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Psychosocial Co-Factors and HIV Disease Status.

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Psychosocial co-factors and HIV disease status

Thomason, Bradley Thomas, Ph.D.
The Louisiana State University and Agricultural and Mechanical Col., 1993
PSYCHOSOCIAL CO-FACTORS
AND HIV DISEASE STATUS

A Dissertation
Submitted to the Graduate Faculty of the
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requirements for the degree of
Doctor of Philosophy

in

The Department of Psychology

by
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December, 1993
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# TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACKNOWLEDGMENTS</td>
<td>ii</td>
</tr>
<tr>
<td>LIST OF TABLES</td>
<td>v</td>
</tr>
<tr>
<td>LIST OF FIGURES</td>
<td>vi</td>
</tr>
<tr>
<td>ABSTRACT</td>
<td>vii</td>
</tr>
<tr>
<td>INTRODUCTION</td>
<td>1</td>
</tr>
<tr>
<td>Human Immunodeficiency Virus</td>
<td>2</td>
</tr>
<tr>
<td>Psychological Factors Associated with HIV</td>
<td>9</td>
</tr>
<tr>
<td>Psychosocial Co-factors in HIV Disease</td>
<td>11</td>
</tr>
<tr>
<td>PURPOSE OF THE STUDY</td>
<td>29</td>
</tr>
<tr>
<td>METHODS</td>
<td>34</td>
</tr>
<tr>
<td>Subjects</td>
<td>34</td>
</tr>
<tr>
<td>Measures</td>
<td>35</td>
</tr>
<tr>
<td>Procedure</td>
<td>40</td>
</tr>
<tr>
<td>RESULTS</td>
<td>42</td>
</tr>
<tr>
<td>Descriptive Statistics</td>
<td>42</td>
</tr>
<tr>
<td>Correlational Statistics</td>
<td>46</td>
</tr>
<tr>
<td>Structural Analyses</td>
<td>48</td>
</tr>
<tr>
<td>DISCUSSION</td>
<td>55</td>
</tr>
<tr>
<td>CONCLUDING REMARKS</td>
<td>62</td>
</tr>
<tr>
<td>REFERENCES</td>
<td>67</td>
</tr>
<tr>
<td>VITA</td>
<td>77</td>
</tr>
</tbody>
</table>
LIST OF TABLES

<table>
<thead>
<tr>
<th>Table</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Demographic Variables: Descriptive Statistics.......................</td>
<td>43</td>
</tr>
<tr>
<td>2. Sample Demographics by Site........................................</td>
<td>44</td>
</tr>
<tr>
<td>3. Experimental Variables: Descriptive Statistics......................</td>
<td>45</td>
</tr>
<tr>
<td>4. Correlational Matrix of Predictor X Outcome Variables................</td>
<td>47</td>
</tr>
<tr>
<td>5. Direct Effects and %Variances of Psychosocial Co-factors on HIV Symptoms in HIV Symptom-Only Structural Model.................</td>
<td>53</td>
</tr>
<tr>
<td>6. Direct Effects and %Variances of Stress, Social Support, and Coping on Mood in HIV Symptom-Only Structural Model..................</td>
<td>54</td>
</tr>
</tbody>
</table>
# LIST OF FIGURES

<table>
<thead>
<tr>
<th>Figure</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Structural Model of Psychosocial Co-factors and HIV Disease</td>
<td>33</td>
</tr>
<tr>
<td>2.</td>
<td>Structural Model of Psychosocial Co-factors and HIV Symptoms</td>
<td>52</td>
</tr>
</tbody>
</table>
ABSTRACT

Concern over the devastating effects of the Human Immunodeficiency Virus (HIV) has been met with rigorous research efforts by allied health professions. Identifying variables, or co-factors, that contribute to the progression of HIV has been of increased interest to health professionals in recent years. Social scientists as well as medical professionals have begun to offer evidence that certain psychosocial co-factors are important in predicting the health status of HIV-positive individuals.

A few models have been offered to illustrate the interrelations among psychosocial co-factors and HIV disease. Stress, social support, coping, and mood states constitute the most frequently researched variables in this area. However, data have been inconclusive and the models have not been substantiated empirically.

The present study examined the impact of stress, social support, coping and mood on the health status of HIV-positive individuals. Correlational data suggested all of these psychosocial co-factors were associated with HIV symptom count. However, no psychosocial variable (with the exception of coping) predicted immune functioning in these same individuals. A model was created and supported that illustrated the links among these psychosocial co-factors and HIV symptoms. The results are discussed with implications for future HIV research and treatment.
INTRODUCTION

Over the past decade, awareness of the devastating effects of Human Immunodeficiency Virus (HIV), primarily with regard to the Acquired Immune Deficiency Syndrome (AIDS), has increased exponentially. Unfortunately, AIDS awareness among the general population often is transformed into misconceptions concerning modes of transmission, phobias of HIV-infected individuals, and prejudices against high risk groups. In contrast, heightened concern among allied health professions is resulting in positive endeavors. Educational programs, effective treatments, and rigorous research geared toward combatting the disease are paving the way toward a final resolution of the HIV crisis.

Social scientists have made significant contributions in the fight against HIV. Psychologists were at the forefront of planning and implementing an AIDS prevention program for the San Francisco area (McKusick, Conant, & Coates, 1985). As a result, relative to other high risk populations, HIV prevalence among homosexual/bisexuals has decreased substantially (Nash & Said, 1992; Winkelstein & Padian, 1987). AIDS dementia and the psychological sequelae of HIV infection are popular topics on research agendas. Additionally, mental health professionals are developing and empirically scrutinizing psychological interventions for HIV patients (Kelly & St. Lawrence, 1988).
The efficacy of psychotherapy has been observed not only in attenuating the psychosocial impact of HIV but also in treating the physical manifestations of HIV disease (Antoni et al., 1991; LaPerriere et al., 1990). Data are beginning to emerge to suggest that psychosocial variables serve as important co-factors in HIV disease susceptibility and progression (Mulder & Antoni, 1992; Solomon, Kemeny, & Temoshok, 1991). These recent investigative efforts are supported by previous research included under the rubrics of psychoimmunology or psychoneuroimmunology (Ader, 1991). Research in this area has elevated the understanding of immune disorders and provided support for multifactorial models of diseases, including HIV (Jemmott & Locke, 1984; Livingston, 1988).

**Human Immunodeficiency Virus (HIV)**

**Definition and Epidemiology**

The HIV clinical spectrum constitutes the United States' primary health concern (Livingston, 1988). A number of clinical manifestations are possible, with AIDS typically being the end result. The virus was named Human T-Cell Lymphotropic Virus Type III (HTLV-III) by the National Cancer Institute and Lymphadenopathy -Associated Virus (LAV) by a group of French researchers (Fenton, 1987). To prevent confusion, the International Committee
on the Taxonomy of Viruses suggested the inclusive term "Human Immunodeficiency Virus (HIV)" (Fenton, 1987).

HIV is a retrovirus, a classification given to viral strains capable of transcribing their own genetic material into a host cell (Wormser, Stahl, & Bottone, 1987). Following invasion of the host cell, HIV's RNA is uncoated and transcribed into DNA. The viral DNA is integrated into the host cell's nucleus, where it may remain dormant or generate buds of genetic material. These buds are released from the cell membrane, replicating the virus and leaving the host cell to die (Fultz, 1989). Because of its ability to lie dormant, HIV is subclassified as a lentivirus. Lentiviruses replicate slowly and the resulting clinical effects are often latent (Wormser, Stahl, & Bottone, 1987).

Current epidemiological information indicates that anywhere from 900,000 to 2 million people in the U.S. are HIV positive. As of December, 1992, over 253,448 cases of AIDS had been reported in the U.S. while 611,589 cases were reported worldwide (Centers for Disease Control Statistics, 1992). High risk adult populations include homosexual/bisexual males (60%), intravenous drug users (21%), homosexual/bisexual male intravenous drug users (7%); heterosexuals (5%), transfusion/tissue recipients (2%), hemophiliacs (1%) and others/undetermined (4%) (Nash & Said, 1992). There is an uneven ethnic distribution of AIDS. Minority groups, namely African-American (30% of
U.S. cases) and Hispanic (17% of U.S. cases), are represented in greater proportions. Eleven percent of U.S. cases are females. Misconceptions concerning potential modes of HIV infection are ubiquitous. However, data indicate that only blood, semen, and vaginal fluid contain significant enough portions for transmission (Centers for Disease Control Statistics, 1993; Fernandez & Ruiz, 1989; Scutchfield & Benenson, 1989).

Pathophysiology

HIV pathogenesis typically begins with altered immune functioning. Traditionally, immune functioning has been delineated into two categories, cellular immunity and humoral immunity (Wormser et al., 1987). Cellular immunity is the category primarily affected by HIV. Two types of cells, helper (T4 or CD4) T-lymphocytes and suppressor (T8 or CD8) T-lymphocytes, regulate cellular immunity. These cells are produced by lymphoid tissue and mature in the thymus (DeVita, Hellman, & Rosenberg, 1985; Dixon & Fisher, 1983). Both the total CD4 count and the CD4/CD8 ratio provide the signals for a variety of immune processes. HIV destroys CD4s and disrupts the critical CD4/CD8 ratio, leaving an individual's immune system compromised (DeVita, Hellman, & Rosenberg, 1985).

Alterations in immune functioning are evident at a number of levels. Several principle changes in cellular immunity occur. First, the number of lymphokines (e.g.,
interleukins 1 and 2, gamma-interferon) is depleted. Lymphokines regulate the activity of other immune substances, mainly phagocytes and natural killer (NK) cells. Both phagocytes and NK cells attack and eliminate invading foreign substances (antigens) that initiate infection and disease. Second, this reduction in lymphokines is paralleled by compromised ability and decreased proliferation of both phagocytes and NK cells. Finally, as these immune substances are eliminated, the individual becomes susceptible to a variety of opportunistic infections and diseases (DeVita, Hellman, & Rosenberg, 1985; Dixon & Fisher, 1983; McDougal et al., 1991).

CD4 count and the CD4/CD8 ratio also signal important humoral immune processes, namely the production and release of antibodies via B-lymphocytes (lymphocytes that mature in bone marrow). As helper T-lymphocytes are destroyed by HIV, the organism fails to produce certain antibodies that destroy antigenic agents (DeVita, Hellman, & Rosenberg, 1985; McCutchan, 1990) while other antibodies (e.g., gammaglobulin) may be elevated (McDougal et al., 1991). Although hypergammaglobulinemia occurs, there is a poor antigen-specific response of the circulating antibodies due to immunodepression (McDougal et al., 1991).

As HIV induces the cascade of immunological deficits, disease manifestations develop over time. However, HIV
symptomatology is highly unpredictable and sometimes transient, making disease course variable among infected individuals (Rogers & Masur, 1989). The Centers for Disease Control (CDC) has developed a four-point classification system of HIV disease (Rogers & Masur, 1989). Type I is associated with acute infection. Recent studies have indicated that a mononucleosis-like syndrome with a duration of approximately two-three weeks is common, but not always present, following acute infection. This syndrome is characterized by fever, diaphoresis, lethargy, muscle ache, headache, and sore throat (Cooper et al., 1985).

Asymptomatic individuals comprise the type II classification, while type III classification is characterized by persistent generalized lymphadenopathy (PGL; Rogers & Masur, 1989). PGL is diagnosed when two lymph node sites, other than the groin area, remain swollen for at least one month (Bartlett & Finkbeiner, 1991). Although frequently considered an ill-defined syndrome, many individuals with PGL exhibit additional symptoms constituting AIDS-Related Complex (ARC). ARC is characterized by lymphadenopathy, fever, weight loss, and a reduction in both red and white blood cells. Signs and symptoms of ARC include shortness of breath, persistent cough, yeast infections, diarrhea, night sweats, fatigue, and malaise. Persons with ARC may display the entire
spectrum of clinical manifestations or a select few (Badgley, 1987).

Type IV classification of HIV disease is AIDS (Rogers & Masur, 1989). AIDS is diagnosed when an infection or disease develops in an HIV-positive individual. The most common diseases associated with AIDS are Pneumocystis carinii pneumonia (present in 60% of U.S. adult cases), oral/pharyngeal candidiasis (present in 45% of U.S. adult cases) and Kaposi's Sarcoma (present in 10% of U.S. adult cases). Other diseases commonly associated with AIDS include cryptococcosis, cytomegalovirus, Mycobacterium avium complex, herpes zoster, herpes simplex, cryptosporidiosis, tuberculosis, immunoblastic sarcoma, and toxoplasmosis (Nash & Said, 1992).

The progression from HIV seroconversion to AIDS is not well understood and varies a great deal among infected individuals (Rogers & Masur, 1989; Scutchfield and Benenson, 1989). Duration of infection has been the only consistent predictor of disease progression (McDougal et al., 1989). Sonnabend, Witkin, and Purtilo (1984) were among the first authors to ascribe importance to possible HIV co-factors in their "immunological overload" hypothesis. Repeated exposure to HIV, excessive drug use, poor nutrition, aberrant health practices, and presence of other viral pathogens present chronic challenges to immunocompetence. These constant insults "overload" the
immune system, leaving the individual susceptible to HIV infection and HIV illness progression (Levy & Ziegler, 1983; Sonnabend, Witkin, & Purtilo, 1984). Other hypothesized co-factors include premorbid immune system competency, lifestyle, therapies, and psychological variables (Livingston, 1988; McDougal et al., 1989).

**Treatment**

Treatment for HIV is in the experimental stage. The most popular treatment thus far has been zidovudine (azidothymidine or AZT), an antiviral agent that slows HIV replication. AZT has a number of untoward side effects (e.g., bone marrow suppression and anemia). Dideoxycytidine (ddC) and dideoxyinosine (ddI) constitute another class of antivirals similar to AZT. Although clinical trials have reported some efficacy in slowing the viral replication, peripheral neuropathy is a common side effect of ddI and ddC (Cohen, Sande & Volberding, 1990). Other experimental treatments are becoming rapidly available. However, concern over their toxicity and efficacy deters the accessibility of these interventions (DeVita, Hellman, & Rosenberg, 1985). Other treatment options commonly employed by HIV patients include physical rehabilitation (Mukand, 1991), psychotherapy (Kelly & St. Lawrence, 1988), and alternative therapies (Badgley, 1987).
Psychosocial Factors Associated with HIV

In addition to the numerous medical complications associated with HIV infection, the psychological sequelae presents a significant complicating factor throughout the HIV clinical spectrum. Even among seronegative individuals in high risk categories, high rates of psychological distress have been observed. Clinical features identified among these "worried well" include panic, anxiety, obsessions, compulsions, somatization, hypochondriasis, substance abuse, depression, and bereavement (Faulstich, 1987; Perry, Jacobsberg, Fishman, 1990).

Following infection, up to 90% of all HIV patients may suffer psychological and/or neuropsychological complications (Fernandez & Ruiz, 1989). According to Fernandez and Ruiz (1989), mental status impairment in HIV likely results from one (or a combination) of the following: "1. direct HIV infection of the brain; 2. a secondary brain infection by an opportunistic organism; 3. an emotional reaction to illness; 4. an unrelated psychiatric disorder, previously or newly diagnosed."

Psychopathology can manifest at any stage of HIV disease. Some studies have demonstrated that higher rates of psychological distress occur during earlier rather than later stages of HIV infection, when uncertainty about prognosis is heightened (Temoshok, 1986; Tross et al., 1986).
Anxiety and depression constitute the most frequently identified psychological symptoms among persons with HIV (Holland & Tross, 1985). Common fears include prognostic uncertainty, imminent death, infecting others, ostracism, and a pervasive feeling of lost control. Up to 30% of HIV patients experience clinical levels of depression. Suicide rates may exceed those of the general population by up to 66 times (Marzuk et al., 1988). Psychiatric morbidity is common, with the most frequent diagnoses being (in order of prevalence) adjustment disorder, major depression, delirium, dementia, treatment-related side effects, and premorbid psychiatric or personality disorder (Holland & Tross, 1985).

Neuropsychological deficits are pervasive among HIV patients, with AIDS dementia complex (also referred to as HIV dementia) being the most common neurological syndrome among HIV patients (Navia & Price, 1987). Prevalence estimates of AIDS dementia range from eight to 66% in individuals diagnosed with AIDS (Maj, 1990). Research has indicated that cognitive impairment may be the sole or presenting feature of AIDS in a large percentage of patients (Navia & Price, 1987). Impairments in memory, concentration, attention, and speed of cognitive processing as well as decreased motor performances (e.g., ataxia, hyper-reflexia, coordination difficulties, and dysarthria) are frequently observed (Maj, 1990). Treatment outcome
studies have shown that cognitive deficits can be successfully managed with AZT (Maj, 1990).

Psychological Co-factors in HIV Disease

A more recent focus of psychosocial investigations concerning HIV has been the impact of psychological variables on HIV disease status. An interactive model explaining HIV susceptibility purports that biological and psychosocial variables combine to leave an individual immunosuppressed and vulnerable to HIV infection (Livingston, 1988). Interaction models explain not only susceptibility to infection but also progression of the HIV clinical continuum (Livingston, 1988). A number of psychosocial variables have been implicated by interaction theorists including classical conditioning, mood, stress, social support, coping, and personality.

Conditioning

In recent years, researchers have suggested that immune processes can be classically conditioned. Animal studies have indicated that both immune suppression (Ader, 1985) and immune enhancement (Ghanata, Hiramoto, Solvason, & Spector, 1985; Gorczynski, Macrae, & Kennedy, 1982) are capable of being conditioned. To date, one human study exists that examined conditioning of the immune response. Smith and McDaniel (1983) conditioned an allergenic hypersensitivity response among tuberculin-positive
individuals. Taken together, these studies provide limited evidence that immune processes can be classically conditioned. Aside from the paucity of data, a primary limitation concerns anthropomorphism. Differences in animal and human immune systems are clearly evident (Dixon & Fisher, 1983). More human studies that include HIV populations are necessary before definite conclusions can be reached.

Despite these limitations, a number of authors (Baumgartner, 1985; Coates, Temoshok, & Mandel, 1984; Kiecolt-Glaser & Glaser, 1988) have proposed that HIV susceptibility and clinical course can be conditioned and have cited the aforementioned studies to support their claims. Basically, the conditioning model proposes that conditioned learning leads to immunosuppression which leaves the individual vulnerable to infection when exposed to HIV. Repeated occurrence of conditioned behaviors facilitates HIV illness progression (Baumgartner, 1985; Coates, Temoshok, & Mandel, 1984; Kiecolt-Glaser & Glaser, 1988). These authors do not clearly identify stimuli and responses in their models. Coates, Temoshok, and Mandel (1984) suggest that an environmental stressor (unconditioned stimulus) leads to immunosuppression (unconditioned response). Reactivated internal states (e.g., anxiety) associated with the stressor become the conditioned stimulus and promote further immunosuppression
(conditioned response), resulting in HIV disease progression or increased susceptibility to HIV infection (second-order conditioned responses).

**Mood**

In addition to conditioning, researchers have been interested in the impact of affective states on physical and psychological well-being of HIV patients (Baum & Nesselhof, 1988; Chuang, Devins, Hunsley, & Gill, 1989). Evidence for a relation between altered mood and immune functioning has been well documented in the literature. Bereavement has been associated with decreased lymphocyte responsivity (Bartrop, Lockhurst, Lazarus, Kiloh, & Penny, 1977; Schleifer, Keller, McKegney, & Stein, 1979) and impaired NK activity (Kiecolt-Glaser et al., 1984). Patients with major depression have demonstrated a deficiency in T-lymphocyte responsivity and lowered levels of T-helper cells (Calabrese, Kling, & Gold, 1987; Kiecolt-Glaser et al., 1984). Severity of depression also has been correlated significantly with reduced NK cytotoxicity (Irwin, Daniels, Bloom, & Weiner, 1986).

The previously described studies suggest that bereavement and depression alter immunity. Few studies, however, have examined mood variables as they relate to the HIV illness process. In a clinical narrative, Laudenslager and Reite (1984) cited evidence for HIV illness progression resulting from bereavement in a 42-year-old HIV-positive
male. Solomon and Temoshok (1987) reported preliminary findings of their longitudinal study examining mood variables and immune functioning in individuals with HIV. Among ARC subjects, increased hostility predicted quicker mortality. Dysphoria and anxiety were correlated with leukocytes, indicating possible higher incidence of opportunistic infections in psychologically distressed individuals with ARC (Solomon & Temoshok, 1987).

As part of the Multicenter AIDS Cohort Study, Ostrow et al. (1989) administered the Center for Epidemiological Studies Depression (CES-D) Scale (Radloff, 1977) to nearly 5,000 homosexual men. Depression was positively associated with lymphadenopathy and prevalence of HIV-related symptoms. Zich and Temoshok (1987) reported similar findings in their comprehensive study of psychosocial functioning and HIV symptoms. Total number of HIV-related symptoms was correlated with self-reported depression and anxiety in 50 men diagnosed with AIDS. However, no mood effects were obtained in the sample of ARC patients.

In their investigation of 113 HIV-positive gay men, Gorman et al. (1991) examined correlations among depression, anxiety, T-cell counts, urinary cortisol, and overall health. Cortisol level was correlated significantly with health status, depression, and anxiety in a positive direction. However, the effects were small,
and nonsignificant associations were found with immune parameters.

**Stress**

A popular theme in behavioral medicine has been the study of stress and its relationship to health. Hans Selye (1950) stimulated much of the interest in stress research with identification of the "General Adaptation Syndrome (GAS)." Stress research has become more sophisticated over the past four decades, allowing specialty fields to emerge. The emergence of psychoneuroimmunology has directed recent investigative efforts toward uncovering a nexus between stress and the immune system. This work has important implications for diseases of the immune system, HIV-related illnesses in particular.

Livingston (1988) proposed a model to explain how stress contributes to immunosuppression and susceptibility to AIDS. In this "sociopsychophysiologic" model, life situations are referred to as stressors and a response to the events is referred to as stress. According to Livingston (1988), stress occurs when an individual’s assessment of a stressor overwhelms his or her "Filter Resource Capability System (FRCS)." That is, the individual’s perceived capabilities are not sufficient to deal with the event. In discussing his model, Livingston (1988) stated that stress suppresses an individual’s immune
functioning, leaving him or her susceptible to opportunistic infection.

Supporters of a stress approach to HIV susceptibility have pointed out that the stressors faced by high risk groups (e.g., gay men and drug abusers) may be greater than those of the general population (Cecchi, 1984; Livingston, 1988). Cecchi (1984) regards stress as the most probable determining factor to explain why gay men are more susceptible to AIDS than heterosexuals. Potential stressors encountered by high risk, HIV-negative individuals include fear of contracting HIV, discrimination and stigmatization by the public, multiple bereavements, and sexual curtailment. Moreover, stress is likely to increase following HIV infection. Lack of empathy and support from the general and medical communities, disbelief in medical technology to find a cure or preventive measure, and financial difficulties make up only a few burdens faced by seropositive individuals (Cecchi, 1984; Livingston, 1988).

Stress models of HIV disease status are supported by research demonstrating that chronic stress causes changes in the body's neuroendocrine pathways. These changes are mediated primarily by the hypothalamic-pituitary-adrenal cortex axis and the sympathetic adrenal-medullary system. During periods of stress, the adrenal cortex secretes steroids, primarily cortisol, while the adrenal medulla
produces catecholamines. Both corticosteroids and catecholamines can produce secondary changes in immune functioning.

A group of researchers at the Center for the Biopsychosocial Study of AIDS (Antoni et al., 1990) recently has begun to examine the effects of hormones and catecholamines as they relate to stress and HIV. From their work, these investigators support the notion that two stress response patterns exist. The first pattern, referred to as "active coping," occurs when an individual has adequate coping resources to meet the demands of the stressor. Active coping is mediated by the sympathoadrenalmedullary system. Early evidence for this biologically mediated coping mechanism is based in Cannon's (1926) classic description of the flight or fight response.

During active coping, the perception of stress causes electrochemical changes in the brainstem. These changes result in sympathetic arousal, mediated by the autonomic nervous system, throughout the organism. During this arousal period, there is a release of epinephrine and norepinephrine by the adrenal medulla (Jemmott & Locke, 1984). Hyperarousal of the sympathoadrenalmedullary system results in elevated catecholamines and, subsequently, immunosuppression (Antoni et al., 1990; McCabe & Schneiderman, 1985). Recent investigations have
demonstrated that catecholamine receptors are present on lymphocytes (Borysenko & Borysenko, 1982). Stimulation of these receptors decreases lymphocyte metabolism, proliferation, and antibody secretion (Strom, Lundin, & Carpenter, 1977) and reduces NK cell cytotoxicity (killing ability) (Antoni et al., 1990).

A second pattern of behavioral responding occurs when no coping response is available when confronting a stressor. Evidence for this type of responding can be found in Selye's (1950) original GAS work. When the individual does not have adequate coping resources, hypervigilance and/or withdrawal may occur. These behaviors activate the hypothalamic-pituitary adrenocortical (HPAC) system. Activation of this system begins as the cortex and limbic system signal the hypothalamus to release corticotropin releasing factor (CRF). CRF causes the pituitary to release adrenocorticotropic hormone (ACTH) which, in turn, signals the release of corticosteroids by the adrenal cortex (Jemmott & Locke, 1984).

Specific immune changes parallel the release of CRF, ACTH, and corticosteroids (Antoni et al., 1990). CRF, using cyclic AMP as its second messenger, inhibits the cytotoxicity of NK cells. ACTH disrupts the intracellular level of calcium in T-lymphocytes, impairing their responsiveness. Corticosteroids cause an array of
alterations in immune capacity. Cortisol binds with receptors located on T-lymphocytes. Chronic elevations of cortisol and other corticosteroids lead to thymus shrinkage, a decrease in circulating T-lymphocytes, and a reduced ability of lymphocytes to ward off infectious agents (Claman, 1972; Livingston, 1988).

Markham, Salahuddin, Veren, Orndorff and Gallo (1986) provided laboratory evidence that stress-related hormones can lead to HIV susceptibility. These researchers supplemented in vitro cultures of normal T-lymphocytes with corticosteroids (mainly hydrocortisone). The cultures were then exposed to HIV. The ability of HIV to infect cells was enhanced significantly when the cultures contained corticosteroids. These authors concluded that individuals under stress may be more vulnerable to HIV infection (Markham et al., 1986).

Gorman et al. (1991), however, failed to find any association between stress-related hormones and immune functioning in 112 HIV positive males. Subjects collected urinary free cortisol samples over a 24-hour period. During the same period, they underwent a battery of medical and psychological tests. These investigators examined correlations between urinary cortisol levels and CD4s, CD8s, and CD4/CD8 ratios. No significant findings were observed.
In the Gorman et al. (1991) study, however, cortisol was significantly correlated with physician-rated HIV disease progression and number of HIV-related symptoms. Cortisol levels also significantly correlated with scores on both the Hamilton anxiety and depression scales in positive directions. Although no direct link between stress and immune functioning was obtained, these authors concluded that stress (via stress-related hormones, i.e. cortisol) may impact the global health status of HIV patients. Moreover, the authors strongly encouraged expeditious delivery of mental health services to HIV patients (Gorman et al., 1991).

By comparing HIV to other retroviruses, the impact of stress on HIV disease progression has been addressed hypothetically. Kiecolt-Glaser and Glaser (1988) hypothesized that stress affects HIV progression similar to the way it affects herpesvirus progression. That is, stress triggers the virus to change from a latent to a symptomatic clinical state. Data has suggested that academic stress increases antibodies to the Epstein-Barr virus, herpes simplex virus type 1, and cytomegalovirus among medical students (Glaser et al., 1985). Kiecolt-Glaser and Glaser (1988) also reported evidence that herpesvirus antibody production can result from multiple sources of stress including marital separation and caregiving.
In a similar vein, Robertson et al. (1990) examined the correlation between psychological distress and herpes simplex in HIV patients. These investigators found significant positive correlations between level of distress and titers to herpes simplex among their 50 HIV positive subjects. They concluded that reactivation of herpes simplex facilitates HIV disease progression. No studies, however, have directly addressed the hypothesis that stress disinhibits the latent nature of HIV.

A noteworthy stressor encountered by all HIV positive individuals concerns the HIV testing and notification process. Anticipating the results of one's antibody status following HIV testing can create inordinate levels of stress. Ironson et al. (1990) examined relations between immune parameters and distress following HIV testing. A significant negative correlation was found between anxiety at the time of notification and NK cytotoxicity one week later in 14 HIV positive men. In addition, changes in scores on the avoidance scale of the Impact of Events Scale (Horowitz, Wilner, & Alvarez, 1979) also negatively correlated with NK cytotoxicity over the same period. These authors concluded that individual differences in stress reactions to HIV notification may affect HIV immunocompetence (Ironson et al., 1990).

Research supporting the hypotheses that stress and mood influence the clinical course of HIV is promising.
However, most researchers would agree that neither stress nor mood alone is sufficient to explain psychologically mediated HIV susceptibility and progression. Multifactorial models provide a comprehensive approach in describing the impact of psychological variables on the HIV clinical spectrum (Livingston, 1988). In addition to stress and mood, multifactorial models present potential moderating variables associated with illness. Among the most frequently mentioned moderators of HIV are social support, coping style, and personality.

Social Support

Social support as an intermediate or mediating variable has received a great deal of attention in health psychology research. Evidence that social support "buffers" the effect of stress on health, both physical and mental, has been well documented in the literature (Cohen & Wills, 1985). Only a few studies, however, have examined the potential influences of social support on HIV symptomatology.

Zich and Temoshok (1987) examined the direct effects of social support on HIV-related symptoms. In their cross-sectional study of over 100 HIV-positive individuals, these investigators reported that the total availability of social support had a significantly negative correlation with reported physical symptoms. Physical symptoms were divided into two categories, "hard" symptoms (e.g.,
infections and anemia) and "soft" symptoms (e.g., insomnia and weight loss). Both categories were associated with a lack of social support availability (Zich & Temoshok, 1987).

In the previously discussed stress model by Livingston (1988), the importance of social support briefly is considered. A number of variables are purported to comprise the Filter Resource Capability System (FRCS), including social support. Although the ability of social support to serve as a stress buffer has been supported elsewhere, Livingston (1988) offers no empirical data to demonstrate the importance of social support in HIV infected individuals.

In their preliminary results, Solomon and Temoshok (1987) reported data linking social support and AIDS progression. These investigators compared the availability of social support in a deceased group of AIDS patients to a surviving group. Scores were obtained on a number of social support dimensions. Survivors had higher levels of available problem-solving assistance than deceased victims. This finding was representative only of Pneumocystis carinii pneumonia patients. No differences in mortality rates attributable to social support were found for Kaposi's sarcoma or ARC patients (Solomon & Temoshok, 1987).
Reillo (1990) also considered social support an important prognostic indicator in a study of 58 AIDS patients. In this study, the author attempts to construct a profile of AIDS patients at risk for early mortality. Lack of social support, in combination with African-American race and a high level of psychological distress, was highly predictive of demise at 12 months among the subjects (Reillo, 1990).

Coping

Coping and personality style both have received empirical support as moderating variables and stress buffers in HIV disease (Livingston, 1988; Solomon, Temoshok, O'Leary, and Zich, 1987; Zich & Temoshok, 1987). The concept of "hardiness" as both a personality trait and coping strategy has been addressed in a few HIV studies. Kobasa, Maddi, and Courington (1980) refer to hardiness as a combination of "challenge, commitment, and control." It is frequently defined as an enduring personality trait characterized by coping ability and adaptivity.

Solomon and Temoshok (1987) reported that hardiness was negatively correlated with total lymphocyte count in AIDS and ARC subjects. These authors speculated that deficits in hardiness are associated with a greater number of secondary infections. Additional significant findings were reported for subscales measuring commitment and control. Higher scores on the commitment scale predicted
survival among ARC patients. Likewise, mortality rates for AIDS patients were lower for individuals with higher control scores (Solomon & Temoshok, 1987). Temoshok, O'Leary, and Jenkins (1990) reported similar data for their 15 subjects. Hardiness composite score, in addition to positive affect and heart rate reactivity, significantly predicted AIDS survival time.

Fletcher et al. (1989) also assessed coping style and hardiness in 24 HIV positive males. They charted the onset of HIV-related illness in their subjects for 1 to 2 years. When compared, subjects who remained asymptomatic throughout the study had significantly higher active coping, overall hardiness, and hardiness-commitment scores than those who developed medical complications. From their results, Fletcher et al. (1989) concluded that psychosocial variables may be important contributing factors in HIV illness progression.

In their prospective studies on immune changes and psychological distress following HIV status notification, Antoni et al. (1990) assessed coping strategies. Subjects completed the COPE inventory (Carver, Scheier, & Weintraub, 1989) at baseline. These investigators particularly were interested in denial as a coping mechanism. Scores on the denial subscale of the COPE were correlated significantly with lymphocyte proliferation responses to phytohemagglutinin antigen (Antoni et al., 1990). These
findings suggest that denial may constitute a poor coping strategy when preparing for serostatus notification following HIV testing.

**Personality**

In the ongoing Biopsychosocial AIDS Project, researchers are studying the association between personality variables and the HIV clinical spectrum (Solomon & Temoshok, 1987). Hypotheses are emerging that suggest an "immunosuppression-prone" personality exists. Profiles of these individuals emphasize "compliance, conformity, self-sacrifice, denial of hostility or anger, and non-expression of emotion" (Solomon & Temoshok, 1987). This personality type may resemble the "Type C" personality which predicts susceptibility to cancer and a relatively poor prognosis (Morris, 1982). Personality theories concerning immunosuppression are purely speculative at this point. No research supporting these theories with reference to HIV susceptibility has been reported.

**Relevant Treatment Outcome Research**

Clinical researchers have demonstrated that immune functioning can be enhanced by psychological treatments (Jemmott & Locke, 1984; Kiecolt-Glaser, 1988). Thus, mental health professionals have been quick to recognize the potential efficacy of psychological interventions in the treatment of HIV. Treatments for HIV have ranged from group therapy to cognitive-behavioral therapy to
metaphysical and alternative therapies (Badgley, 1987; Hand, 1989; Kelly & St. Lawrence, 1986). Although these treatments are becoming routine, reported treatment outcome studies have been sparse.

A group of researchers at the Center for the Biopsychosocial Study of AIDS in Miami recently began a series of studies on the efficacy of stress reduction as a potential treatment for HIV illness (Antoni et al., 1990). These investigators are examining the effects of cognitive-behavioral therapies, stress management, and aerobic exercise training on immunocompetence in individuals with HIV. Preliminary results suggested that both seropositive and seronegative individuals had increased T-helper counts following 10 weeks of aerobics training. In addition, aerobics significantly reduced psychological distress in HIV-infected men compared to nontreatment seropositive controls (Antoni et al., 1990). Results of the stress management condition are forthcoming.

Additionally, the same group of researchers found that these psychotherapeutic treatment strategies buffered the affective distress and immune decrements associated with HIV testing (Antoni et al., 1991; LaPerriere et al., 1990). In a study of 50 HIV positive men, individuals who participated in aerobics training had neither marked emotional distress nor significant deficiency in NK cell count upon serostatus notification. Seropositive controls
who received no treatment, however, displayed clinical levels of anxiety and depression as well as a significant reduction in NK cells following notification of their HIV status (LaPerriere et al. 1990).

Antoni et al. (1991) reported similar findings with a stress management intervention. Forty-seven men either received an assessment-control condition or stress management condition prior to notification of their HIV status. In the treatment condition, cognitive-behavioral strategies (assertiveness training, cognitive restructuring, relaxation, and behavioral rehearsal) were implemented. Subjects in the treatment condition demonstrated significant elevations in CD4 and NK cell counts and in proliferative responses to phytohemagglutinin at postnotification of HIV status. HIV positive subjects in the control condition significantly differed from experimental subjects and displayed decreases in immune parameters following notification. In addition, a reduction in depression was correlated with relaxation practice among experimental subjects. These authors concluded that stress management buffered the emotional reaction and immunological compromise associated with HIV testing and notification (Antoni et al., 1991).
PURPOSE OF THE STUDY

Research examining psychosocial factors as they relate to HIV is in its infancy. Many of the existing studies rely heavily on data concerning immune patterns in non-HIV populations. From the available data, however, preliminary evidence suggests that potential psychological co-factors in HIV disease may exist. Stress, coping, social support, and mood constitute the most empirically supported co-factors thus far. A few authors (Antoni et al., 1990; Livingston, 1988) have offered hypothetical models to illustrate the relations among some of these variables and HIV. However, no study has attempted to investigate these variables in an integrated fashion.

In the present study, a model (see Figure 1) was proposed based on previous data. Major stress (e.g., job loss, death of a loved one) and minor stress (e.g., car trouble, argument with spouse) were included as two separate constructs in the proposed model. Contemporary stress researchers recognize this conceptual delineation and have reported that major and minor stress differentially impact health status. Data have been offered to suggest that minor stress has a greater impact on illness fluctuations than major stress (Brantley & Jones, 1989, Delongis et al., 1982).

The combined impact of stress and social support on mental and physical well-being, commonly referred to as the
"buffering hypothesis," has been given noteworthy attention in health psychology literature (Cohen, 1988; Cohen & Wills, 1985). Likewise, coping style may alter the impact of stress (Lazarus & Folkman, 1984). Coping and social support, therefore, were hypothesized to be possible mediators of stress in the proposed model.

Mood (or distress) has been considered an outcome or function of stress, social support, and coping (Cohen & Willis, 1985; Folkman & Lazarus, 1984). In contrast, current studies on HIV have indicated that mood may be a predictor of HIV disease status (e.g., Ostrow et al, 1989). Thus, mood has been included as an intermediate variable in the proposed model.

In the existing studies of psychological influences on HIV, a number of methodological problems are evident. First, sample sizes are small in many of the studies. Second, virtually all of the subjects have been young adult, homosexual/bisexual males. Given that HIV prevalence rates are decreasing in homosexual males while rising in other risk groups (Nash & Said, 1992), the generalizability of these data is limited. Third, most of research has been conducted in large U.S. cities where the availability of resources for HIV and prevalence rates are greatest. Finally, a few studies have failed to discover significant links between HIV disease with stressful life events (Coates, McKusick, Kuno, & Stites, 1989; Kessler et
al., 1991), mood (Van Griensven et al., 1989), or social support (Van Griensven et al., 1989). The present study attempted to address the methodological shortcomings of previous studies by including a heterogenous sample of adequate size.

In summary, the primary purpose of the present study was to provide empirical support for the proposed model and thus create a broader understanding surrounding the links among psychosocial co-factors and HIV disease. From the study, the following questions were addressed:

1. Does stress impact HIV disease status? It was hypothesized that stress levels significantly would predict HIV disease parameters.
2. Do major stress and minor stress differentially impact HIV disease status? It was hypothesized that major stress and minor stress, separately, would contribute significant amounts of variance to HIV disease parameters.
3. Does social support impact HIV disease status? It was hypothesized that social support availability significantly would predict HIV disease parameters.
4. Does coping impact HIV disease status? It was hypothesized that problem-focused coping significantly would predict HIV disease parameters.
5. Does mood impact HIV disease status? It was hypothesized that negative affective states significantly would predict HIV disease parameters.
6. Do stress, social support, and coping impact mood among individuals with HIV? It was hypothesized that stress levels, social support availability, and problem-focused coping style would contribute significant amounts of the variance to affective distress.

7. What are the interrelationships among stress, social support, coping, mood, and HIV disease status? After controlling for degree of disease progression, it was hypothesized that the proposed structural equations model (see Figure 1) would fit the data significantly.
Figure 1. Structural Model of Psychosocial Co-factors and HIV Disease
METHODS

Subjects

One hundred HIV-positive volunteers were recruited from the Early Intervention Clinic at Louisiana State University Medical Center-Baton Rouge and Los Angeles County-University of Southern California Medical Center's AIDS clinic to participate in the study. All adult, registered patients at the clinics were eligible to participate. Patients who were unable to read English or who had a history of dementia or psychosis were excluded from the study. Nearly all patients scheduled to have lab work completed on days when experimenters were present were invited to participate. While exact numbers were not documented, only a small percentage of available patients were excluded or refused to participate.

Demographic data on subjects are summarized in Tables 1 and 2. Eighty male and 20 female adults completed the study. Mean age was 37, with a range of 18 to 54 years. Racial breakdown occurred as follows: 59% African-American, 33% Caucasian, 7% Hispanic, and 1% Native-American. Years of education ranged from six to 21, with a mean of 12. The sample was of low socioeconomic status, with 86 percent of the sample having a yearly income less than $10,000.

Sample demographics were highly similar across data sites for age, education, and socioeconomic status (see Table 2). Two noteworthy differences were observed with
regard to sex and race. A larger percentage of women were included in the Baton Rouge sample while a substantial number of Hispanics participated in the Los Angeles sample. In addition, the majority of subjects were from Baton Rouge (n=77, versus n=23 from Los Angeles).

All subjects were ambulatory outpatients, receiving medical treatment for HIV. A wide range of HIV-related illness was displayed among the subjects. The average length of time since testing HIV-positive was 25.4 months, with a range of zero to 120 months. CD4 counts ranged from two to 1089, with a mean of 356.

Measures

The Social Readjustment Rating Scale

(SRRS; Holmes & Rahe, 1967). The SRRS is a 43-item self-report questionnaire designed to assess the degree of adjustment associated with recent major life events. A weighting is assigned to each item, a priori, according to the amount of life change (positive or negative) associated with the event. Weightings are summed to generate the SRRS total score. The weightings were derived from a series of scaling studies on a sample of 394 subjects with a wide range of demographics. Subjects' ratings of the events yielded a high coefficient of concordance (W=.48, p=.0005).

The SRRS is the oldest and one of the most frequently employed major stress scales. A great deal of evidence
supporting its psychometric quality has been offered (Miller, 1989). In addition, research has indicated that elevated scores on the SRRS predict the onset of various medical and psychiatric disorders (Miller, 1989).

The Weekly Stress Inventory

(WSI; Jones & Brantley, 1989). The WSI is an 87-item, self-report scale measuring stressful events a person is likely to experience throughout the week. Items are rated on a Likert-type format, ranging from 0 ("did not occur") to 7 ("most stressful"). Three scores are generated from the WSI. These include an Event score (WSI-E; the number of items endorsed), an Impact score (WSI-I; the sum of all item ratings), and an I/E ratio score. The WSI-I was used in the present study.

Adequate concurrent validity (.61) and test-retest reliability (.6) of the WSI have been demonstrated. A principal components analysis yielded one construct with an alpha coefficient of .95, indicating that the items consistently measure the same construct ("stress") (Jones & Brantley, 1992). In a study of 100 cardiac patients, the WSI significantly predicted pain and physical symptoms (Mosley & Brantley, 1989; unpublished data).
The Interpersonal Support Evaluation List

(ISEL; Cohen, Mermelstein, Kamarck, & Hoberman, 1985). The ISEL is a 40-item, self-report questionnaire that assesses an individual's perception of social support availability. Responders rate on a 4-point Likert-type scale how accurately each item describes their access to social support. The ISEL's development was influenced by the current notion that social support buffers the effect of stressful events (Cohen & Willis, 1985).

A total aggregate score and four subscale scores (appraisal, belonging, self-esteem, and tangible) are derived from the ISEL. Only the total score will be incorporated in the present study. Validation studies have indicated adequate concurrent ($r = .46$) and discriminant ($r = -.64$) validities (Cohen & Willis, 1985). Reliability of the ISEL is good, with test-retest coefficients averaging .87 for the total aggregate score (Cohen, Mermelstein, Kamarck, & Hoberman, 1985).

Ways of Coping Questionnaire-Revised

(WCQ-R; Folkman & Lazarus, 1985). The WCQ-R is a 66-item questionnaire designed to assess coping strategies used during specific stressful events (e.g., medical procedures, exams). The authors conceptualize coping as a changing process, suggesting that coping strategies vary within a given individual as result of his/her appraisal of
stress. The questionnaire was designed to assess the dynamics of coping rather than static coping styles or traits (Folkman & Lazarus, 1985). A factor analysis on the WCQ-R yielded eight factors that represent theoretically distinct, coping strategies (e.g., emotion-focused coping, problem-focused coping, social support utilization). From these factors, eight subscales were developed. In the present study, only the Problem-focused Coping subscale was used, as it demonstrates the best reliability (alpha=.85).

Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977). The CES-D is a 20-item questionnaire that evaluates depression, with an emphasis on measuring affective symptomatology. Responses are scored on a 4-point Likert-type scale according to weekly occurrence ("0-rarely or none of the time/less than a day" to "3-most or all of the time/5 to 7 days").

The CES-D has demonstrated good reliability (split-half correlations: .77-.85; Spearman-Brown coefficients: .87-.92). Validation studies have been met with moderate results. Correlations with other depression measures have ranged from .37 to .90. Interviewer ratings correlate from .46 to .53 with the CES-D (Radloff, 1977).
**State-Trait Anxiety Inventory**

(STAI; Spielberger, 1983). The STAI is a widely used self-report questionnaire measuring anxiety symptoms. Two forms, each with 20 items, comprise the STAI. The State-Anxiety (STAI-S) scale assesses an individual’s current level of anxiety at the time of administration, while the Trait-Anxiety (STAI-T) scale evaluates the individual’s general level of anxiety. The STAI-T scale was used in the present study.

Extensive findings on the stability and validity of the STAI-T scale have been reported (Speilberger, 1983). Reliability coefficients (test-retest) ranged from .70 to .76. A median alpha coefficient of .90 indicated that the STAI-T scale is internally consistent. Average correlation with other anxiety measures was .80, suggesting good concurrent validity.

**Symptom Checklist for HIV Infection**

(SCL-HIV; Gorman et al., 1991). Recently, a group of medical researchers designed a checklist to assess the degree of HIV symptomatology among their subjects. The 20-item checklist is composed of signs, symptoms, and common infections associated with HIV. Each item is given equal weight and a frequency score is generated. No data have been reported on the measurement properties of the SCL-HIV.
T-Cell Count.

As part of their medical protocol, HIV patients undergo routine laboratory analyses of immune functioning. CD4 counts are considered the best indicator of disease progression in HIV and are monitored closely by medical professionals in order to regiment treatments (Bartlett & Finkbeiner, 1991). HIV-negative individuals have CD4 counts averaging 1000 cells per milliliter of blood. Following HIV infection, CD4 counts decrease at a rate of approximately 85-100 cells per year. HIV-related symptoms are likely to develop when an individual’s count drops below 300. CD4 counts typically range between 50-100 for AIDS patients (Bartlett & Finkbeiner, 1991).

Procedure

HIV-positive adults who were registered patients at the data collection sites were eligible to participate. During a clinic appointment when HIV patients were having blood drawn for T-cell counts, attending hospital staff informed potential subjects about the study and asked if they were interested in participating. If subjects volunteered, they signed informed consents explaining the nature of the study and allowing the experimenter access to their medical charts. Medical charts were reviewed to document laboratory results and other medical data (e.g., medications, diagnoses).
All data were collected on the day of the clinic appointment. A graduate research assistant gathered a brief demographic and medical history. Following the history, subjects completed questionnaires on stress, social support, coping, and mood. As part of their routine medical protocol, subjects had blood drawn and sent to a laboratory. Immune parameters were analyzed from the blood sample, and an immune profile was later documented in each subject's medical chart. Subjects received $5.00 for participating.
RESULTS

Descriptive Statistics

Descriptive statistics were calculated on demographic and experimental variables. Profiles of the sample's demographic and experimental characteristics are presented in Tables 1, 2, and 3. Mean scores of the psychosocial variables revealed significant affective distress in the sample. The mean CES-D depression score (22.4) is clearly above the cut-off score (16) indicative of clinical depressive symptomatology. Similarly, STAI-T mean anxiety score (45.7) approached the 90th percentile for normal adults.

Several relations between demographic and outcome variables were statistically significant. T-tests revealed a few significant sex differences. Males had been diagnosed HIV-positive longer than females \[ t(99)=2.49, p=.014 \], and females had higher total T-cell counts \[ t(95)=-1.99, p=.049 \]. Female subjects also reported greater trait anxiety \[ t(95)=-2.40, p=.018 \].

T-tests were conducted to examine racial differences across all experimental variables. Race was dummy-coded, dividing the sample into two groups - Caucasians \( (n=33) \) and racial minorities \( (n=67) \). Caucasians reported higher major stress scores \[ t(100)=2.53, p=.013 \], endorsed more HIV-related symptoms \[ t(97)=2.85, p=.005 \], and had been
Table 1

Demographic Variables: Descriptive Statistics

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Table 2
Sample Demographics by Site

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**University of Southern California Medical Center:**

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</tr>
<tr>
<td># of HIV Symptoms (SCL-HIV)</td>
<td>4.1</td>
<td>0-13</td>
<td>3.1</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
diagnosed HIV-positive longer \( t(99) = 2.58, \ p = .011 \) than racial minorities.

**Correlational Statistics**

Pearson product-moment correlations were generated for age and education across all experimental variables. Older subjects had been diagnosed HIV-positive longer \( r = .20, \ p = .049 \) and had fewer CD4 cells \( r = -.24, \ p = .020 \). Years of education was significantly correlated with anxiety \( r = -.23, \ p = .026 \) and depression \( r = -.26, \ p = .010 \), both in negative directions. Years of education also significantly correlated with illness duration \( r = .22, \ p = .026 \).

A correlational matrix of Predictor by Outcome variables was generated and the results can be found in Table 4. Only one predictor variable, problem-focused coping, significantly correlated with total CD4 count \( r = -.214, \ p = .04 \) but in the unexpected direction. No other predictor variable significantly correlated with CD4 count. Problem-focused coping also significantly predicted HIV symptom count, but again in the unexpected direction \( r = .310, \ p = .002 \).

All remaining predictor variables correlated with HIV symptom count in statistically significant and expected directions. HIV symptoms positively correlated with both major stress \( r = .210, \ p = .039 \) and minor stress \( r = .238, \ p = .02 \). Social support correlated negatively with HIV
Table 4
Correlational Matrix of Predictor X Outcome Variables

<table>
<thead>
<tr>
<th></th>
<th>Total CD4 Count</th>
<th># of HIV Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td># of months HIV+</td>
<td>-.119</td>
<td>.181</td>
</tr>
<tr>
<td>Major Stress (SRRS)</td>
<td>-.039</td>
<td>.210*</td>
</tr>
<tr>
<td>Minor Stress (WSI)</td>
<td>-.038</td>
<td>.238*</td>
</tr>
<tr>
<td>Problem-focused Coping (WCQ-R)</td>
<td>-.214*</td>
<td>.310**</td>
</tr>
<tr>
<td>Social Support (ISEL)</td>
<td>.101</td>
<td>-.294**</td>
</tr>
<tr>
<td>Anxiety (STAI-T)</td>
<td>.132</td>
<td>.275**</td>
</tr>
<tr>
<td>Depression (CES-D)</td>
<td>.003</td>
<td>.290**</td>
</tr>
</tbody>
</table>

*p<.05.  **p<.01
symptoms \( r = -0.294, \ p = 0.004 \). HIV symptom count correlated positively with both mood variables, anxiety \( r = 0.275, \ p = 0.007 \) and depression \( r = 0.290, \ p = 0.004 \)

Structural Analyses

To further test main hypotheses of the present study, a linear structural analysis was performed on the model proposed in Figure 1 (see page 33). The EQS program (Bentler, 1989) was used to conduct the analysis. In the model, an arrow indicates the direct effect of one variable on another. Direct effects were represented statistically by path coefficients, i.e. beta weights (B).

From the analysis, indirect and total effects were calculated. An indirect effect is the influence of one variable on another that is transmitted via an intermediate variable(s). Numerically, it is the product of the path coefficients that make up the nondirect links between two variables. For example, in the proposed model, the indirect effect of social support on HIV disease = (social support’s direct effect on mood) \( \times \) (mood’s direct effect on HIV disease). Total effects then were calculated by summing direct and indirect effects (Bentler, 1990).

Data have indicated that duration of infection is the only consistent predictor of HIV disease progression (McDougal et al. 1989). Therefore, illness duration (number of months HIV-positive) was entered first into the
model to control for current stage of HIV disease progression. Surprisingly, however, duration of infection was not significantly correlated with either CD4 count \( r = -0.119, \text{ns} \) or HIV symptom count \( r = 0.181, \text{ns} \).

Following illness duration, variable entry into the model occurred according to the following logic: Given that minor stress may exhibit a greater influence on disease than major stress (Delongis et al, 1982), major stress preceded minor stress in building the model. Because both have been observed to mediate stress effects, coping and social support followed stress and were entered simultaneously into the model. Anxiety and depression may result from a combination of inordinate stress, faulty coping strategies, and/or lack of social support (Cohen & Wills, 1985). Thus, anxiety and depression were collapsed to form the latent variable "mood" which was entered next into the model. Finally, symptoms, CD4 count, and CD4/CD8 ratio were combined to form "HIV disease," the model's latent outcome variable.

To test how well the overall model fit the data, four indices were generated. The indices suggested the data did not fit the model well - Chi-square \( (23, N = 100) = 47.28, p > .002 \), Bentler/Bonnet Normed Fit Index = .87, Bentler-Bonett Nonnormed Fit Index = .85, and Comparative Fit Index = .93. The model's latent outcome variable, "HIV disease", received statistically significant loadings from total CD4
count and CD4/CD8 ratio (−.89 and −.84, respectively) but not from HIV symptoms (−.49).

A second model (see Figure 2) was constructed omitting the two T-cell outcome variables. HIV symptom count comprised the only disease outcome variable in the second model. Additionally, because illness duration did not predict the latent variable "HIV disease" in the original model or correlate individually with CD4 count or symptoms, it was excluded as a control variable in the second model. The second model fit the data extremely well. All statistical fit indices highly supported the model - Chi-squared (5, N=100) = 1.396, p=.92; Bentler-Bonett Normed Fit Index = .99; Bentler Bonnett Nonnormed Fit Index = 1.0; Comparative Fit Index = 1.0. Direct effects and percent variances of the psychosocial variables on HIV symptoms are listed in Table 5. Direct effects and percent variances of stress, social support, and coping on mood are listed in Table 6.

In the second model, coping (direct effect = .263, p < .05) and mood (direct effect = .226, p < .05) significantly impacted HIV symptoms. Neither of the stress variables nor social support predicted HIV symptoms. However, minor stress (direct effect = .424, p<.01) and social support (direct effect = −.428, p < .05)
demonstrated significant effects on the latent variable, "mood." Coping and major stress had no direct effects on mood.
Figure 2. Structural Model of Psychosocial Co-factors and HIV Symptoms
### Table 5

**Direct Effects and Variances of Psychosocial Co-factors on HIV Symptoms in HIV Symptom-Only Structural Model**

<table>
<thead>
<tr>
<th>Variable</th>
<th>direct effect</th>
<th>%variance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major Stress</td>
<td>.088</td>
<td>0.7</td>
</tr>
<tr>
<td>Minor Stress</td>
<td>-.073</td>
<td>0.5</td>
</tr>
<tr>
<td>Problem-focused Coping</td>
<td>.263*</td>
<td>6.9</td>
</tr>
<tr>
<td>Social Support</td>
<td>-.130</td>
<td>1.7</td>
</tr>
<tr>
<td>Mood</td>
<td>.226*</td>
<td>5.1</td>
</tr>
<tr>
<td>(Total)</td>
<td></td>
<td>14.9</td>
</tr>
</tbody>
</table>

*p<.05*
Table 6

Direct Effects and %Variances of Stress, Social Support, and Coping on Mood in HIV Symptom-Only Structural Model

<table>
<thead>
<tr>
<th>Variable</th>
<th>direct effect</th>
<th>%variance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major Stress</td>
<td>.111</td>
<td>1.2</td>
</tr>
<tr>
<td>Minor Stress</td>
<td>.424*</td>
<td>18.0</td>
</tr>
<tr>
<td>Problem-focused Coping</td>
<td>-.113</td>
<td>1.3</td>
</tr>
<tr>
<td>Social Support</td>
<td>-.428*</td>
<td>18.3</td>
</tr>
<tr>
<td>(Total)</td>
<td></td>
<td>38.8</td>
</tr>
</tbody>
</table>

*p<.01
DISCUSSION

The results suggest the originally proposed structural model did not represent the data in a statistically significant fashion. Despite failure of the model, Pearson product-moment correlations revealed a strong association between each of the proposed psychosocial co-factors and number of HIV-related symptoms. This trend, however, was not observed in relations among the same psychosocial variables and T-cell counts. With these results in mind, a second model was constructed to illustrate the impact of the psychosocial variables on HIV symptom count alone, excluding the immunological measures. The second model was highly significant with psychosocial co-factors accounting for 15% of the variance in HIV symptoms.

Lack of significance in the original model can be explained theoretically and statistically. Theoretically, the manifest variables (HIV symptom count and T-cell counts) selected to constitute the latent variable "HIV disease" may not have been the best or only choices. Pathophysiology of HIV disease is highly variable, complex, and unpredictable (Rogers & Masur, 1989). Additional disease manifestations (e.g., natural killer cell count and opportunistic infections not included in the SCL-HIV) may be equally important in describing HIV illness as a construct. Bentler and Chou (1987) have emphasized a major theoretical limitation of structural modeling is that key
variables often are omitted when designing a model. Breckler (1990) encourages the exploration of alternate models when using structural modeling procedures.

In the original model, CD4 count (-.89) and CD4/CD8 ratio (-.84) loaded highly on the latent variable "HIV disease" whereas symptoms (.49) did not. Statistically, the model attempted to maximize the variance of "HIV disease" as a construct. As a result, the model mainly predicted T-cells. HIV symptoms were not captured well by the latent variable "HIV disease" and the overall model was invalidated.

Although none of the psychosocial variables were linked to T-cell counts, a few demographic variables correlated significantly with T-cells. Lower T-cell counts were associated with increased age and being male. These findings were not surprising given that males and older subjects in this sample had significantly longer illness duration. Additionally, age is associated with a poorer prognosis and accelerated HIV disease activity (Cohen, Salde, & Volberding, 1990). Although sex is considered to be a potential co-factor in HIV by some authors (Rothenberg, Woelfel, Stoneburner, Milberg, Parker, & Truman, 1987), the relatively small female sample size (n=20) does not permit valid interpretations of sex differences from the present study.
Collectively, health professionals appear perplexed when discerning what factors predict HIV disease course. No consistent pattern of HIV disease progression has been defined (Gorman & Kertzner, 1990). A similar enigma challenging researchers is identifying co-factors that predict mortality and/or longevity. Researchers are beginning to explicate reasons why long-term AIDS survivors remain alive and healthy despite depleted T-cells and over a decade of seropositivity (Solomon, Temoshok, O’Leary & Zich, 1987). Numerous co-factors including nutrition, repeated exposure to HIV, presence of concomitant viruses, genetic make-up, and psychosocial co-factors have been hypothesized with varying degrees of empirical support (Gorman & Kertzner, 1990; Livingston, 1988; Watson, 1990).

To date, data supporting an association between psychosocial co-factors and immunological markers in HIV remain inconclusive. Consistent with the present results, the majority of recently published studies have not supported a strong relationship between psychosocial co-factors and T-cell counts. Sahs et al. (1993) reported no significant effects for psychological distress or presence of comorbid psychiatric disorders on immune profiles in a cross-sectional study of 120 HIV-positive individuals. Two prospective studies, both with sample sizes over 100, also failed to find significant associations between multiple psychosocial variables and T-cell counts. Nevertheless,
both studies reported significant relations between the same psychosocial variables (i.e. stress, depression, anxiety, social support, hardiness, psychiatric symptomatology, grief, and hopelessness) and number of HIV symptoms (Perry, Fishman, Jacobsberg & Frances, 1992; Rabkin et al., 1991).

Of the psychosocial variables in the present study, coping and mood demonstrated the strongest association with HIV symptomatology. Significant influences of coping and mood on HIV symptoms were evidenced in both the correlational matrix and the symptom-only structural model. The impact of coping on HIV symptoms, however, occurred in the unexpected direction. That is, as problem-focused coping increased, so did the number of symptoms. One explanation for this finding may be the questionable validity of the subscale. Development and validation of the WCQ-R appear to be in the infancy stages. The Problem-focused subscale is only 12 items, and no information on its content validity is available. Additionally, no concurrent or discriminant validity studies on the WCQ-R or Problem-focused subscale have been reported.

Another explanation for the link between coping and HIV symptoms concerns what Bentler and Chou refer to as a "nonrecursive" model. In nonrecursive models, the directionality of influence between certain pairs of variables may be two-way. In the symptom-only model, the
connection between coping and symptoms may be more accurately represented by a double-headed arrow. The relationship may indeed be significant, with problem-focused coping being a response to exacerbations in HIV illness as well as an antecedent. Administration of WCQ-R supports this explanation. When responding, subjects were asked to rate how they coped with HIV specifically. They did not report on general coping style.

The impact of mood on HIV disease status has been documented in previous studies. In the present study, increased anxiety and depression were associated with higher HIV symptom counts. Although data supporting an association between affective distress and immune parameters remains inconclusive in the current literature (Mulder & Antoni, 1991), the present results add to the few available studies (Ostrow et al., 1989; Perry, Fishman, & Jacobsberg, 1992; Rabkin et al., 1991; and Temoshok, O’Leary, & Jenkins, 1990) suggesting mood significantly impacts HIV symptomatology. Future studies could expand current literature by including additional affective variables (e.g., hostility) and examining the differential effects of particular mood states on HIV disease.

The correlational matrix provided evidence that major stress, minor stress, and social support significantly correlate with the number of HIV symptoms. In the symptom-only model, however, none of these variables exhibited
significant effects on HIV symptomatology. It appears from the model that minor stress and social support have significant indirect effects on HIV symptoms via mood, as both minor stress and social support significantly impacted mood in the model. Thus, the impact of minor stress and social support on HIV symptoms may be substantial but is likely mediated by anxiety and/or depression.

The present findings revealed no differential effects of major stress versus minor stress on HIV disease status. However, minor stress had a significant impact on mood whereas major stress did not. This datum supports the notion that minor stress exhibits a greater influence than major stress on global health (Brantley & Jones, 1989; Delongis et al., 1982). Moreover, with the effect of social support on mood controlled, the effect of minor stress on mood remained significant in the symptom-only model. This finding suggests social support does not buffer the effect of stress on mood in HIV-positive individuals.

As expected, increased minor stress and decreased social support availability were significantly linked to elevations in anxiety and depression. Together, minor stress and social support accounted for 36% (18% each) of mood variance in the symptom-only model. Major stress and problem-focused coping, however, demonstrated no significant effects on mood. Therefore, the roles of major
stress and problem-focused coping in HIV-positive individuals' affective regulation remain unclear.

As previously mentioned, a concern when employing structural modeling is that important variables may be omitted that better explain the constructs. Numerous medical co-factors (e.g., nutrition, viral load, presence of other illnesses) have been purported to contribute to the HIV illness spectrum (Watson, 1990). There may be additional psychosocial co-factors, aside from the ones in the present study, that are paramount in describing the psychosocial-HIV nexus.

High risk behaviors constitute an important category of psychosocial influences on HIV not included in the present study. Recently, data have supported the idea that increased stress is associated with unsafe sexual practices (Folkman, Chesney, Pollack, & Phillips, 1992; Coates, McKusick, Kuno, & Stites, 1989) and intravenous drug use (Hartgers, Vanden-Hoek, Coutinho, & Vander-Pligt, 1992) among HIV-positive individuals. That is, when stressed, individuals engaged in high risk behaviors more frequently. Collectively, these studies indicated high risk behaviors may mediate stress' impact on repeated exposure to the virus. Repeated exposure to additional and perhaps more virulent HIV strains is considered a possible co-factor in the progression of HIV disease (Sonnabend, Witkin & Purtilo, 1984).
CONCLUDING REMARKS

The present results add to a growing literature suggesting certain psychosocial co-factors contribute to the progression of HIV disease. These findings have important implications for the direction of future psychosocial HIV research. Biopsychosocial models provide a valuable heuristic when studying illness. The structure of the present model was theoretically derived and empirically supported. As the connection between psychosocial co-factors and HIV disease status becomes more evident, investigators should attempt to identify alternate models and additional mechanisms for this nexus.

Psychoneuroimmunological research has provided strong evidence for the interrelationships among various psychological states, neurochemicals, and immunological parameters in normal human and animal subjects (Ader, 1991). Based on these data, Solomon, Kemeny, and Temoshok (1991) authored a comprehensive review, hypothesizing probable psychoneuroimmunological pathways in HIV. Evidence supporting an association between biological markers of psychological distress (e.g., corticosteroids, neuropeptides, and catecholamines) and HIV pathophysiology are sparse but are beginning to emerge in the literature (Antoni et al., 1990; Gorman et al., 1992; Gorman et al.). These data increase objectivity and provide stronger support for biopsychosocial models of HIV. Given the
present findings, researchers should continue to investigate HIV psychoneuroimmunology as it relates to HIV symptom expression.

As health professionals recognize biopsychosocial models of HIV with more confidence, implications for mental health services in the treatment of HIV become paramount. Applied researchers are beginning to offer evidence for the efficacy of psychotherapeutic approaches in managing HIV disease (Antoni et. al., 1991; Coates, McKusick, Kuno, & Stites, 1989; LaPerriere et al., 1990). Given the chosen psychosocial variables in the present study, stress management and support groups may be important treatment strategies for combatting the high morbidity of affective disorders in individuals with HIV. Moreover, the present findings support the notion that mood management and alterations in coping style may attenuate HIV symptomatology.

Perhaps the most cardinal contribution of the present study concerns the sample demographics. The sample overincluded females (20% of the sample) and African-Americans (59% of the sample), relative to the U.S. prevalence of HIV-positive individuals in these groups (11% and 30%, respectively). Hispanics (7% of the sample) also were included but did not match the U.S. prevalence of 17%. Ninety-five percent of the sample had annual incomes totaling less than $10,000 per year.
These sample characteristics have several important implications. First, HIV research in general has been criticized for including primarily Caucasian, socioeconomically stable, homosexual/bisexual males. HIV studies that include minorities, females, and the economically challenged are a rarity. Demographic heterogeneity in the sample makes the results more generalizable and represents a large portion of the U.S. HIV population long ignored by research efforts.

Second, although insufficient and unbalanced sample sizes prevented appropriate statistical comparisons of race and sex in the present study, data identifying race and sex as important co-factors in predicting survival among HIV-positive individuals are emerging. Poorer prognosis among AIDS patients has been shown to be associated with African-American race (Reillo, 1990; Rothenberg, Woelfel, Stoneburner, Milberg, Parker, & Truman, 1987) and being female (Rothenberg et al., 1987). Future studies should continue to address ethnic and sex differences as they relate to the progression and treatment of HIV disease.

Finally, as a result of education and prevention efforts among the gay/lesbian community, the prevalence of HIV infection among homosexual males relative to other risk groups is declining (Nash & Said, 1992; Winkelstein & Padian, 1987). Unfortunately, infection rates among the other risk groups that include minorities, women, and the
economically challenged in large proportions are rising dramatically (Nash & Said, 1992). The present findings justify meeting the ever-present demands for increased research agendas, treatments, and prevention efforts targeting these special populations.

In summary, the present study supported the contention that psychosocial co-factors influence disease status in HIV-positive individuals. The results indicated psychosocial co-factors contributed to symptom expression but not immune functioning in HIV-positive individuals. Coping and mood were observed to have substantial direct impacts on HIV symptomatology. Stress and social support contributed to the overall health of HIV-positive individuals by their significant effects on mood. Given that major stress did not have significant direct or indirect effects on HIV symptoms, eliminating major stress from the model would improve its parsimony.

The results validated a model illustrating the interrelations among psychosocial co-factors and HIV. However, as statisticians have indicated, alternate models may explain the data equally well or better (Bentler & Chou, 1987). Future investigative efforts should continue to replicate the present results as well as look for alternate models to explain the links among psychosocial co-factors and HIV. Including other psychosocial predictors, such as frequency of high risk behaviors, may
account for additional variance in HIV disease. Disease parameters not included in the present study, such as natural killer cell count and opportunistic infections not captured by the SCL-HIV, may improve validity of the model as well.
REFERENCES


VITA

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Major Field: Psychology

Title of Dissertation: Psychosocial Co-factors and HIV Disease Status

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