on psychosocial measures. In addition, the small size and heterogeneity of the sample
were problematic in meeting the assumptions of multivariate statistics. In addition, the sample was comprised of individuals within a relatively small region in Southern California, which may also present a bias. Furthermore, the method in which the data was collected may pose a threat to outcomes studies due to the sensitive relationship between ever changing life events and immune functioning. Specifically, meaningful relationships may be washed out when stress and social support are not measured in tandem with immunological measures.

**Directions for Future Research**

The reluctant conclusions in the present study raise some viable solutions in an attempt to control for confounding variables and methodological shortcomings. It may prove beneficial for future research to undertake the following suggestions. One suggestion may be to limit the time period in which immune function data is collected. For example, the three-month intervals may still apply but utilize intervals before, during, and after the psychosocial measures are administered. Another possible benefit may be found in administering pre- and post-psychosocial measures. However, this possible endeavor is time consuming and not very cost effective, thus the preceding suggestion would be favored. Also, it may prove beneficial for researchers to setup post administration interviews or include item analyses to validate some of the extreme scores that were deleted from the original data set. The following are a few suggestions that may be advantageous when exploring the SIH model in the HIV/AIDS population: 1) include various treatment facilities to examine the wide-ranging support available; 2) examine how possible labeling and stigma is handled within treatment of different facilities; and 3) examine the perceptions of family members with regard to beliefs and
emotions related to HIV/AIDS. Studies evaluating these related issues may uncover specific treatment models that are able to observe self-reported variability effectively and provide further support for the SIH model in the HIV/AIDS population. This method may quite possibly clarify those relationships between stress, immunity, and health outcomes, which may otherwise be diminished when treatment takes on a more holistic approach that resembles the SIH model.
References


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