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The Impact of Psychosocial Variables on Immune System Functioning in a Sample of HIV-Positive Males

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THE IMPACT OF PSYCHOSOCIAL VARIABLES ON IMMUNE SYSTEM FUNCTIONING IN A SAMPLE OF HIV POSITIVE MALES

by

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Gary K. Richey

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ABSTRACT

The Impact of Psychosocial Variables on
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by

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This study addressed the issue of the relationship between psychological well-being and immune function in a sample of HIV seropositive homosexual and bisexual males. A control group of HIV seronegative gay males was included. The study assessed the relationship between various psychological independent variables and immune system functioning over a 24-month time period for the seropositive subjects. Data on depression, coping style, psychosocial stress, and psychosomatic symptoms were collected at baseline, as well as data on depression at 12 months and CD4 counts at 6-month intervals over a 2-year period. Preliminary analyses comparing HIV seropositive to HIV seronegative subjects showed differences on four of eight coping style scales, as well as on all of the psychogenic attitudes scales reflecting stress levels.

There were no effects of eight coping styles on immune system functioning for the seropositives. However, there were significant

relationships among four of six psychogenic attitudes scales (chronic tension, premorbid pessimism, future despair, and somatic anxiety) and immune system functioning for the seropositives. There were also significant effects of three scales measuring psychosomatic symptoms (Allergic Inclination, Gastrointestinal Susceptibility, and Cardiovascular Tendency) for the seropositives. However, there was no effect of level of depression on immune system functioning.

The final chapter discusses the findings given the existing body of research. The emphasis is on the need to develop interventions targeting stress levels among persons with AIDS, as well as on conducting further research utilizing carefully constructed longitudinal research designs.

(140 pages)

CHAPTER I

INTRODUCTION

Background to the Problem

Acquired Immunodeficiency Syndrome (AIDS) is a disease which attacks the body's immune system and renders its victims vulnerable to opportunistic illnesses which would otherwise be nonthreatening. AIDS, initially identified in 1981 (Centers for Disease Control, 1981), has subsequently been traced back to 1979 (Curran, 1983). As of September, 1992, there were 242,146 reported AIDS cases, with 160,372 having resulted in death (CDC National AIDS Hotline, personal communication, October 26, 1992). Current projections are that there will be between 390,000 to 480,000 AIDS cases by 1993. It is also estimated that 1 to 2 million Americans have been infected with the human immunodeficiency virus (HIV) but have no symptoms of the illness. The number of affected individuals is constantly increasing, particularly in high-risk populations (as defined by past or present unprotected sexual activity or intravenous drug use). It appears that the absolute number of cases is more than doubling every 12 months, although many experts believe that the actual occurrence far exceeds reported incidence (Quadland & Shattis, 1987).

Although approximately three-quarters of all AIDS cases in the United States involve homosexual or bisexual men (Harowski, 1987; Quadland & Shattis, 1987), AIDS is definitely not restricted to this population. It is becoming clear that individuals who engage in

certain high-risk behaviors are at risk for AIDS, regardless of membership in any particular group.

When AIDS was first identified in 1981, it was poorly defined and understood only as "a cluster of rare diseases that had suddenly become alarmingly common in homosexual men" (Redfield & Burke, 1988, p. 90). By 1984, researchers at the Centers for Disease Control and at Walter Reed Army Medical Center had developed a more precise definition of the disease. AIDS was redefined as representing the culmination of a multi-stage process through which certain types of white blood cells are depleted. These cells are crucially important in the body's immune system, and are also involved in the process of T-cell activation. More specifically, they activate B-lymphocytes, which normally multiply and produce antibodies that can bind and inactivate infected cells. Consequently, the body's resistance to a variety of infections is reduced; in particular, the ability of the body to ward off attack by viruses, fungi, parasites, and bacteria is impaired (Redfield & Burke, 1988).

In order to develop an understanding of the linkage between psychosocial variables and immune system functioning, it is useful to explore the way in which the human immune system works. Generally speaking, the immune system's primary function is to identify and eliminate foreign agents that attempt to enter the body (e.g., bacteria, viruses, parasites, and fungi). The immune system itself is made up of highly specialized cells that originate in the body's bone marrow; after such cells become active, they are concentrated in certain organs such as the thymus, the peripheral lymphoid organs, and

the lymph nodes. The immune cells are then released from these organs into the blood stream, where they attempt to ward off the foreign antigens.

The most prominent and functionally important cells in the body's immune system are known as leukocytes (white blood cells). These leukocytes may be classified as (a) granulocytic cells, (b) monocytes/macrophages, or (c) lymphocytes. The lymphocytes, which make up about 20% of the leukocytes in the blood, may be either B-cells or T-cells. The T-cells (which derive their name from the fact that they mature in the thymus) are the cells that make direct contact with the invading agent, and are essentially the primary defense of the immune system.

Our understanding of the physiological and biochemical effects of the HIV virus has increased tremendously over the last few years. However, the exact manner in which psychosocial variables affect the process of HIV infection is as yet poorly understood.

Although still in its infancy, there is an important and growing body of research attempting to empirically assess the relationship between psychosocial factors and immunologically mediated illnesses. There are several potential pathways through which the effect of psychological variables on the immune system may be expressed, including reactivation of latent viruses, reduced functioning of peripheral lymphocytes as a result of altered neuroendocrine processes, and a greater vulnerability to initial infection (Solomon & Temoshok, 1990).

Much of this recent research has focused on the relationship between psychological stress and psychological well-being on the one

hand and immune system functioning on the other (Jemmott, 1985). The current study builds on this literature by focusing on the effects of coping styles, stress, and depression on immune system functioning.

Statement of the Problem

Persons with AIDS (PWAs) are faced with tremendous challenges in all areas of their lives, and it is not surprising that many PWAs fall victim to a variety of psychological and neuropsychological difficulties, ranging from the expected anxiety to clinical depression and dementia. While many of these disorders, particularly in the terminal stages, fall into the category of physiologically determined neuropsychological disorders, many other disorders are primarily psychological in nature. This raises the issue of the relationship of psychological well-being to immune function. To the degree that there is such a relationship, the prevention and/or mediation of psychiatric disorders may be important in limiting the degree of immune system breakdown.

The current study addressed the issue of the relationship between psychological well-being and immune function in a sample of seropositive homosexual and bisexual males drawn from the patient population at the Veteran's Affairs Medical Center in West Los Angeles.

Research Questions

This study addressed the following research questions.

1. What is the relationship between specific styles of coping and immune system functioning? Are there certain coping styles that are

positively related to the body's ability to maintain its immune response, despite the HIV infection?

2. What is the relationship between specific areas of stress and immune system functioning? Are some types of stress more closely associated with impaired immune system functioning than others?

3. What is the relationship between psychosomatic symptoms in specific areas of the body and immune system functioning?

4. What is the relationship between depression and immune system functioning?

The remainder of this chapter will discuss the importance of the research problem, present a conceptual framework for the study, and define terms as used in the study.

Importance of the Problem

As Van Gorp, Satz, Hinkin, Evans, and Miller (1989) pointed out, it is somewhat difficult to compare the results of various studies of psychological functioning, neurological impairment, and immune system functioning in this population due to a lack of definitional clarity. While there has been some recent research addressing the relationship between psychological functioning and immune system function, many existing studies suffer from methodological limitations. There are inherent difficulties involved in obtaining reliable responses from HIV-infected subjects, particularly in terms of neuropsychological instruments. Many studies have not clearly differentiated between symptomatic and asymptomatic patients, thus making it more difficult to distinguish the dynamics in an HIV-infected population. Other

weaknesses have included poorly selected control groups and failure to control for possible confounding variables such as anxiety, depression, and neurological disorders.

Nevertheless, there is an emerging body of literature suggesting that psychosocial variables play an important role in influencing immune system functioning in HIV-spectrum illness (Temoshok, 1988). The exact nature of this relationship is unclear, however, and may vary considerably depending on the stage of the illness.

The current study represents an improvement in research methodology compared to much previous research. The study tapped into an existing pool of HIV seropositive subjects already participating in research at the West Los Angeles Veterans Affairs Medical Center, and utilized a control group of seronegative gay male subjects. The sample of HIV seropositive subjects also included individuals at various stages of the illness, allowing systematic comparisons to be done.

The study also had the advantage that CD4 data were collected at 6-month intervals for a 2-year period, allowing the relationship of baseline measures of psychological functioning to be correlated and compared to outcomes over a period of time. The longitudinal component of the study should strengthen the validity of study findings and allow a more objective assessment of the effects of psychological characteristics (e.g., coping style, depression) on immune system functioning over a period of time. In addition, a variety of methodological tools were used to assess the research questions. Each of the instruments utilized has been shown to be reliable and valid in previous research, with the concepts being measured quite clearly

defined. This should contribute to the definitional clarity of the proposed study.

The importance of the current study is also reflected in the growing interest among AIDS researchers regarding the specific psychological effects of AIDS on PWAs. This interest is illustrated by research presented at the recent Eighth International AIDS Conference in Amsterdam (Burack, Stall, Barrett, & Coates, 1992). Burack et al., in a study conducted at the University of California, San Francisco, studied 330 gay and bisexual men who were part of the San Francisco Men's Health Study (J. Burack, personal communication, August 19, 1992). Researchers found that HIV-infected men who were depressed exhibited a much more rapid decline and death. They found that CD4 counts dropped 38% faster among depressed subjects than in non-depressed subjects. More specifically, the average rate of CD4 cell decrease was 81 cells per microliter among depressed subjects as compared to 59 cells per microliter among nondepressed subjects. The current study will elaborate this line of research.

More generally, the findings of the current study on the relationship between specific coping styles and immune system functioning should be valuable in helping therapists design interventions for PWAs. Coping strategies shown to be correlated with impaired immune system functioning are explored, as is the relationship between various types of stress and immune system functioning. The results provide some guidance for interventions designed to teach individuals to switch to more productive coping styles. By enlarging our knowledge of coping styles among AIDS patients, it should be

possible to develop appropriate interventions to be put into effect prior to the patient's becoming symptomatic. Ultimately, such interventions could be valuable in enhancing the quality of life for persons living with AIDS.

Theoretical Framework

The traditional biomedical model focuses on the identification and treatment of disease. The basic assumption of this approach is that any disease is a pathological process which can be observed, and for which the cause can be empirically identified (Shaver, 1985). Engel (1977) pointed out that the underlying philosophical approach of the biomedical model is one of reductionism; it is assumed that complex phenomena can be broken down into their component parts.

Originally developed by Descartes, this principle lies at the core of most modern medicine. Although there were some nineteenth century physicians and researchers who were aware that disease should be seen as involving psychological as well as physiological variables, they were the exception rather than the rule.

In contrast to this approach, the current research assumes that it is necessary to distinguish between disease and illness (Pfifferling, 1983). "Disease" refers to the specific medical diagnosis, and is consistent with the way most physicians trained in the biomedical model view their patients. "Illness," in contrast, refers to the overall condition of the individual, encompassing social and psychological variables as well as physical. The key point is that it is essential

to distinguish the various components of illness, and to reconceptualize health care in terms of a health-illness continuum.

The Emerging Field of Psychoneuroimmunology

Some of the most interesting research is being done in the area of psychoneuroimmunology (PNI), an evolving, multidisciplinary field dealing with the interactions of psychological factors such as emotions and behavior, the central nervous system, and the immune system. The origins of the field can be traced to the work of Solomon and Moos (1964), who coined the term "psychoimmunology" to refer to the effects of psychological and psychosocial variables (such as stress, affects, traits, and coping styles) on the onset and course of immunologically related diseases. At that time, Solomon and Moos proposed the single speculative hypothesis that "stress can be immunosuppressive."

Early psychoimmunology research focused on the effects of acute, short-term stressors on immune system functioning. Research by Solomon (1969) demonstrated the stress-induced suppression of the immune system in rats. The somewhat broader term psychoneuroimmunology (PNI) refers to the general realm of the complex interactions between the central nervous system and the immune system (Ader, Felton, & Cohen, 1991). PNI has been defined as a:

> ...multidisciplinary field dealing with the complex bidirectional interactions of psychological factors, such as emotions and behavior, the central nervous system, and the immune system. (Solomon & Temoshok, 1990, p. 240)

One of the early studies that inspired further research was carried out by Locke (1982), who found that the blood samples of Apollo III astronauts taken during the recovery phase of a space mission had

elevated white blood cell counts. He hypothesized that the stress involved in space flight leads to an increased invasion of antigens, which explained the elevations in white blood cell counts.

The specific nature of stress and its controllability are critical issues in psychoneuroimmunology research. Given the inherent variability of stressful stimuli and the fact that individuals continually learn new coping strategies, physiological and biological responses are not stereotyped. Borysenko (1984), for example, surveyed the various mechanisms involved in immune system response, and cited various research findings supporting the mind's role in enhancing that response. She developed a model of immune system responses integrating hereditary, environmental, and behavioral variables as predictors of disease susceptibility. Her model is based on the premise that disease can be caused by any of these three variables acting in isolation, or that it may be caused by two or more of the variables acting simultaneously. For example, certain diseases involve genetic factors and are associated with particular histocompatibility types; at the same time, the disease may have been exacerbated by exposure to an environmental agent (e.g., a pathogen). Similarly, behavioral variables may interact by compromising the body's immune function, creating a predisposition to infection by a disease-specific pathogen. However, the disease might have occurred as a result of the original genetic factor acting alone.

Gorman and Kertzner (1990) explored the nature of the relationship between cognitive functioning and immune system functioning, with particular reference to human immunodeficiency virus immunopathology.

They pointed out that there is an emerging body of research using both animals and human subjects supporting the argument that there is an ongoing reciprocal relationship between the central nervous system (CNS) and the immune system. Among the variables identified as most important is the reduction in sympathetic tone and noradrenergic turnover in the hypothalamus and the peripheral lymphoid organs that occurs during an acute immune response. These reductions suggest that psychological states, and particularly depression, may have an immunosuppressive component.

In summary, the psychoneuroimmunological perspective provides the basis for developing testable hypotheses with regard to human T-lymphotropic virus Type III (HTLV-III) diseases. Solomon and Temoshok (1987) derived nine such hypotheses, dealing with the effects of stress on HTLV-III, correlations between psychological variables and alterations in immune function, the effects of the prenatal endocrine environment on sexual orientation and immune competence, and the influence of psychological interventions on psychic distress associated with AIDS.

The implication of adopting such an approach is that the research study must attempt to assess the effects on the immune system of stress, emotions, personality, cognition, and other psychological variables (Jemmott, 1985). PNI represents a major departure from previous approaches to the study of immunologic processes in that the immune system has traditionally been conceptualized as being autonomous from and unaffected by psychological variables. However, a considerable body of research now suggests that psychological variables

have a significant influence on the body's susceptibility to conditions that are associated with impaired immunologic functioning. This impact of psychological variables may be particularly salient in the case of Persons with AIDS.

The PNI framework implies that it is insufficient to focus on immunologic factors alone; instead, it is essential to incorporate a variety of other potential independent variables into the research design (Ratliff, Temoshok, Kiecolt-Glaser, & Tamrakin, 1990). Other important blocks of variables to be considered include:

1. Antecedent variables such as age, sex, and sociodemographic background;

2. Use of common medications such as beta-blockers for hypertension; and

3. Psychological distress, including especially levels of anxiety and depression.

Theories of Personality and Implications for Measurement

Researchers exploring the relationship between personality characteristics and disease have generally focused on one of several alternative dimensions (Millon, Green, & Meagher, 1979). Some researchers have focused their attention on personality style, which can be defined as a set of enduring personality traits which shape the individual's susceptibility to a disease, as well as how one copes with the disease after its onset (Kahana, 1972; Lipowski, 1977). The underlying assumption of this approach is that the individual's personality characteristics form a consistent matrix that strongly

influences the way the individual reacts to the stimuli presented by the disease. These reactions encompass the dimensions of emotions, cognition, behavior, and the body's physiological responses.

Other researchers have concentrated on specific psychological traits rather than an overall personality pattern. Examples of this approach include studies on the impact of chronic stress on onset and progress of disease (Holmes & Masuda, 1974; Rahe, 1977); the effects of the learned helplessness syndrome (Boyd, Yeager, & McMillan, 1973); and the effects of social isolation (Cobb, 1976).

Another, and ultimately more productive approach, has emphasized the overall coping ability of the individual faced with the disease diagnosis. Various researchers have stressed that understanding the nature of these personality traits and their impact on the coping process is centrally important (Kahana, 1972; Millon, Green, & Meagher, 1981). This approach assumes that there are certain enduring personality traits that either contribute to or detract from effective coping. More specifically, the effects of the personality traits take the form of prediposed tendencies to react to stressors with relatively predictable patterns of emotional, cognitive, behavioral, and physiological responses.

The current study relies heavily on Millon's (1969) theory of personality pathology. Millon noted that most existing psychodiagnostic tools are not readily applicable in terms of focus or content for the accurate diagnosis of medical patients seen in inpatient settings (Millon, Green, & Meagher, 1982). Millon et al. (1982) argued that using instruments with psychiatrically oriented

norms to assess a medically ill or rehabilitative population is likely to yield misleading results. Among the problems noted were inappropriate norms and the possibility that inappropriate clinical symptoms would be identified.

In contrast to the traditional view of psychopathology as an abnormal phenomenon, Millon argued that:

> Etiology in psychopathology may be viewed as a developmental process in which intraorganismic and environmental forces display not only a reciprocity and circularity of influence but an orderly and sequential continuity throughout the life of the individual. (Millon, 1969, p. 492)

That is, normality and pathology are seen as endpoints on a continuum, with no clear division between the two. Millon developed the concept of the "personality pattern" to describe the developmental nature of intrapsychic processes. He argued that each individual has certain "intrinsic and pervasive modes of functioning," and that these emerge out of a complex developmental history.

Millon argues that personality patterns should be evaluated primarily in terms of coping abilities or styles. A healthy personality pattern is characterized by a flexible and adaptive manner, while a pathological personality pattern is characterized by inflexible or defective responses. More specifically, Millon defines three criteria for evaluating the normal-pathological continuum: adaptive inflexibility (rigid use of limited coping strategies); vicious circles (attitudes and behaviors that intensify old difficulties and create self-defeating consequences); and tenuous stability (lack of resilience to conditions of stress).

The Millon Behavioral Health Inventory (MBHI) is based explicitly on Millon's theory of personality. The focus of the instrument is on interpersonal behaviors; specifically, the instrument taps information regarding the kind of reinforcements sought (positive or negative), where the individual looks for help (self or others), and the types of behaviors engaged in to acquire reinforcements (active or passive). The MBHI has eight primary subscales measuring Coping Strategies, and these are directly linked to Millon's theory of personality.

The key elements of Millon's (1969) theory are defined in terms of the interaction of (a) interpersonal style, and (b) nature and source of reinforcements. In other words, the two cross-cutting dimensions form a matrix with the nature and source of reinforcements defining the rows and the passive-active dimension of interpersonal style defining the columns. This matrix is shown graphically in Table 1.

Table 1

Millon's Personality Matrix

		Interpersonal Style	
		Passive	Active
Nature and Source of Reinforcements	Detached	Passive-Detached	Active-Detached
	Dependent	Passive-Dependent	Active-Dependent
	Independent	Passive Independent	Active-Independent
	Ambivalent	Passive-Independent	Active-Independent

This model was later applied to the development of the MBHI instrument (see more detailed discussion in Chapter III).

Millon's model encompasses eight distinct "normal" variants of personality. These are as follows:

Introversive Style: Keeps to self, unemotional, not easily excited, rarely gets socially involved, lacks energy, vague about symptoms, passive about self-care.

Inhibited Style: Shy, socially ill at ease, avoids close relationships, fears rejection, feels lonely, distrustful, is easily hurt, requires sympathetic support.

Cooperative Style: Soft-hearted, sentimental, reluctant to assert self, submissive, lacks initiative, eager to take advice, compliant, dependent, devalues self-competence.

Sociable Style: Charming, emotionally expressive, histrionic, talkative, stimulus seeking, attention seeking, unreliable, capricious in affect, easily bored with routine.

Confident Style: Self-centered, egocenteric, self-assured, exploitive, takes others for granted, expects special treatment, benignly arrogant.

Forceful Style: Domineering, abrasive, intimidates others, blunt, agressive, strong-willed, assumes leadership role, impatient, easily angered.

Respectful Style: Serious-minded, efficient, rule conscious, proper, correct in behavior, emotions constrained, self-disciplined, avoids unpredictable, orderly, socially conforming.

Sensitive Style: Unpredictable, moody, passively aggressive, negativistic, complainer, guilt ridden, anticipates disappointments, displeased with self and others.

One of the implications of Millon's theory is that psychopathology is not the result of outside factors, but rather emerges from the individual's existing personality patterns. In a sense, then, "normality and pathology" are relative concepts, with no clear dividing line between them. Psychopathology occurs as part of an ongoing developmental process; more specifically, psychopathology should be seen as occurring when an individual adopts certain maladaptive coping mechanisms that lead to undesirable outcomes.

Coping with AIDS

Coping can be conceptualized as efforts to manage environmental stresses and/or to regulate the emotions aroused by the stress (Lazarus, 1982). Positive coping strategies such as information seeking appear to have a positive influence on adjustment. Information seeking may be seen as representing an active, presumably instrumental, confrontative approach to illness, and thus linked to decreased negative affect (Felton & Revenson, 1984). In contrast, wish-fulfilling fantasy represents an avoidant strategy of diverting attention from the reality at hand; thus, it is an indicator of poor adjustment.

Felton and Revenson (1984) conducted a longitudinal study exploring the coping strategies employed by middle-aged and older adults faced with one of four different chronic illnesses. The study design differentiated illnesses offering few opportunities for control (rheumatoid arthritis and cancer) from illnesses more responsive to individual and medical interventions (hypertension and diabetes). Results showed that patients who sought out information (as a primary

coping strategy) had better psychological adjustment than patients who coped using wish-fulfilling fantasies.

The clinical implication is that efforts to prevent mental health problems among normal adults faced with serious illness (such as AIDS) might fruitfully be directed at boosting the use of information seeking and at breaking the destructive cycle in which poor adjustment and wish-fulfilling fantasy reinforce each other's ill effects.

Definition of Terms

AIDS (group IVC) (Centers for Disease Control, 1986). A diagnosis of AIDS is warranted when any of several conditions exist in an HIV positive individual. These conditions include Pneumocystis carinii pneumonia, an opportunistic neoplasm (e.g., Kaposi's sarcoma), and dementia.

HIV-related encephalopathy. This may be defined as:

> ...Clinical findings of disabling cognitive and/or motor dysfunction interfering with occupation or activities of daily living, or loss of behavioral developmental milestones...progressing over weeks or months, in the absence of a concurrent illness or other condition than HIV infection that could explain the findings. Methods to rule out such concurrent illness and conditions must include cerebrospinal fluid examination and either brain imaging or autopsy. (Centers for Disease Control, 1986)

The American Academy of Neurology AIDS Task Force, working collaboratively with the researchers developing upcoming versions of the International Classification of Diseases (ICD-10) and the Diagnostic and Statistical Manual of the American Psychiatric Association (DSM-IV), has recently developed a revised, clinically

sensitive set of diagnostic criteria for AIDS (Working Group, 1991). The following definitions are derived from this source.

HIV-1 associated dementia complex. Also referred to as HIV-1 associated myelopathy, and consistent with the earlier definitions of HIV encepalopathy, this complex is somewhat more elaborate than the earlier definitions of AIDS dementia complex (Navia, Jordon, & Price, 1986; Navia, Cho, & Petito, 1986). The essential feature of the complex is disabling cognitive impairment, typically accompanied by motor dysfunction and/or significant behavior change. The definition is consistent with DSM-III-R's definition of dementia, which requires that there be a memory deficit, accompanied by an impairment in abstract thinking, judgment, or the cognitive functions (i.e., aphasia, apraxia, agnosia, or constructional difficulty). DSM-III-R defines dementia as characterized by "a loss of intellectual abilities of sufficient severity to interfere with social or occupational functioning." The Working Group's diagnostic criteria allow for the following distinctions:

1. Those who have all three conditions--cognitive impairment, motor dysfunction, and behavioral change--are referred to as having HIV-1 associated dementia complex.

2. Those with cognitive impairment and motor dysfunction, but without behavioral change, are referred to as having HIV-1 associated dementia complex (motor).

3. Those with cognitive impairment behavioral change, but without motor dysfysfunction, are referred to as having HIV-1 associated dementia complex (behavior).

HIV-1 associated minor cognitive/motor disorder. Somewhat less severe than HIV-1 associated dementia complex, this disorder does not involve fundamental disruption of life function. When a seropositive individual has cognitive impairment of unknown etiology, but is not impaired to the degree required for the dementia diagnosis, this is the appropriate diagnosis.

Additionally, the following working definitions will be employed in this study

Styles of Coping. These will be assessed with the eight basic coping scales of the Millon Behavioral Health Inventory (MBHI) (Millon, Green, & Meagher, 1982). These coping styles are: introversive, inhibited, cooperative, sociable, confident, forceful, respectful, and sensitive.

Psychogenic attitudes. These attitudes, which are essentially measures of stress, will be assessed with the six psychogenic attitudes scales of the MBHI: chronic tension, recent stress, premorbid pessimism, future despair, social alienation, and somatic anxiety.

Psychosomatic correlates. These will be assessed with the three psychosomatic correlates scales of the MBHI: allergic inclination, gastrointestinal susceptibility, and cardiovascular tendency.

Depression. Depression will be assessed with the Beck Depression Inventory.

CHAPTER II

REVIEW OF THE LITERATURE

Chapter Overview

This chapter reviews relevant literature in several interrelated areas, organized from the general to the specific. The first section deals with some of the most important research on coping with stress, with an emphasis on cognitive theories of coping. This general section provides relevant background for further exploration of coping with psychological stress by PWAs.

The remainder of the chapter includes a review and critique of the existing body of research on the psychological impact of HIV infection. This includes a specific discussion of the psychological correlates of HIV infection and AIDS. The following section deals with the potential relationship between psychosocial variables and immune system functioning. The final section of the chapter briefly discusses the effects of HIV on cognitive functioning and the central nervous system.

Coping with Stress

Empirical research on adaptation to psychological stress was first conducted by the military during World War II to better understand how soldiers functioned under combat conditions. Psychologists had observed that brutal conditions (e.g., combat, concentration camps, natural disasters) led to breakdowns in the psychological functioning of some individuals, while others seemed to find ways to cope with the stress. Explaining the sources of these utterly different outcomes

could obviously be important in identifying the factors which would predict the best strategies for coping with situations of extreme stress.

Individual interactions with (and attempts to manage) the surrounding environment have long been a concern of psychologists with diverse theoretical orientations. Not surprisingly, the theoretical explanations differ considerably. Psychoanalytic theories and ego psychology use the concept of conflict resolution to explain those ego processes which mediate between the person's impulses and the constraints of external reality. Developmental psychology focuses on the accumulation of coping abilities and resources over the life span as providing individuals with the ability to handle problems during each stage of the life cycle. Evolutionary and behavioral approaches emphasize the problem-solving aspects of coping to explain how individuals cope with problems they encounter (Moos & Billings, 1982).

At one level, coping is a dynamic cognitive process which occurs in the interaction of individual with environment. However, coping also involves psychological, physiological, behavioral, and social components (Lazarus & Folkman, 1984; Menaghan, 1983). Selye observed that exposure to a stressor may be followed by physical and chemical changes in the body. Prolonged psychological and emotional stressors may be translated into physical illness, and physical coping involves both voluntary and involuntary changes in the body to manage the effects of stress (Selye, 1956, 1974). Behavioral coping occurs when individuals engage in specific, conscious behaviors to manage stress.

Examples include relaxation exercises and specific tasks intended to improve the situation (Lazarus & Folkman, 1984).

Perhaps the most comprehensive and useful theoretical framework is that provided by the cognitive theories pioneered by Dr. Richard Lazarus and his colleagues at the University of California at Berkeley (Lazarus, 1981, 1982; Lazarus, Averill, & Opton, 1974). Their approach is based on individual level-assessment of events and the ability to cope effectively. Coping was originally seen as a problem- solving effort which occurs when an individual faces stressors that are directly related to his or her welfare and which are taxing to the individual's adaptive ability (Lazarus et al., 1974).

The recognition that there is considerable individual variation in response to stress was an important factor in the development of cognitive theories of stress (Holroyd & Lazarus, 1982). These theories begin with the assumption that thinking and conscious reasoning have an effect on emotions, physical reactions, and behavior. At the most basic level, such cognitive theories are based on the observation that what differentiates human beings from other animals is their possession of language skills and their ability to think, reason, and solve problems.

The occurrence of stressors, individual appraisals, and the adoption of specific coping behaviors and strategies should be seen as a dynamic process. Only when an event is appraised as representing a threat does coping occur. Both the original appraisal of an event as threatening or nonthreatening and the specific coping behavior chosen

are a function of beliefs and experiences over the course of an individual's life span.

The primary dimensions of cognitive coping include coping styles (generalized attitudes, values, and skills), appraisals (intrapsychic evaluations of events), and coping behaviors such as problem solving or palliative adjustments of emotional reactions. Most researchers have incorporated these basic dimensions into their conceptual frameworks, although the terminology may differ from study to study.

Lazarus' (1981) cognitive-based approach was influenced by the clinical Gestalt methods and phenomenological processes. These theories seek to explain individual variation by focusing on cognitive processes as shaping emotions. What a person thinks is shaped by beliefs, values, commitments, skills, and physical constitution. These become part of a dynamic transactional process among the individual, the environment, and particular events or situations. The individual's views come into play at the point of appraisal, and influence the original decision as to whether an event is important in the specific context, and thus whether it is necessary to generate a coping effort.

Appraisals. By definition, coping begins as the reaction to an event (Fleishman, 1984). However, it is not a reaction to the event itself, but rather to the individual's perception of that event. Individuals perceive an event as having meaning for their well-being, an intrapsychic cognitive process known as primary appraisal. In order for persons to make satisfactory adaptations, they must be able to distinguish between benign and harmful stimuli or events (Lazarus, 1982). If the primary appraisal is that the event is irrelevant to the

individual, no more mental energy will be expended. However, if the event has some potential meaning to the individual's welfare, the preliminary appraisal becomes a primary factor in determining if it is potentially harmful or challenging (Lazarus et al., 1974).

Events appraised as being potentially harmful or threatening receive a secondary appraisal, in which alternatives for managing the event and resources available are weighed and balanced. An individual will make several different appraisals of events. When an event has been evaluated as being significant to the person's well-being it will be reconsidered a number of times. Secondary appraisals represent an opportunity for individuals to undertake active problem solving or to reappraise the event as not being threatening. These reappraisals may lead individuals to change the meanings which they attribute to the event (Lazarus & Folkman, 1984). Situations which are perceived as changeable often lead to positive reappraisals and renewed coping efforts (e.g., new problem-solving efforts) which may lead to more satisfactory outcomes.

Personal experiences, and especially past experience with stressful situations, are important in determining outcome as well. Individuals who have had successful coping experiences in the past are likely to acquire feelings of personal efficacy, greater self-esteem, and self-confidence (Fleischman, 1984). In contrast, individuals who have had negative experiences are likely to feel less competent to deal with the stressor (Lazarus & Folkman, 1984).

Resources. Each individual has a specific set of resources available for use as coping strategies. Resources include coping

skills, beliefs, commitments, and values. These resources provide motivation and direction for coping activities. Coping behaviors are also situation-specific. The selection of one response over another depends on the situation and the person's coping resources.

Coping responses. Following an appraisal of any event as representing a threat, individuals will initiate coping responses to manage the event. Two primary modes of coping responses, emotion-focused and problem-solving, may be distinguished (Lazarus, 1981). Each individual develops a coping style, shaped by patterns of behaviors and experience over the life span. Coping responses take many forms, and may include direct involvement of other persons or the use of their assets in managing stressful situations.

Coping outcomes. Stressful events are of varying length, and the evaluation of the outcome of particular coping behaviors is problematic. Lazarus and Folkman (1984) categorized coping outcomes as either satisfactory or unsatisfactory based on self-reports. Events with satisfactory outcomes were frequently associated with changeable situations, positive reappraisals, and higher levels of planful problem solving. Events with unsatisfactory outcomes are typically situations perceived as unchangeable or involved a loss of respect for someone else.

Coping, then, can be conceptualized as efforts to manage environmental stresses and/or to regulate the emotions aroused by the stress; these efforts may be both action-oriented and intrapsychic. Positive coping strategies such as information seeking appear to have a positive influence on adjustment. Information seeking may be seen as

representing an active, presumably instrumental, confrontative approach to illness, and thus linked to decreased negative affect (Felton & Revenson, 1984). In contrast, wish-fulfilling fantasy represents an avoidant strategy of diverting attention from the reality at hand; thus, it is an indicator of poor adjustment.

The Psychological Impact of HIV

Psychological adaptation to any life-threatening illness is a function of variation across three distinct dimensions (Holland, 1982): medical factors (symptoms, course of the disease, complications); psychological variables (especially coping skills and social support); and sociocultural milieu (the key variable being degree of social stigma experienced). Given the fact that the consequences of HIV infection are so unrelenting and grim, it is not surprising that most infected individuals experience a great deal of distress, anxiety, and depression. The HIV test itself is anxiety-producing and has tremendous psychological implications (Ostrow, 1985). Individuals are likely to react to a positive HIV test with feelings of helplessness and depression.

Persons with AIDS (PWAs), particularly gay males, are likely to suffer from social isolation and stigmatization at the very time when they most need increased levels of social support. They may have close friends who are HIV-positive or who have AIDS, and are likely to experience continual grief combined with deep-seated anxiety about their own fate (Ostrow, 1986). It is not surprising that the feelings of distress and anxiety often develop into clinical syndromes such as

generalized anxiety disorder or major depressive disorder (Rubinow, Berrettini, Brouwers, & Lane, 1988).

Coates, Temoshok, and Mandel (1984) focused on the relationship between biological and psychosocial variables in the development of AIDS. They pointed out that there are significant psychosocial determinants of both health-promoting and health-damaging behaviors, and that the way in which individuals cope with AIDS may be a crucial independent variable for research.

Some researchers have studied the effects of disruption of the social environment on immune system functioning. For the individual with AIDS, one of the primary disruptions is likely to be the death of friends and the process of grief and bereavement itself. There is a clear relationship between the loss of a spouse and immune system functioning (Schleifer, Keller, Camerino, Thornton, & Stein, 1983). Others have demonstrated a correlation between loneliness and social isolation and immune response (Kielcolt-Glaser et al., 1984).

As the disease progresses, social support becomes increasingly important. The PWA is likely to experience weakness, frequent infections, anorexia, fever, and myalgias. In the latter phases, frequent infections (particularly pneumonia) are likely to require repeated hospitalizations. Neurological complications (impaired function) are common, often manifesting as forgetfulness and inability to concentrate.

The typical pattern experienced by the seropositive individual involves a normal stress response at the time of diagnosis, characterized by disbelief, numbness, and denial. This is followed by

anger, acute turmoil, and disruptive feelings of anxiety. In the case of anxiety, the most common symptoms include panic attacks, agitation, insomnia, tension, and tachycardia.

Perry, Jacobsberg, Fishman, and Weiler (1990) studied the emotional impact of HIV antibody testing on 218 subjects undergoing testing (179 seronegative and 39 seropositive), both before and after test results were disclosed. The testing was accompanied by extensive counseling, at both pretest and posttest. Subjects were given the Hamilton Depression Scale, Beck Depression Inventory, and Brief Symptom Inventory. Results showed that the degree of psychological distress declined significantly among the seronegative subjects after learning the results of the antibody testing. Contrary to expectations, seropositive subjects did not demonstrate an increased level of psychological distress after learning the results.

Rabkin, Williams, Remien, and Goetz (1991) studied the impact of psychiatric disorders, psychological distress, and psychosocial stressors on the course of HIV in a sample of 124 seropositive homosexual men. Subjects were assessed at baseline and at 6-month follow-up, with data collected including the Hamilton Depression Scale and the Brief Symptom Inventory. No consistent pattern of correlation was found between depression and psychological distress and immune status.

Frigo et al. (1986) studied a group of HIV-positive individuals, and found that there were "significant adverse reactions" in 48% of their sample. Typical reactions included depression, anxiety, and preoccupation with AIDS. An additional 10% of their subjects were

found to experience "serious difficulty" in coping with day-to-day life.

The individual who moves from HIV-positive status to the symptomatic phase of the illness is likely to experience psychological distress. The PWA who experiences serious opportunistic infections for the first time will inevitably experience high levels of stress as a result. Tross, Holland, and Wetzler (1985) compared 89 gay males with AIDS, 39 with AIDS-Related Complex (ARC), and 149 asymptomatic patients. They found that the men who had developed ARC experienced as equally high levels of general and AIDS-specific stress as the AIDS patients.

The progression of AIDS may be seen in terms of models of reaction to the stress involved in any life-threatening illness.

Initial crisis. During this phase of the illness, the typical reaction pattern involves an acute response of denial, alternating with periods of intense anxiety (Horowitz, 1973). Some patients may engage in such complete denial that they ignore medical advice or continue to engage in further high-risk sexual behavior. Most AIDS patients demonstrate emotional reactions involving shock, denial, guilt, anger, and fear (Nichols, 1985).

The initial crisis is exacerbated by the tremendous social stigma attached to the HIV diagnosis. The diagnosis may bring with it substantial disruption of the individual's social support networks. At the same time, the individual may suddenly find himself in a position where disclosure of such issues as sexual orientation and drug usage may become necessary.

Transitional state. The transitional state begins when the initial denial begins to recede, replaced by alternating states of anger, guilt, anxiety, and self-pity (Nichols, 1985). This period is characterized by a great deal of distress and confusion. Typical distressing events include social ostracization and estrangement from families, leading to loss of self-esteem and even suicidal attempts. The transitional state may also be made more difficult by the loss of economic resources as the individual becomes less able to hold down a job.

Deficiency state (acceptance). According to Nichols (1985), a new and more stable identity may finally develop during this state. The individual learns to realistically accept the limitations that accompany AIDS, and make adjustments in his lifestyle that allows him to function as optimally as possible.

Tross and Hirsch (1988) cite several serious consequences that are typically faced by PWAs, all likely to be extremely stressful. For example, they may experience job loss, eviction, denial or termination of insurance and public services (police, sanitation, fear of being refused burial), delays in getting needed health care, and generalized anxiety. They may also fear that health care workers will exercise excessive infection control precautions, and that physicians and nurses may be insensitive to their psychological needs. It should also be noted that most PWAs reach a point at which they must exhaust all of their financial resources in order to survive. In many cases, their insurance expires (or is cancelled), and they find themselves at the mercy of the American health care system. According to Dane (1989),

PWAs may find themselves in a state of crisis as a result of (a) a hazardous event, (b) a vulnerable state, or (c) an inability to respond to stress with adequate coping mechanisms.

Insufficient attention has been given to the massive experience of grief, particularly for PWAs living in major urban centers with large gay populations. Such individuals are likely to undergo a series of progressive bereavements, as they lose multiple friends to the disease. In many cases this is equivalent to destroying their primary social support network.

Any such grief is often accompanied by profound sadness, numbing, withdrawal, and "vegetative" depressive symptoms, as well as much higher rates of alcohol and substance abuse (maladaptive coping mechanisms). Perhaps most fundamentally, the person diagnosed with AIDS must come to grips with the reality that he is going to die. According to Kubler-Ross (1969), the primary psychological task to be attained is to come to a personal adaptation with this new and disturbing reality. Kubler-Ross characterized this process as involving a set of overlapping bereavement processes. This cycle typically begins with denial, which is followed by anger, bargaining, depression, and eventually an acceptance of the situation. There is a similarity here with the anticipatory grief reaction that occurs among parents and families of other types of dying patients.

The Relationship Between Psychosocial Variables and Immune System Functioning

Various researchers have explored the relationship between levels of depression and immune system function. Schleifer, Keller, and Meyerson (1984) found that patients hospitalized with major depressive disorder had impaired lymphocyte response to mitogens and lowered CD4 cell counts. This was followed up with a study of ambulatory patients with major depressive disorder. When compared with groups of matched controls and inpatient schizophrenia, CD4 cell counts were found to be significantly lower among the depressed patients.

Solomon and Temoshok (1987) conducted one of the first studies to systematically examine the effects of psychosocial variables on immune function in persons with AIDS and ARC. They found that individuals who were psychologically distressed (defined as high levels of anxiety, hopelessness, and depressed mood state) were more likely to have impaired immune response as measured by CD4 and CD8 cell counts.

In a follow-up study, the same group of researchers (Solomon, Temoshok, O'Leary, & Zich, 1987) conducted in-depth psychosocial interviews with AIDS patients over a 5-week period, while collecting data on immune function during each of the 5 weeks. Results identified several correlates of enhanced T-cell functioning: higher levels of tension and anxiety, lower levels of depression, and lower levels of hostility.

Temoshok, Zich, Solomon, and Stites (1987) studied the relationship among psychological, immunological, and neuropsychological variables in a sample of 100 seropositive gay men. Data on absolute

number of CD4 (helper) cells and psychological variables were collected at both baseline and 6-months follow-up. Results showed that measures of tension/anxiety, trait/anxiety, anger/hostility, and loneliness were all associated with impaired T-cell counts. These researchers stressed that the precise relationship between psychosocial variables and immune system functioning may vary substantially depending on the stage of the illness.

There is an emerging consensus that everyday stressful events can have a significant impact on the immune system (Kielcolt-Glaser & Glaser, 1988). The effect of stress on immune function may be associated with actual changes in cellular immunity. Glaser and Kielcolt-Glaser (1988) argued that this effect persists even when the effects of age, loss of sleep, smoking, drinking, and caffeine intake are controlled for.

Dorian and Garfinkel (1987) studied immune system function in a comparative study of psychiatry residents undergoing an oral fellowship examination and a control group of similar residents not taking the examination. They found that there were considerable differences in both B-cell and T-cell counts during the weeks leading up to the exam. Similar studies were undertaken by Glaser, Kielcolt-Glaser, Speicher, and Holliday (1985), who observed a changing pattern of immune system functioning in a sample of medical students tracked through a semester of medical school. Immune system functioning was found to be impaired during examination periods. In a related study, researchers examined immunological changes in blood samples drawn from medical students during examinations as compared to a baseline period one month earlier

(Glaser, Rice, Speicher, Stout, & Kielcolt-Glaser, 1986). Results showed that the blood samples drawn during the examination period had much lower natural killer (NK) cell activity. A related finding was that students who were more socially isolated showed greater degrees of immunosuppression.

Chuang, Devins, Hunsley, and Gill (1989) studied levels of psychological distress in 65 HIV positive gay and bisexual men. They found that all the subjects demonstrated higher than expected levels of psychosocial distress, defined as depression, mood swings, anxiety, negative affect, and suicidal ideation. Asymptomatic individuals and individuals with AIDS-Related Complex were found to be more distressed than persons with full-blown AIDS.

Temoshok et al. (1987) conducted one of the first in-depth psychoimmunologic studies of PWAs. Although their sample was quite small (N = 12), they found that there were striking correlations between psychological outcomes and immune system functioning.

Temoshok (1988) conducted a psychoimmunologic study of 55 AIDS/ARC subjects, collecting data on a range of variables measuring overall dysphoric affect. Subjects completed the psychosocial self-report measures within 2 to 8 weeks after diagnosis. The dependent variable was assessed using standard measures for anxiety, mood state, and hopelessness. Results showed that overall dysphoric affect was positively correlated with overall white blood cell counts.

It is still not clear exactly when the effects of psychological variables make themselves felt; in part, this can be attributed to the lack of clear understanding of the precise pathogenisis of CD4 cell

destruction by HIV (Gorman & Kertzner, 1990). Some researchers have suggested that the existence of previous impairment of the immune system may be a precondition for the psychological variables to have a further impact (Schliefer, Keller, & Bond, 1989).

Several recent studies have also clarified the relationship between psychosocial variables and immune system functioning. Particularly noteworthy is the work of Kemeny et al. (1992) and Burack et al. (1992).

Kemeny et al. (1992) studied the relationship among bereavement, depressed mood, and immunologic patterns. While they found no effect of bereavement, they reported that more depressed mood was positively associated with fewer CD4 cells.

Recently, Burack et al. (1992) reported an important set of findings at the Eighth International Conference on AIDS in Amsterdam. They looked specifically at the correlation between depressive symptomatology and CD4 counts in a sample drawn from the San Francisco Men's Health Study. They studied 308 HIV seropositive subjects in a 6-year prospective study (following subjects from 1985 to 1991). Individuals who were depressed, as measured with the Center for Epidemiological Studies Depression Scale, showed a higher 4-year mortality rate than less-depressed individuals. The researchers concluded that HIV seropositive patients meeting the criterion for clinical depression experienced a faster rate of CD4 cell depletion, as well as the observed higher mortality rate.

Burack pointed out in a press release issued at the conference that the mechanisms through which depression affects immune system

functioning are not clear. It is possible that depressed persons may be less compliant, or less likely to take all possible steps to maintain their immune system functioning. Alternatively, they might also be more likely to continue engaging in high-risk behaviors.

The Effects of HIV on Cognitive Functioning and the Central Nervous System

Effects on Cognitive Functioning

Recent research has suggested that subtle types of cognitive dysfunction may be present even in the earliest stages of HIV infection (Kovner et al., 1989). Various researchers have reported that AIDS patients suffer from memory impairment (Levy, Fernandez, Holmes, Gagen & Pirozzolo, 1987) and psychomotor slowing (Saykin et al., 1987).

However, it is not entirely clear whether early cognitive dysfunction results from the physiological effects of the HIV virus on brain tissue, or whether it is a psychophysiological response by the individual to the knowledge of the life-threatening nature of the illness (Rubinow et al., 1988).

There are also direct organic effects on the brain, as evidenced by studies demonstrating the presence of HIV-infected giant cells (Koenig et al., 1986) and HIV receptors (Levy, Shimabururo, & Hollander 1985) in the brain. Other researchers have found substantial evidence of cognitive impairment in AIDS patients, despite the lack of physiological evidence of opportunistic infections having directly affected the central nervous system of the brain (Joffe, Rubinow, & Squillace, 1986). In other words, the effects of HIV on cognition and

personality function may be significant even in asymptomatic individuals.

Joffe et al. (1986) compared cognitive functioning between a group of 13 AIDS patients (without overt CNS dynfunction) and a matched group of 10 age- and education-matched gay male controls. Findings were that the AIDS patients demonstrated significant impairment in performance on the WAIS (full-scale IQ, verbal IQ, vocabulary subtest, similarities subtest, and the symbol subtest), the Halstead Category Test, and the Trail Making Test. However, the researchers did not have serological test results available for the control group, and thus were not able to control for possible undiagnosed HIV infection.

Rubinow et al. (1988) conducted a follow-up study, comparing a group of 13 medication-free AIDS patients, an HIV-positive control group (N = 9), a chronic illness control group with chronic active hepatitis (N = 9), and a group of health controls (N = 6). Subjects were given a battery of neuropsychological tests, including the WAIS, the Halstead Category Test, Trail Making Test (trail B), and cancellation tasks. Consistent with the earlier findings of Joffe et al. (1986), the AIDS patients were found to score significantly lower on all of the cognitive tasks.

Kovner et al. (1989) conducted a prospective study of 26 HIV-positive subjects who were categorized (per CDC criteria) as having AIDS (group IVC), ARC (IVA), or seropositive for HIV (group II). Subjects were given a test battery of 23 measures tapping neuropsychological processes (cognitive, attentional, and personality). The objective of the study was to evaluate whether cognitive/

neuropsychological impairment could be explained as a function of mood and attentional changes accompanying the HIV diagnosis. Results showed that the cognitive impairments found in HIV-infected ambulatory subjects could not be explained by variation in affective and attentional factors.

Ayers, Abrams, Newell, and Friedrich (1987) evaluated the differences on the Luria-Nebraska Neuropsychological Battery, the MMPI, and the Beck Depression Inventory in a sample of 60 males classified by level of infection. Results showed significant impairments in writing, memory, tactile, and intellectual function for subjects at all levels of infection. The researchers pointed out that some degree of impairment appears to exist even from the early (asymptomatic) stage, and that early intervention is essential.

Praus, Brown, Rundell, and Paolucci (1990) examined the correlations between anxiety and depression and immune system function in a sample of 98 subjects undergoing HIV antibody testing. Almost all (95%) of the subjects tested seronegative. Results showed significant inverse correlations between levels of depression (measured by the Hamilton Depression Scale) and CSF nucleated cell counts and protein levels. In other words, there was a relationship between high levels and depression and impaired immune system functioning.

Effects on the Central Nervous System

The effects of HIV on the central nervous system have been well established (Perry, Belsky-Barr, Barr, & Jacobsberg, 1989). One of the more common CNS complications is nonfocal encephalopathy, with dementia as the dominant feature (Snider et al., 1984). The symptoms of

encephalopathy are quite similar to those of clinical depression, at least in the early stages (Holland & Tross, 1985). The typical initial symptoms are forgetfulness and inability to concentrate, followed by psychomotor retardation, apathy, withdrawal, and loss of libido. Over several months, further symptoms include confusion, disorientation, seizures, and profound dementia.

In one study of 180 patients receiving full neurological examinations during a 3-year period, nine were initially diagnosed with dementia. By the end of the period, over half had developed significant cognitive dysfunction (Tross et al., 1985). The rate of progression of the symptoms shows considerable variation, ranging from a few weeks to several months after onset.

Chapter Summary

The first section of this chapter discussed the basic coping model which provides a key part of the theoretical framework for the dissertation. The emphasis was on the cognitive approach to stress and coping developed by Dr. Richard Lazarus. The second section dealt with the psychological impact of HIV and AIDS. These effects are potentially devastating, ranging from depression to impaired self-esteem to suicidal ideation.

This was followed by a review of some of the recent empirical studies that have been done on the relationship between psychosocial variables and immune system functioning. The literature reviewed in this section of the chapter builds on the literature from psychoneuroimmunology reviewed as part of the "Theoretical Framework"

presented in the first chapter. The final section of this chapter discusses the effects of AIDS on both cognitive functioning and on the central nervous system.

The following chapter presents the research methods utilized in the current study of the relationship between coping styles, stress, and immune system functioning.

CHAPTER III

METHODOLOGY

Restatement of the Problem

Persons with AIDS (PWAs) are vulnerable to a variety of psychological difficulties, ranging from the expected anxiety to clinical depression and dementia. While many of these disorders, particularly in the terminal stages, fall into the category of physiologically determined neuropsychological disorders, many other disorders are entirely psychological in nature. There is a growing body of research, particularly in the evolving field of psychoneuroimmunology, which suggests that psychological variables have a significant influence on immune system function. To the degree that there is such a relationship, the prevention and mediation of psychiatric disorders is important in limiting the degree of immune system breakdown.

The current study, then, addressed the issue of the relationship between psychological well-being and immune function in a sample of seropositive males drawn from the patient population at the Veteran's Affairs Medical Center in West Los Angeles.

Research Design

In an "ideal" scenario, an experimental research study addressing this topic would systematically compare various groups of patients, including subjects who are seropositive but who have not been informed. This would allow the potential confounding effects of previous health

and the impact of the knowledge of diagnosis to be controlled for in the research design itself. This type of research design would be ethically questionable under American Psychological Association guidelines. In other words, the current research topic is a classic example of a situation where it was not practical or ethical to control the independent variable using experimental techniques such as random assignment to groups.

Instead, the current study employed a quasi-experimental longitudinal design to assess the relationship between various psychological independent variables and immune system functioning, operationalized as CD4 counts, in a cohort of AIDS patients over a 24-month time period.

More specifically, the study involved the collection of a battery of psychological and neuropsychological instruments and CD4 data at baseline, as well as data on depression (collected at baseline and twelve months) and CD4 counts at 6-month intervals over a 2-year period. The study analyzed the effect of coping style, stress, and somatic symptoms (the MBHI measures), as well as the effects of baseline depression, on immune sytem functioning over the two-year period.

Variables in the study design included:

Dependent variable. Immune system functioning (CD4 cell counts), collected every six months over the 24-month time period. This is the most widely used indicator of immune system functioning in AIDS research.

The difficulties involved in analyzing time series data are summarized by Kerlinger (1973), who points out that:

> The statistical analysis of time measures is a special and troublesome problem; the usual tests of significance applied to time measures can yield spurious results. One reason is that such data tend to be highly variable...in time data, individual and mean scores tend to move around a good bit. (p. 345)

Given these difficulties inherent in collecting time series data, the focus was on evaluating the CD4 data at 6, 12, 18, and 24 months, while controlling for the baseline CD4 values. This is effectively the same as conceptualizing the dependent variable as the change in CD4 values from baseline to each of the subsequent time periods, or alternatively as the slope of changing CD4 values over time. However, by conceptualizing the model as predicting CD4 values while controlling for baseline values, the statistical difficulties involved in analyzing change scores are addressed.

The rationale for predicting CD4 cell counts is that such counts are widely treated in the AIDS literature as the primary indicator of adequate immune system functioning. The different types of immune system cells work together, with the CD4 cells regulating the immune response of the body, while the CD8 cells and other immune cells actually fight off infected cells. In other words, the primary characteristic of the human immunodeficiency virus is the progressive depletion of CD4-bearing T-lymphocytes preceded by functional deficits in cell-mediated immunity. These immunologic defects are accompanied by clinical manifestations that are initially relatively minor, but as the immune dysfunction progresses, severe opportunistic infections or malignancies develop, fulfilling the established criteria for AIDS.

Independent variables. Independent variables in the study included the first set of independent variables, derived from the Millon Behavioral Health Inventory (MBHI), which were:

1. The eight basic MBHI coping styles: Introversive Style, Inhibited Style, Cooperative Style, Sociable Style, Confident Style, Forceful Style, Respectful Style, and Sensitive Style.

2. The three MBHI psychosomatic correlates: Allergic Inclination, Gastrointestinal Susceptibility, and Cardiovascular Tendency.

3. The six MBHI psychogenic attitudes scales, essentially indicators of stress: Chronic Tension, Recent Stress, Premorbid Pessimism, Future Despair, Social Alienation, and Somatic Anxiety. Additionally, the Beck Depression Inventory was utilized as an independent variable.

Research Hypotheses

Each research hypothesis was statistically evaluated at the $p < .05$ level of significance. The primary statistical technique, discussed in more detail below, was analysis of covariance with the baseline CD4 value as the covariate controlled for in the model.

The first eight hypotheses refer to the relationship between the measures of coping style provided by the MBHI and immune system functioning. As discussed in the "Theoretical Framework" section in Chapter I, these scales are based explicitly on Millon's theory of personality and psychopathology. Higher scores on each subscale represent higher scores on that particular type of maladaptive coping.

While there are no specific cutpoints defining psychopathology on any of the dimensions, higher scores can be assumed to reflect greater degrees of that particular style of maladaptive coping.

Each of the hypotheses is stated in null form.

Hypothesis #1. Subjects who score higher on Introversive Coping at Baseline will not differ on CD4 counts at 6 months, 12 months, 18 months, and 24 months from subjects with lower Introversive Coping scores at Baseline, while controlling for the effects of Baseline CD4 counts.

Hypothesis #2. Subjects who score higher on Inhibited Coping at Baseline will not differ on CD4 counts at 6 months, 12 months, 18 months, and 24 months from subjects with lower Inhibited Coping scores at Baseline, while controlling for the effects of Baseline CD4 counts.

Hypothesis #3. Subjects who score higher on Cooperative Coping at Baseline will not differ on CD4 counts at 6 months, 12 months, 18 months, and 24 months from subjects with lower Cooperative Coping scores at Baseline, while controlling for the effects of Baseline CD4 counts.

Hypothesis #4. Subjects who score higher on Sociable Coping at Baseline will not differ on CD4 counts at 6 months, 12 months, 18 months, and 24 months from subjects with lower Sociable Coping scores at Baseline, while controlling for the effects of Baseline CD4 counts.

Hypothesis #5. Subjects who score higher on Confident Coping at Baseline will not differ on CD4 counts at 6 months, 12 months, 18 months, and 24 months from subjects with lower Confident Coping scores at Baseline, while controlling for the effects of Baseline CD4 counts.

Hypothesis #6. Subjects who score higher on Forceful Coping at Baseline will not differ on CD4 counts at 6 months, 12 months, 18 months, and 24 months from subjects with lower Forceful Coping scores at Baseline, while controlling for the effects of Baseline CD4 counts.

Hypothesis #7. Subjects who score higher on Respectful Coping at Baseline will not differ on CD4 counts at 6 months, 12 months, 18 months, and 24 months from subjects with lower Respectful Coping scores at Baseline, while controlling for the effects of Baseline CD4 counts.

Hypothesis #8. Subjects who score higher on Sensitive Coping at Baseline will not differ on CD4 counts at 6 months, 12 months, 18 months, and 24 months from subjects with lower Sensitive Coping scores at Baseline, while controlling for the effects of Baseline CD4 counts.

The next six hypotheses refer to the relationship between the psychogenic attitudes scales of the MBHI, each measuring a different dimension of stress, and immune system functioning.

Hypothesis #9. Subjects who score higher on Chronic Tension at Baseline will not differ on CD4 counts at 6 months, 12 months, 18 months, and 24 months from subjects with lower Chronic Tension scores at Baseline, while controlling for the effects of Baseline CD4 counts.

Hypothesis #10. Subjects who score higher on Recent Stress at Baseline will not differ on CD4 counts at 6 months, 12 months, 18 months, and 24 months from subjects with lower Recent Stress scores at Baseline, while controlling for the effects of Baseline CD4 counts.

Hypothesis #11. Subjects who score higher on Premorbid Pessimism at Baseline will not differ on CD4 counts at 6 months, 12 months, 18 months, and 24 months from subjects with lower Premorbid Pessimism

scores at Baseline, while controlling for the effects of Baseline CD4 counts.

Hypothesis #12. Subjects who score higher on Future Despair at Baseline will not differ on CD4 counts at 6 months, 12 months, 18 months, and 24 months from subjects with lower Future Despair scores at Baseline, while controlling for the effects of Baseline CD4 counts.

Hypothesis #13. Subjects who score higher on Social Alienation at Baseline will not differ on CD4 counts at 6 months, 12 months, 18 months, and 24 months from subjects with lower Social Alienation scores at Baseline, while controlling for the effects of Baseline CD4 counts.

Hypothesis #14. Subjects who score higher on Somatic Anxiety at Baseline will not differ on CD4 counts at 6 months, 12 months, 18 months, and 24 months from subjects with lower Somatic Anxiety scores at Baseline, while controlling for the effects of Baseline CD4 counts.

The next three hypotheses refer to the relationship between the psychosomatic correlate measures provided by the MBHI and immune system functioning.

Hypothesis #15. Subjects who score higher on Allergic Inclination at Baseline will not differ on CD4 counts at 6 months, 12 months, 18 months, and 24 months from subjects with lower Allergic Inclination scores at Baseline, while controlling for the effects of Baseline CD4 counts.

Hypothesis #16. Subjects who score higher on Gastrointestinal Susceptibility at Baseline will not differ on CD4 counts at 6 months, 12 months, 18 months, and 24 months from subjects with lower

Gastrointestinal Susceptibility scores at baseline, while controlling for the effects of Baseline CD4 counts.

Hypothesis #17. Subjects who score higher on Cardiovascular Tendency at Baseline will not differ on CD4 counts at 6 months, 12 months, 18 months, and 24 months from subjects with lower Cardiovascular Tendency scores at baseline, while controlling for the effects of Baseline CD4 counts.

The next hypothesis refers to the relationship between the Beck Depression Inventory and immune system functioning.

Hypothesis #18. Subjects who score higher on Depression at Baseline will not differ on CD4 counts at 6 months, 12 months, 18 months, and 24 months from subjects with lower Depression scores at baseline, while controlling for the effects of Baseline CD4 counts.

The next hypothesis postulated that it is possible to predict the change in CD4 levels by utilizing all of the above independent variables in a multivariate framework. This multivariate hypothesis is also stated in null form.

Hypothesis #19. CD4 levels at each time period after baseline (6 months, 12 months, 18 months, and 24 months) cannot be predicted by a multivariate combination of the coping styles, the psychosomatic correlate scales, the psychogenic attitudes scales, and level of Depression, while controlling for the effects of Baseline CD4 counts.

Procedures

This study was conducted as a supplementary component of a Veterans Affairs-funded study being conducted at the West Los Angeles

Veterans Affairs Medical Center. The current study tapped into the existing patient population at the West Los Angeles Veterans Affairs Medical Center.

All instruments were administered in an outpatient clinic setting on a one-on-one basis. All examiners were doctoral or post-doctoral students in the UCLA-Veterans Administration Neuropsychology program who had been thoroughly trained by qualified neuropsychologists in administering the instruments.

CD4 and CD8 levels were obtained via laboratory analysis of blood samples obtained from the subjects. All blood samples were drawn by certified laboratory technicians employed by the Veterans Affairs Administration. Certain physiological tests were administered only to patients who had become symptomatic. These included a Magnetic Resonance Imaging (MRI), Computerized Tomography (CT) Scan, and/or a Positron Emission Tomography (PET) Scan.

Subjects

HIV Seropositive

Subjects consisted of community-dwelling, self-identified gay or bisexual males or IV drug users enrolled in a longitudinal study of the psychological and neuropsychological effects of HIV infection. Subjects were enrolled from the VA outpatient immune deficiency clinic. All of the HIV positive subjects had been found to be HIV-1 seropositive within the previous six months based on ELISA testing with Western blot confirmation.

There were 92 subjects diagnosed as HIV seropositive; of these subjects, baseline data were available for 79 subjects. Fifteen of the seropositive subjects had been diagnosed as having full-blown AIDS. The primary mode of transmission was homosexual contact, with only three HIV seropositive subjects indicating they had been infected via heterosexual contact or IV drugs. Over a third (37%) of the seropositive subjects were identified having abused alcohol at one point or another in their lives, with 40% having abused drugs. Substance abuse was defined based on clinical evaluation using DSM-III-R criteria (305.00 for Alcohol Abuse and 305.90 for Substance Abuse, N.O.S.).

A demographic profile of the seropositive subjects is shown in Table 2. Mean age was 42.1 years. Educational attainment was moderate, with 28.3% of seropositive subjects having only a high school diploma or less. Only 16.2% had a college degree or graduate work. More than half (58%) were employed, with 18.5% being unemployed specifically due to HIV. Most of the subjects were white, although there was some minority representation.

In the sample of seropositive patients, slightly less than a third (30%) of the seropositive subjects were taking AZT, an anti-retroviral agent that is thought to inhibit replication of HIV. The proportion of subjects taking AZT was much higher among those with full-blown AIDS (67%) than it was among those who were still asymptomatic (13%). A series of chi-square analyses was completed comparing AZT drug use to high and low groups for each hypothesis. Results showed no significant differences.

Table 2

Demographic Profile of Seropositive Subjects

Variable	Number	Percentage
Age		
20-30	12	13.0%
31-40	31	33.7%
41-50	33	35.9%
51-60	10	10.9%
61+	6	6.5%
Education		
High School or Less	26	28.3%
Some College	40	43.5%
4-Year Degree	13	8.1%
Graduate Work	13	8.1%
Employment Status		
Working	53	57.6%
Unemployed	22	23.9%
Unemployed due to HIV	17	18.5%
Ethnicity		
White	74	80.4%
African American	11	12.0%
Latino	5	5.4%
Asian American	4	2.2%

CD4 data were available for a subsample of the HIV seropositive subjects, with the number of available data points declining for each "wave" of data collection. Descriptive statistics on the available CD4 data are shown in Table 3.

HIV Seronegative

A seronegative control group consisting of 29 gay males who had received a negative test result were included. Control group members were similar to the seropositive group on most background variables. However, they were somewhat more educated than the HIV-positive group

Table 3

Available CD4 Data and Study Attrition

CD4 Counts	N	Mean	SD	Mortality Rate	Withdrawal
Baseline	79	320.462	203.084	--	--
6 Months	57	331.491	228.461	--	--
12 Months	45	282.178	209.853	--	--
18 Months	26	230.731	187.258	--	--
24 Months	24	223.917	201.801	21	10

*Normal CD4 range is 800-1300

(15.4 years versus 14.3 years) ($p < .05$), more likely to be employed (attributable the the fact that the HIV- positive subjects were more likely to be on disability), and had less self-reported drug and alochol abuse.

Instruments

The Millon Behavioral Health Inventory

Van Gorp and Cummings (1989) have pointed out that changes in mood and affect are commonly associated with certain types of brain dysfunction regardless of age; in fact, during the early stages of some progressive conditions, changes in mood and affect may precede abnormalities in the cognitive sphere. This implies that it is important for clinicians and researchers to develop and apply appropriate measures of mood, affect, and personality designed specifically for--and validated on--individuals in whom brain disease is suspected. If it is necessary to rely on traditional personality

instruments (i.e., instruments validated on normal populations), the results must be interpreted with caution.

The MBHI is a 150-item inventory that yields 20 scores, as well as a three-item validity scale. The MBHI is one of the few instruments designed "specifically with physically ill patients and medical-behavioral decision-making issues in mind" (Millon et al., 1981, p. 163).

Basic coping styles. The eight basic coping styles that comprise the MBHI are derived from Millon's (1969) theory of personality.

1. Introversive Style (32 items)
2. Inhibited Style (43 items)
3. Cooperative Style (33 items)
4. Sociable Style (40 items)
5. Confident Style (33 items)
6. Forceful Style (33 items)
7. Respectful Style (42 items)
8. Sensitive Style (48 items)

Psychogenic attitudes scales. Six psychogenic attitudes scales were developed to reflect psychosocial stressors identified in the literature as likely to be predictive of physical illness. These reflect the respondents' perceptions of present and future stressors.

A. Chronic Tension (29 items)
B. Recent Stress (20 items)
C. Premorbid Pessimism (40 items)
D. Future Despair (38 items)
E. Social Alienation (33 items)

F. Somatic Anxiety (34 items)

Psychosomatic correlates scales. Three psychosomatic correlates scales were developed:

MM. Allergic Inclination (34 items)

NN. Gastrointestinal Susceptibility (27 items)

OO. Cardiovascular Tendency (38 items)

Prognostic indices scales. An additional three scales measuring prognosis were developed:

PP. Pain Treatment Responsivity (42 items)

QQ. Life Threat Reactivity (42 items)

RR. Emotional Vulnerability (12 items)

Development of the MBHI. Initially, a pool of over 1,000 items was identified. Items for the pool were identified based on items from other instruments, abnormal and personality texts, and analysis of personality dimensions. Items were developed with the objective of covering the full range of possible coping styles and personality characteristics. Items were balanced so that approximately half were phrased in a positive direction, and half in a negative direction.

This pool of items was then administered to several samples in medical settings, with the results then analyzed to reduce the pool of items to those that discriminated patients on the role played by psychosocial factors in their illnesses. This analysis was supplemented with item-scale correlation analysis and content analysis of the items. Items were dropped that were determined to be too complicated, showed desirability bias, or lacked clarity in phrasing. The results of these statistical and substantive analyses were used in

reducing the pool to 64 items (for eight basic coping styles), 83 items (for six Psychogenic Attitude scales), and 3 validation items.

Millon et al. (1979) assessed the reliability characteristics of the MBHI, and found that Kuder-Richardson reliability coefficients (the KR-20 version of coefficient alpha for internal consistency) ranged from .66 to .90 for the MBHI scales. They also conducted a test-retest, with 89 patients tested at intervals ranging from 1 to 8 months. They found test-retest coefficients for the scales ranging from .72 to .93 (median test-retest coefficient of .83).

Murphy, Sperr, and Sperr (1986) conducted a validation study in which they assessed the predictive validity of the MMPI and the MBHI simultaneously. Utilizing a sample of patients presenting with chronic pain, the researchers assessed the perceptions of medical psychologists, nonmedical psychologists, and physicians regarding the relative usefulness of the two instruments in predicting outcome. The practitioners were asked to rate the instruments on ten dimensions of clinical utility; they expressed a preference for the MBHI on nine of the ten dimensions.

Sweet, Breuer, Hazelwood, Toye, and Pawl (1985) assessed the validity of the MBHI by correlating the MBHI subscales with selected MMPI subscales and the Beck Depression Inventory. Their findings were somewhat inconsistent. Correlations between the MBHI and the Hypochondriasis (Hs) and Hysteria (Hy) MMPI scales were low; this was contrary to expectations, in that these MMPI scales are generally thought to reflect health-related concerns and inadequate adjustment to chronic pain or illness. However, findings did show that the MBHI

subscales tended to be correlated with MMPI scales reflecting emotional distress (Depression, Psychasthenia) or psychopathology (Psychopathic Deviate, Paranoia, Schizophrenia, Social Isolation).

Weisberg and Page (1988) conducted a study of 42 dialysis patients, and found that the MBHI Coping Style and Psychogenic Attitude Prognostic scales were highly predictive of patient welfare and satisfaction. They also found that the MBHI scales had considerable predictive validity in terms of differentiating patients having dialysis in the home as compared to the hospital setting. That is, patients with fewer pathological coping styles were found to be more likely to obtain their dialysis treatment in the home setting.

Beck Depression Inventory

The Beck Depression Inventory (BDI), which was developed originally to assess the presence and severity of a depressive syndrome in adults (Beck, Ward, Mendolsson, Mock, & Erbaugh, 1961), is a paper and pencil, self-rating scale that taps various components of depression: affective, cognitive, motivational, vegetative, and psychomotor. Both the BDI's reliability and validity are well-established, and it has been used successfully both with normal and patient populations.

The Beck Depression Inventory is a 21-item test, on which scores can range from 0-63. Higher scores represent higher levels of depression. It consists of 21 items; each answered on a continuum of severity ("I do not feel sad" versus "I am so sad or so unhappy that I can't stand it"). Seven of the 21 items reflect physical health or fatigue.

On each of the 21 items, the subject is presented with four choices and is asked to pick out the one statement in each group that best describes the way he or she feels at the present time. It is possible for a subject to circle more than one statement, but in that case, the scorer uses the one with the higher depression rating. The four items in each group are rated 0-3 and indicate increasing degrees of distress. For example, on the first of the 21 items, the subject is asked to decide among the following four choices:

0 = I do not feel sad.

1 = I feel sad.

2 = I am sad all the time and I can't snap out of it.

3 = I am so sad or unhappy that I can't stand it.

Beck et al. (1961) originally reported adequate test-retest and split-half reliability for the instrument, and these findings were confirmed for both a normal, nondepressed elderly sample and an elderly depressed sample using Research Diagnostic Criteria (RDC).

In general, any self-report measure of depression may suffer from bias in that respondents are hesitant to acknowledge a "mental illness." However, the BDI has been shown to be both reliable and valid. Test-retest reliability for psychiatric patients was .82 over a 7-day period; the alpha coefficient of internal consistency for the same sample was .87. The correlation between scores on the test and ratings made by a psychiatrist was .69 for hospitalized psychiatric patients. The test also has been shown to discriminate significantly between patients diagnosed as depressive and patients diagnosed as having nonaffective disorders.

Data Analysis Techniques

Comparisons of HIV Seropositive to HIV Seronegative

Initially, independent group *t* tests were done between HIV seropositive and HIV seronegative subjects on each of the study variables. This included 20 subscales derived from the MBHI (all collected at baseline), and the Beck Depression Inventory (collected at baseline and 6-month intervals). This allowed a systematic comparison of psychological status, as measured by these particular instruments.

Hypothesis Tests

The wording of the hypothesis tests was modified (from the proposal) to incorporate mention of CD4 counts at each time period (6 months, 12 months, 18 months, and 24 months) rather than only at 24 months. The dependent variable for each hypothesis was the particular CD4 count at each data collection point after baseline.

The independent variables were measures of Coping Style (Hypotheses 1-8), Psychogenic Attitudes (Hypotheses 9-14), Psychosomatic Correlates (Hypotheses 15-17), or Depression (Hypothesis 18). In order to differentiate different levels on each independent variable, a transformed version of each independent variable was created based on a median split. Subjects scoring below or at the median were defined as being "low" on each scale, with subjects scoring above the median being classified as "high."

For each hypothesis test, a set of analysis of covariance models was tested. This technique was selected in order to obtain a specific

hypothesis test for each hypothesis stated. For example, the first hypothesis stated that:

> Subjects who score higher on Introversive Coping at Baseline will not differ on CD4 counts at 6 months, 12 months, 18 months, and 24 months from subjects with lower Introversive Coping scores at Baseline, while controlling for the effects of Baseline CD4 counts.

This was tested with the four analyses of covariance models shown in Table 4. This analysis was then elaborated for each hypothesis

Table 4

Analysis of Covariance Framework for Hypothesis Tests

Dependent Variable	Predictors
CD4 count (6 mo.)	1) Introversive Coping (High vs. Low) 2) CD4 Baseline (covariate)
CD4 count (12 mo.)	1) Introversive Coping (High vs. Low) 2) CD4 Baseline (covariate)
CD4 count (18 mo.)	1) Introversive Coping (High vs. Low) 2) CD4 Baseline (covariate)
CD4 count (24 mo.)	1) Introversive Coping (High vs. Low) 2) CD4 Baseline (covariate)

utilizing a multivariate analysis of variance (MANOVA) model incorporating CD4 measures at 6 and 12 months as simultaneous dependent variables to be predicted. While it would have been desirable to incorporate all four data points (6, 12, 18, and 24 months) as dependent variables in the MANOVA analysis, this was not possible given the attrition and loss of CD4 data at the later data points.

Elaboration Using Hierarchical Stepwise Multiple Regression

In addition to the specific hypothesis tests, exploratory multiple regression models were generated to explore the relative predictive effects of the independent variables on CD4 counts while controlling for baseline CD4 counts. The multivariate hypothesis was rephrased as:

> CD4 levels at each time period after baseline (6 months, 12 months, 18 months, and 24 months) can be predicted by a multivariate combination of the coping styles, the psychosomatic correlates scales, the psychogenic attitudes scales, and level of Depression, while controlling for the effects of Baseline CD4 counts.

This hypothesis was tested with a set of stepwise multiple regression models with Baseline CD4 counts being entered as a control variable in each model.

In addition to the hypothesis tests, the relationship between background/demographic variables and all study variables was assessed.

Assumptions and Limitations

This study was necessarily limited by the relatively small sample size. Another limitation is that no single factor is truly the cause of an outcome, with some combination of interacting variables jointly yielding particular outcomes. This type of research design does not allow for random assignment to study groups. Another limitation to the study pertains to the validity of the instruments used. Self-report inventories are notoriously vulnerable to misrepresentation, and it is necessary to assume that subjects were sincere and honest in their responses to the battery of instruments.

The issue of attrition should also be addressed. There was considerably less data available for analysis at subsequent waves (i.e., waves of data collection after the Baseline). A certain amount of this attrition was attributable to mortality and physical incapacitation, with additional loss of data being due to difficulties in following up. The proportion of subjects using AZT was similar for each of the "wave" groups (the subjects for whom data were available at 6-months, 12-months, 18-months, and 24-months). No statistically significant differences were found in the high or low groups for any MBHI scales or for the BDI.

The attrition issue was particularly salient in the data analysis phase, in that the analysis required that data be available for both Baseline and the later data point (i.e., in the analysis of covariance framework). This led to smaller sample sizes than would have been optimal.

Ethical Assurances

This study is a component of a larger existing study, which has already undergone extensive Institutional Review (see Appendix for Consent Form). The larger study involved invasive procedures, and all ethical standards of the APA were followed.

The participants were reassured that all data would be kept confidential. In order to insure confidentiality, each participant was assigned an anonymous identification number. All data forms were stored in a locked filing cabinet in the Neuropsychology Laboratory of the Brentwood Division of the West Los Angeles Veterans Affairs Medical

Center. Each form was identified only by an anonymous code number, and the codebook with identifying information was kept in a locked cabinet in the office of the Principal Investigator.

The data utilized in the current study were strictly questionnaire data, and did not involve clinical or medical intervention. Participants were informed that they were participating in a research project as part of a doctoral dissertation, and had all aspects of their participation explained to them. They also had any questions answered. Each subject was also informed that he had the right to withdraw from the study at any time without prejudice, and that all data would be treated confidentially and used for research purposes only. Each subject was also told that results of the study would be made available to him if he expressed an interest.

It was not anticipated that the content of the questionnaires would be upsetting to the subjects, and no adverse psychological reactions were observed.

CHAPTER IV

RESULTS

Preliminary Analysis

Initial analysis was done comparing HIV seropositive subjects to the HIV seronegative control group on the Baseline MBHI Coping Styles. Comparisons were made for all subjects for whom baseline MBHI data were available. Differences between the two groups were evaluated using the independent groups *t*-test statistic. Results, shown in Table 5, indicated that the HIV seropositive subjects scored significantly lower on Social Coping ($p < .001$) and Confident Coping ($p < .05$), and significantly higher on Inhibited Coping ($p < .01$) and Sensitive Coping ($p < .01$).

Table 5

Comparison of HIV Seropositive Subjects (N = 73) to HIV Seronegative Subjects (N = 28) on MBHI Coping Scales

	Group				
	HIV+		HIV-		
Coping Style	Mean	SD	Mean	SD	*t* test
Introversive	41.52	28.27	48.75	20.82	1.51
Inhibited	61.51	35.25	39.61	24.26	9.12**
Cooperative	45.80	28.38	53.64	32.34	1.43
Social	35.79	29.63	60.68	24.81	15.54***
Confident	39.90	28.22	53.50	23.93	5.09*
Forceful	46.06	30.99	40.39	33.68	0.67
Respectful	52.19	20.33	48.39	17.16	0.77
Sensitive	56.92	32.16	35.21	23.98	10.49**

*$p<.05$; **$p<.01$; ***$p<.001$

Thus, there were differences on four of the eight MBHI Coping Style Scales, all in the expected direction.

A similar analysis was done with the six Baseline Psychogenic Attitudes scales as outcomes. Results, shown in Table 6, indicated significant differences on all six variables. Analysis of the three Baseline Psychosomatic Correlates scales also showed significant

Table 6

Comparison of HIV Seropositive Subjects (N = 73) to HIV Seronegative Subjects (N=28) on MBHI Psychogenic Attitudes Scales

Psychogenic Attitude	Group				
	HIV+		HIV-		
	Mean	SD	Mean	SD	t test
Tension	49.62	30.94	37.03	24.62	3.72*
Stress	64.56	26.57	39.14	19.86	21.05***
Pessimism	59.72	30.58	31.14	21.49	20.52***
Despair	63.62	35.84	40.39	33.35	8.82**
Alienation	56.29	34.53	37.64	25.18	2.99**
Anxiety	62.25	23.82	34.86	19.88	29.18***

*$p<.05$; **$p<.01$; ***$p<.001$

differences on all three scales; in all three cases, HIV seropositive subjects scored significantly higher on the psychosomatic correlates scales (Table 7). A final comparison between the two groups was done with the Beck Depression Inventory at Baseline and 12-month time frames as dependent variable. Results, shown in Table 8, revealed that HIV

seropositive subjects were significantly more depressed at both Baseline ($p < .001$) and 12 months ($p < .01$).

Table 7

Comparison of HIV Seropositive Subjects (N = 73) to HIV Seronegative Subjects (N=28) on MBHI Psychosomatic Correlates Scales

Psychosomatic Correlates	Group				
	HIV+		HIV-		
	Mean	SD	Mean	SD	t-test
Allergic Inclination	70.71	24.22	44.79	20.49	25.15***
Gastrointestinal	71.34	18.88	40.50	13.36	62.54***
Cardiovascular	68.51	24.47	50.75	17.55	12.28***

***p<.001

Table 8

Comparison of HIV Seropositive Subjects (N = 73) to HIV Seronegative Subjects (N = 28) on Beck Depression Scores, Baseline, and 12-Month Time Periods

	Group				
	HIV+		HIV-		
	Mean	SD	Mean	SD	t-test
Depression, Baseline	13.39	8.52	3.67	4.09	32.46***
Depression, 12 Months	7.25	10.39	1.97	2.87	7.27**

**p < .01; *** p <.001

Hypothesis Tests

The first set of eight hypotheses utilizes the eight coping style scales of the MBHI as independent variables.

Hypothesis #1

Subjects who score higher on Introversive Coping at Baseline will not differ on CD4 counts at 6 months, 12 months, 18 months, and 24 months from subjects with lower Introversive Coping scores at Baseline, while controlling for the effects of Baseline CD4 counts.

This hypothesis was tested using a set of four analysis of covariance models. Each model had one of the four CD4 counts as dependent variable (6, 12, 18, and 24 months), with CD4 counts at Baseline being used as a covariate. The independent variable is a dichotomous variable reflecting low versus high levels of Introversive Coping, defined by the median split. This method was then replicated for the other hypothesis tests, with the independent variable in each case being a 2-level nominal level variable reflecting low versus high levels of that particular coping style.

Results of the first hypothesis test are shown in Table 9. As can be seen, there are no significant differences on CD4 counts at any of the time periods, while controlling for Baseline CD4 levels. Thus, the null hypothesis is supported, and there is no evidence of a relationship between Introversive Coping and immune system functioning.

Table 9

Effects of Introversive Coping Style on CD4 Counts at Each Time Period, While Controlling for Baseline CD4 Counts

		Introversive Coping				
		Low		High		
	N	Mean	SD	Mean	SD	F Test
CD4 Count, 6 Months	57	381.7	249.3	327.5	208.6	0.38
CD4 Count, 12 Months	45	293.1	184.9	305.3	218.2	0.43
CD4 Count, 18 Months	26	297.8	189.9	198.4	198.5	0.80
CD4 Count, 24 Months	24	236.6	178.9	252.6	247.5	0.46

Note: Each ANCOVA model controls CD4 at Baseline as a covariate in the model; Table shows F test for the effect of Coping style with covariate in the model.

Hypothesis #2

Subjects who score higher on Inhibited Coping at Baseline will not differ on CD4 counts at 6 months, 12 months, 18 months, and 24 months from subjects with lower Inhibited Coping scores at baseline, while controlling for the effects of Baseline CD4 counts.

Results of the second hypothesis test, shown in Table 10, also show no significant differences between those who exhibit low levels of Inhibited Coping as compared to those with high levels of Inhibited Coping. Thus, the second null hypothesis is supported, and there is no evidence of a relationship between Inhibited Coping and immune system functioning.

Table 10

Effects of Inhibited Coping Style on CD4 Counts at Each Time Period, While Controlling for Baseline CD4 Counts

		Inhibited Coping				
		Low		High		
	N	Mean	SD	Mean	SD	F Test
CD4 Count, 6 Months	57	283.3	144.19	407.1	265.5	2.33
CD4 Count, 12 Months	45	263.3	196.8	327.9	203.7	0.01
CD4 Count, 18 Months	26	218.2	177.6	292.8	205.9	0.01
CD4 Count, 24 Months	24	207.0	200.8	266.9	179.5	0.04

Note: Each ANCOVA model controls CD4 at Baseline as a covariate in the model; Table shows F test for the effect of Coping style with covariate in the model.

Hypothesis #3

Subjects who score higher on Cooperative Coping at Baseline will not differ on CD4 counts at 6 months, 12 months, 18 months, and 24 months from subjects with lower Cooperative Coping scores at Baseline, while controlling for the effects of Baseline CD4 counts.

Results of the third hypothesis test, shown in Table 11, are similar, with no significant differences being found between those who exhibit low levels of Cooperative Coping as compared to those with high levels of Cooperative Coping. Thus, the third null hypothesis is supported, and there is no evidence of a relationship between Cooperative Coping and immune system functioning.

Table 11

Effects of Cooperative Coping Style on CD4 Counts at Each Time Period, While Controlling for Baseline CD4 Counts

		Cooperative Coping				
		Low		High		
	N	Mean	SD	Mean	SD	F Test
CD4 Count, 6 Months	57	344.7	238.9	373.8	226.3	0.87
CD4 Count, 12 Months	45	291.5	166.5	307.5	237.4	0.17
CD4 Count, 18 Months	26	300.2	148.4	193.9	257.7	2.60
CD4 Count, 24 Months	24	255.5	118.2	223.5	290.0	0.34

Note: Each ANCOVA model controls CD4 at Baseline as a covariate in the model; Table shows F test for the effect of Coping style with covariate in the model.

Hypothesis #4

Subjects who score higher on Sociable Coping at Baseline will not differ on CD4 counts at 6 months, 12 months, 18 months, and 24 months from subjects with lower Sociable Coping scores at Baseline, while controlling for the effects of Baseline CD4 counts.

Results of the fourth hypothesis test, shown in Table 12, show statistically significant differences between those who exhibit low levels of Sociable Coping as compared to those with high levels of Sociable Coping at 18 months. There is a tendency for subjects with higher levels of sociable coping to have somewhat lower CD4 cell counts at 6 months, as well, but this difference was not significant. Thus, the fourth null hypothesis is not supported, and there is evidence of a relationship between Sociable Coping and immune system functioning.

Table 12

Effects of Sociable Coping Style on CD4 Counts at Each Time Period, While Controlling for Baseline CD4 Counts

		Sociable Coping				
		Low		High		
	N	Mean	SD	Mean	SD	F Test
CD4 Count, 6 Months	57	422.6	264.4	268.6	138.2	3.27
CD4 Count, 12 Months	45	341.0	197.3	253.2	191.7	0.01
CD4 Count, 18 Months	26	349.9	191.7	156.8	142.2	4.41*
CD4 Count, 24 Months	24	269.8	170.8	209.5	236.9	0.04

Note: Each ANCOVA model controls CD4 at Baseline as a covariate in the model; Table shows F test for the effect of Coping style with covariate in the model.

*$p<.05$

Hypothesis #5

Subjects who score higher on Confident Coping at Baseline will not differ on CD4 counts at 6 months, 12 months, 18 months, and 24 months from subjects with lower Confident Coping scores at Baseline, while controlling for the effects of Baseline CD4 counts.

Results of the fifth hypothesis test, shown in Table 13, revealed no significant differences between those who exhibit low levels of Confident Coping as compared to those with high levels of Confident Coping. Thus, the fifth null hypothesis is supported, and there is no evidence of a relationship between Confident Coping and immune system functioning.

Table 13

Effects of Confident Coping Style on CD4 Counts at Each Time Period, While Controlling for Baseline CD4 Counts

		Confident Coping				
		Low		High		
	N	Mean	SD	Mean	SD	F test
CD4 Count, 6 Months	57	379.1	257.1	335.2	204.2	0.12
CD4 Count, 12 Months	45	314.6	182.1	287.6	209.8	0.41
CD4 Count, 18 Months	26	290.4	234.2	235.6	151.0	0.01
CD4 Count, 24 Months	24	247.0	210.9	239.2	202.1	0.16

Note: Each ANCOVA model controls CD4 at Baseline as a covariate in the model; Table shows F test for the effect of Coping style with covariate in the model.

Hypothesis #6

Subjects who score higher on Forceful Coping at Baseline will not differ on CD4 counts at 6 months, 12 months, 18 months, and 24 months from subjects with lower Forceful Coping scores at Baseline, while controlling for the effects of Baseline CD4 counts.

Results of the sixth hypothesis test, shown in Table 14, showed no significant differences between those classified as having low levels of Forceful Coping as compared to those with high levels of Forceful Coping. Thus, the sixth null hypothesis is supported, and there is no evidence of a relationship between Forceful Coping and immune system functioning.

Table 14

Effects of Forceful Coping Style on CD4 Counts at Each Time Period, While Controlling for Baseline CD4 Counts

		Forceful Coping				
		Low		High		
	N	Mean	SD	Mean	SD	F Test
CD4 Count, 6 Months	57	338.2	232.7	376.2	233.4	0.22
CD4 Count, 12 Months	45	300.5	232.1	296.8	172.1	1.20
CD4 Count, 18 Months	26	201.0	237.3	304.3	156.3	1.36
CD4 Count, 24 Months	24	237.3	276.8	245.9	139.4	0.01

Note: Each ANCOVA model controls CD4 at Baseline as a covariate in the model; Table shows F test for the effect of Coping style with covariate in the model.

Hypothesis #7

Subjects who score higher on Respectful Coping at Baseline will not differ on CD4 counts at 6 months, 12 months, 18 months, and 24 months from subjects with lower Respectful Coping scores at Baseline, while controlling for the effects of Baseline CD4 counts.

Results of the seventh hypothesis test are shown in Table 15. Results show no significant differences between those subjects with low levels of Respectful Coping as compared to those with high levels of Respectful Coping. Thus, the seventh null hypothesis is supported, and there is no evidence of a relationship between Respectful Coping and immune system functioning.

Table 15

Effects of Respectful Coping Style on CD4 Counts at Each Time Period, While Controlling for Baseline CD4 Counts

		Respectful Coping				
		Low		High		
	N	Mean	SD	Mean	SD	F Test
CD4 Count, 6 Months	57	390.3	254.7	335.8	216.3	0.01
CD4 Count, 12 Months	45	340.3	204.1	276.5	193.9	0.51
CD4 Count, 18 Months	26	289.8	185.5	245.2	205.4	0.72
CD4 Count, 24 Months	24	318.5	237.6	198.9	170.6	1.43

Note: Each ANCOVA model controls CD4 at Baseline as a covariate in the model; Table shows F test for the effect of Coping style with covariate in the model.

Hypothesis #8

Subjects who score higher on Sensitive Coping at Baseline will not differ on CD4 counts at 6 months, 12 months, 18 months, and 24 months from subjects with lower Sensitive Coping scores at Baseline, while controlling for the effects of Baseline CD4 counts.

Results of the eighth hypothesis test, shown in Table 16, are similar. There are no significant differences between those who exhibit low levels of Sensitive Coping compared to those with high levels of Sensitive Coping. There was a tendency for subjects who scored lower on sensitive coping to have somwhat lower CD4 counts at 6 months, but this difference was not quite significant. Thus, the eighth null hypothesis is supported, and there is no evidence of a relationship between Sensitive Coping and immune system functioning.

Table 16

Effects of Sensitive Coping Style on CD4 Counts at Each Time Period, While Controlling for Baseline CD4 Counts

		Respectful Coping				
		Low		High		
	N	Mean	SD	Mean	SD	F Test
CD4 Count, 6 Months	57	290.5	136.8	394.6	264.5	2.72
CD4 Count, 12 Months	45	281.3	178.2	309.8	211.9	0.01
CD4 Count, 18 Months	26	200.7	169.8	314.0	204.9	0.48
CD4 Count, 24 Months	24	235.0	225.9	247.5	190.9	0.23

Note: Each ANCOVA model controls CD4 at Baseline as a covariate in the model; Table shows F test for the effect of Coping style with covariate in the model.

Results of the first eight hypotheses, each of which postulated one of the MBHI Coping Styles as dependent variables, showed no significant findings, except for Sociable Coping at 18 months. Thus, it appears that there is little or no relationship between Millon's coping styles and immune system functioning in an HIV-infected sample.

The next set of hypotheses was tested in similar fashion. Each hypothesis states a null relationship between one of the MBHI psychogenic attitudes scales and immune system functioning, as operationalized by CD4 cell counts.

Hypothesis #9

Subjects who score higher on Chronic Tension at Baseline will not differ on CD4 counts at 6 months, 12 months, 18 months, and 24 months

from subjects with lower Chronic Tension scores at Baseline, while controlling for the effects of Baseline CD4 counts.

As shown in Table 17, there was a significant effect of Chronic Tension on CD4 counts at both 6 months and 18 months. In both cases, those with higher levels of chronic tension had lower CD4 counts ($p < .01$ for both 6 and 18 months). Thus, the null hypothesis can be rejected. There is a degree of support for the directional hypothesis, with higher levels of chronic tension being associated with impaired immune system functioning.

Table 17

Effects of Chronic Tension on CD4 Counts at Each Time Period, While Controlling for Baseline CD4 Counts

		Chronic Tension				
		Low		High		
	N	Mean	SD	Mean	SD	F Test
CD4 Count, 6 Months	57	421.9	254.2	227.3	172.1	7.88**
CD4 Count, 12 Months	45	324.8	190.8	266.9	205.4	1.60
CD4 Count, 18 Months	26	356.9	131.3	169.1	206.2	7.51**
CD4 Count, 24 Months	24	354.2	149.2	230.6	249.1	0.14

Note: Each ANCOVA model controls CD4 at Baseline as a covariate in the model; Table shows F test for the effect of Coping style with covariate in the model.

**$p < .01$

Hypothesis #10

Subjects who score higher on Recent Stress at Baseline will not differ on CD4 counts at 6 months, 12 months, 18 months, and 24 months from subjects with lower Recent Stress scores at Baseline, while controlling for the effects of Baseline CD4 counts.

Results of the tenth hypothesis test, shown in Table 18, reveal that there is no significant difference between those subjects with low levels of Recent Stress and those with high levels of Recent Stress. Thus, the tenth null hypothesis is supported, and there is no evidence of a relationship between Recent Stress and immune system functioning.

Table 18

Effects of Recent Stress on CD4 Counts at Each Time Period, While Controlling for Baseline CD4 Counts

		Recent Tension				
		Low		High		
	N	Mean	SD	Mean	SD	F Test
CD4 Count, 6 Months	57	390.0	280.9	313.4	131.9	0.99
CD4 Count, 12 Months	45	313.3	210.9	278.5	181.5	0.01
CD4 Count, 18 Months	26	265.6	187.4	214.6	222.5	0.02
CD4 Count, 24 Months	24	314.0	204.9	200.7	169.8	0.48

Note: Each ANCOVA model controls CD4 at Baseline as a covariate in the model; Table shows F test for the effect of Coping style with covariate in the model.

Hypothesis #11

Subjects who score higher on Premorbid Pessimism at Baseline will not differ on CD4 counts at 6 months, 12 months, 18 months, and 24

months from subjects with lower Premorbid Pessimism scores at Baseline, while controlling for the effects of Baseline CD4 counts.

As shown in Table 19, there is a significant effect of Premorbid Pessimism on CD4 counts at 6 months. Individuals with higher levels of Premorbid Pessimism had lower CD4 counts at 6 months ($p < .05$). Thus, the null hypothesis of no relationship can be rejected. The directional hypothesis is supported, with results showing an effect of Baseline Pessimism on CD4 counts 6 months later.

Table 19

Effects of Premorbid Pessimism on CD4 Counts at Each Time Period, While Controlling for Baseline CD4 Counts

		Premorbid Pessimism				
		Low		High		
	N	Mean	SD	Mean	SD	F Test
CD4 Count, 6 Months	57	405.9	273.0	291.5	138.2	5.29*
CD4 Count, 12 Months	45	338.4	213.3	260.6	177.6	1.53
CD4 Count, 18 Months	26	303.1	214.1	214.0	164.5	1.27
CD4 Count, 24 Months	24	266.1	180.7	208.2	233.6	0.46

Note: Each ANCOVA model controls CD4 at Baseline as a covariate in the model; Table shows F test for the effect of Coping style with covariate in the model.

*$p < .05$

Hypothesis #12

Subjects who score higher on Future Despair at Baseline will not differ on CD4 counts at 6 months, 12 months, 18 months, and 24 months

from subjects with lower Future Despair scores at baseline, while controlling for the effects of Baseline CD4 counts.

As shown in Table 20, there is a significant effect of Future Despair on CD4 counts at 6 months. Individuals with higher levels of future despair had lower CD4 counts at 6 months ($p < .05$). Thus, the null hypothesis of no relationship between Future Despair and immune system functioning can be rejected. The directional hypothesis is supported for the effect of Future Despair on CD4 counts 6 months later.

Table 20

Effects of Future Despair on CD4 Counts at Each Time Period, While Controlling for Baseline CD4 Counts

		Future Despair				
		Low		High		
	N	Mean	SD	Mean	SD	F Test
CD4 Count, 6 Months	57	399.6	240.5	300.2	210.4	3.87*
CD4 Count, 12 Months	45	324.6	171.2	270.6	222.7	1.09
CD4 Count, 18 Months	26	307.2	203.8	181.0	154.4	0.92
CD4 Count, 24 Months	24	264.6	172.6	203.5	250.6	0.12

Note: Each ANCOVA model controls CD4 at Baseline as a covariate in the model; Table shows F test for the effect of Coping style with covariate in the model.

*$p<.05$

Hypothesis #13

Subjects who score higher on Social Alienation at Baseline will not differ on CD4 counts at 6 months, 12 months, 18 months, and 24

months from subjects with lower Social Alienation scores at Baseline, while controlling for the effects of Baseline CD4 counts.

Results of this hypothesis test, shown in Table 21, show that there were no significant differences between subjects with low Alienation as compared to those with high levels of Alienation. There was a tendency for those with high levels of Social Alienation to have somewhat lower CD4 counts at 6 months; however, this is not a significant finding. Thus, the null hypothesis is supported, and there is no evidence of a relationship between Social Alienation and immune system functioning.

Table 21

Effects of Social Alienation on CD4 Counts at Each Time Period, While Controlling for Baseline CD4 Counts

		Social Alienation				
		Low		High		
	N	Mean	SD	Mean	SD	F Test
CD4 Count, 6 Months	57	398.3	264.6	290.5	145.3	2.02
CD4 Count, 12 Months	45	327.9	203.7	263.3	188.7	0.01
CD4 Count, 18 Months	26	274.5	207.9	241.6	178.1	0.04
CD4 Count, 24 Months	24	249.7	184.1	229.6	240.1	0.01

Note: Each ANCOVA model controls CD4 at Baseline as a covariate in the model; Table shows F test for the effect of Coping style with covariate in the model.

Hypothesis #14

Subjects who score higher on Somatic Anxiety at Baseline will not differ on CD4 counts at 6 months, 12 months, 18 months, and 24 months

from subjects with lower Somatic Anxiety scores at Baseline, while controlling for the effects of Baseline CD4 counts.

As shown in Table 22, there is a significant effect of Somatic Anxiety on CD4 counts at 6 months. Individuals with higher Somatic Anxiety had lower CD4 counts at 6 months ($p < .01$). There is a tendency at the 18 month time period, with the effect holding up in the predicted direction (but not quite significant). Thus, the null hypothesis can be rejected, and there is an effect of Somatic Anxiety on CD4 counts 6 months later.

Table 22

Effects of Somatic Anxiety on CD4 Counts at Each Time Period, While Controlling for Baseline CD4 Counts

		Somatic Anxiety				
		Low		High		
	N	Mean	SD	Mean	SD	F Test
CD4 Count, 6 Months	57	407.7	259.6	275.2	112.4	6.86**
CD4 Count, 12 Months	45	317.8	219.6	272.5	165.5	0.24
CD4 Count, 18 Months	26	329.7	213.9	162.9	104.8	2.86
CD4 Count, 24 Months	24	284.7	184.9	200.1	215.6	0.32

Note: Each ANCOVA model controls CD4 at Baseline as a covariate in the model; Table shows F test for the effect of Coping style with covariate in the model.

**p<.01.

Hypothesis #15

Subjects who score higher on Allergic Inclination at Baseline will not differ on CD4 counts at 6 months, 12 months, 18 months, and 24

months from subjects with lower Allergic Inclination scores at Baseline, while controlling for the effects of Baseline CD4 counts.

As can be seen in Table 23, there is a significant effect of Allergic Inclination on CD4 counts at 6 months. Individuals with higher levels of Allergic Inclination had lower CD4 counts at 6 months ($p < .05$). Thus, the null hypothesis can be rejected, with the findings showing a significant effect of Allergic Inclination on CD4 counts 6 months later.

Table 23

Effects of Allergic Inclination on CD4 Counts at Each Time Period, While Controlling for Baseline CD4 Counts

		Allergic Inclination				
		Low		High		
	N	Mean	SD	Mean	SD	F Test
CD4 Count, 6 Months	57	389.9	273.6	299.2	106.7	5.62*
CD4 Count, 12 Months	45	313.8	215.8	277.9	173.5	0.34
CD4 Count, 18 Months	26	322.2	207.3	174.3	139.7	1.77
CD4 Count, 24 Months	24	262.8	180.3	213.0	235.3	0.66

Note: Each ANCOVA model controls CD4 at Baseline as a covariate in the model; Table shows F test for the effect of Coping style with covariate in the model.

*$p<.05$

Hypothesis #16

Subjects who score higher on Gastrointestinal Susceptibility at Baseline will not differ on CD4 counts at 6 months, 12 months, 18 months, and 24 months from subjects with lower Gastrointestinal

Susceptibility scores at Baseline, while controlling for the effects of Baseline CD4 counts.

Table 24 shows that there is a significant effect of Gastrointestinal Susceptibility on CD4 counts at both 6 months and 18 morths (both significant at $p < .01$).

Table 24

Effects of Gastrointesntinal Susceptibility on CD4 Counts at Each Time Period, While Controlling for Baseline CD4 Counts

		Gastrointestinal Susceptibility				
		Low		High		
	N	Mean	SD	Mean	SD	F Test
CD4 Count, 6 Months	57	398.7	258.6	266.7	116.5	7.03**
CD4 Count, 12 Months	45	331.4	206.4	248.8	177.1	2.79
CD4 Count, 18 Months	26	324.9	197.3	118.5	76.0	7.21**
CD4 Count, 24 Months	24	280.0	169.6	188.1	238.8	0.95

Note: Each ANCOVA model controls CD4 at Baseline as a covariate in the model; Table shows F test for the effect of Coping style with covariate in the model.

**p<.01

In both cases, those with higher levels of Gastrointestinal Susceptibility had lower CD4 counts. Additionally, there is a nearly significant difference at the 12-month time period. Thus, the null hypothesis can be rejected. There is support for the effect of Allergic Inclination on CD4 counts 6 and 18 months later.

Hypothesis #17

Subjects who score higher on Cardiovascular Tendency at Baseline will not differ on CD4 counts at 6 months, 12 months, 18 months, and 24 months from subjects with lower Cardiovascular Tendency scores at Baseline, while controlling for the effects of Baseline CD4 counts.

Table 25 shows that there is a significant effect of Cardiovascular Tendency on CD4 counts at both 6 months ($p < .01$) and 18 months ($p < .05$). In both cases, those with higher levels of Cardiovascular Tendency had lower CD4 counts. This implies that the null hypothesis can be rejected. These results provide statistical support for the effect of Cardiovascular Tendency on CD4 counts at 6 and 18 months.

Table 25

Effects of Cardiovascular Tendency on CD4 Counts at Each Time Period, While Controlling for Baseline CD4 Counts

	Cardiovascular Tendency					
	Low			High		
	N	Mean	SD	Mean	SD	F Test
CD4 Count, 6 Months	57	403.6	276.4	294.7	131.7	6.44**
CD4 Count, 12 Months	45	309.9	221.0	284.7	169.8	0.26
CD4 Count, 18 Months	26	348.5	195.1	158.4	138.9	5.24*
CD4 Count, 24 Months	24	283.9	170.6	192.6	231.1	1.49

Note: Each ANCOVA model controls CD4 at Baseline as a covariate in the model; Table shows F test for the effect of Coping style with covariate in the model.

*$p<.05$; **$p<.01$

Hypothesis #18

Subjects who score higher on Depression at Baseline will not differ on CD4 counts at 6 months, 12 months, 18 months, and 24 months from subjects with lower Depression scores at Baseline, while controlling for the effects of Baseline CD4 counts.

Results of this hypothesis test, shown in Table 26, show that there are no significant differences between those who exhibit low levels of Depression compared to those with high levels of Depression. There was a tendency for subjects who scored lower on Depression to have somewhat higher CD4 counts at 6 months, but this difference was not quite significant. Thus, the null hypothesis is supported, and there is no evidence of a relationship between Baseline Depression and immune system functioning.

Hypothesis #19

CD4 levels at each time period after baseline (6 months, 12 months, 18 months, and 24 months) cannot be predicted by a multivariate combination of the coping styles, the psychosomatic correlate scales, the psychogenic attitudes scales, and level of Depression, while controlling for the effects of Baseline CD4 counts.

This multivariate hypothesis was tested by specifying a stepwise multiple regression. The initial regression analysis, shown in Table 27, has CD4 counts at 6 months as the dependent variable. In the first step, Baseline CD4 count is forced into the regression model as an independent variable. As expected, it is highly significant and accounts for 54.3% of the variance in 6 month CD4 counts.

Table 26

Effects of Depression (Baseline) on CD4 Counts at Each Time Period, While Controlling for Baseline CD4 Counts

		Depression				
		Low		High		
ANCOVA	N	Mean	SD	Mean	SD	F Test
CD4 Count, 6 Months	57	408.4	262.1	278.2	142.5	3.06
CD4 Count, 12 Months	45	315.2	201.4	273.0	170.9	0.05
CD4 Count, 18 Months	26	244.3	210.9	199.7	170.8	0.03
CD4 Count, 24 Months	24	246.4	192.3	214.6	222.5	0.01

Note: Each ANCOVA model controls CD4 at Baseline as a covariate in the model; Table shows F test for the effect of Coping style with covariate in the model.

Table 27

Exploratory Multiple Regression Analysis: Model #1 Dependent Variable: CD4 Counts, 6 Months

	Step of the Model	
Independent Variable	1	2
CD4 Count, Baseline	0.74***	0.72***
Cardiovascular		0.35***
F statistic	50.87***	41.52***
Adjusted r-square (variance explained)	54.3%	65.9%

Note: Cells show standardized beta coefficients.

***p<.001

In the second step of the analysis, the entire set of independent variables was offered for consideration in a stepwise regression framework (see Table 27). The only independent variable selected as significant was Cardiovascular Tendency, which explained an additional 11.6% of the variance in the dependent variable.

The second stepwise regression model, shown in Table 28, replicated this procedure with CD4 counts at 12 months as the dependent variable. Baseline CD4 counts, again forced in the first step of the model, accounted for 64.1% of the variance in 12-month CD4 counts. In this stepwise model, both Cooperative and Introversive Coping styles were selected in subsequent steps.

Table 28

Exploratory Multiple Regression Analysis: Model #2 Dependent Variable: CD4 Counts, 12 Months

	Step of the Model		
Independent Variable	1	2	3
CD4 Count, Baseline	0.77***	0.83***	0.92***
Cooperative Coping		0.24*	0.36**
Introversive Coping			0.26*
F-statistic	77.79***	34.32***	29.18***
Adjusted r-square (variance explained)	64.1%	66.9%	71.9%

Note: Cells show standardized beta coefficients.

*$p<.05$; **$p<.01$; ***$p<.001$

Although both of these coping styles are significant (Cooperative at $\underline{p} < .01$ and Introversive at $\underline{p} < .05$), the combination of the two increases the variance explained only slightly.

In addition, the hypothesis tests were elaborated by calculating a repeated measures MANOVA for each hypothesis. Each such analysis incorporated CD4 measures as joint dependent variables and the specific scale for each hypothesis as independent variables (predictors). These MANOVA models, shown in Table 29, incorporated three simultaneous dependent variables: CD4 counts at baseline, 6 months, and 12 months. It was impractical to include CD4 counts at 18 and 24 months as dependent variables due to attrition in the sample (i.e., due to inadequate cases with full CD4 data). A separate MANOVA model was run for each hypothesis test. In each case the three dependent variables where CD4 counts at baseline, 6 months, and 12 months. The independent variable was the MBHI measure specified in Hypotheses 1 through 17, or the Beck Depression Scale for Hypothesis 18.

The lack of significance in the MANOVA models is surprising, especially given the significant findings in the original hypothesis tests for the psychogenic attitudes and psychosomatic correlates scales. However, this can probably be attributed to the drop in the number of valid cases, and to statistical artifacts resulting from insufficient degrees of freedom in the MANOVA models.

Table 29

Multivariate Analysis of Variance With CD4 at Baseline, 6 Months, and 12 Months as Dependent Variables

H#	Variable	F test	p value
1	Introversive	0.96	0.39
2	Inhibited	1.02	0.36
3	Cooperative	1.40	0.26
4	Social	1.14	0.33
5	Confident	1.41	0.25
6	Forceful	1.26	0.29
7	Respectful	0.53	0.59
8	Sensitive	0.83	0.44
9	Tension	1.21	0.30
10	Stress	1.00	0.38
11	Pessimism	1.12	0.33
12	Despair	1.13	0.33
13	Alienation	1.05	0.36
14	Anxiety	1.14	0.33
15	Allergic Inclination	1.07	0.35
16	Gastrointestinal	1.32	0.27
17	Cardiovascular	1.14	0.33
18	Depression	1.18	0.31

CHAPTER V

DISCUSSION

The current study addressed the issue of the relationship between psychological well-being and immune function in a sample of HIV seropositive males drawn from the patient population at the Veteran's Affairs Medical Center in West Los Angeles. This chapter will (a) summarize the methods of the study and the results reported in Chapter IV, (b) discuss those findings in light of the literature review, (c) draw relevant conclusions from the findings, and (d) discuss some fruitful directions for future research.

Summary

Summary of Methods

The current study employed a longitudinal, quasi-experimental design to assess the relationship between various psychological independent variables and immune system functioning, operationalized as CD4 counts, in a cohort of AIDS patients over a 24-month time period. The study involved the collection of a battery of psychological and neuropsychological instruments and CD4 data at Baseline, as well as data on depression at Baseline and 12-months and CD4 counts at 6-month intervals over a 2-year period. The study analyzed the effect of coping style, stress, and somatic symptoms (the MBHI measures), as well as the effects of Baseline depression, on immune system functioning over the 2-year period.

Summary of Preliminary Analysis

Results of the preliminary analyses comparing HIV seropositive to HIV seronegative subjects were generally consistent with what might be expected given what we know about the effects of the HIV diagnosis.

There were differences on four of the eight MBHI coping style scales. HIV seropositive subjects scored significantly higher on the inhibited and sensitive coping scales. Similarly, they scored lower on confident and social coping scales. There were consistent significant differences between the two groups on all of the psychogenic attitudes, with the HIV seropositive subjects experiencing a great deal more stress, as measured by these variables.

Summary of Hypothesis Tests

There was only one observed effect of the MBHI Coping Styles (Sociable at 18 months). No other effects were observed on immune system functioning. Based on the findings of the first set of eight hypotheses with the Coping Styles as dependent variables, it appears that there is little or no relationship between Millon's coping styles and immune system functioning, at least in this sample of HIV-positive subjects.

The second set of hypotheses (Hypotheses 9 through 14) dealt with the six psychogenic attitudes as dependent variables. Results were somewhat more encouraging with this block of MBHI variables, with significance being found on four of the six psychogenic attitudes. Specifically, results of these hypotheses tests are summarized in Table 30.

Table 30

Summary of Hypothesis Tests With Psychogenic Attitudes as Predictors of CD4 Counts

Psychogenic Attitude	Result
Chronic Tension	Significant at 6 and 18 Months
Recent Stress	Not Significant
Premorbid Pessimism	Significant at 6 Months
Future Despair	Significant at 6 Months
Social Alienation	Not Significant
Somatic Anxiety	Significant at 6 Months

There were also significant effects of the Psychosomatic Correlates scales on immune functioning, with significant findings on Allergic Inclination, Gastrointestinal Susceptibility, and Cardiovascular Tendency. However, there was no effect of level of depression on immune system functioning.

Discussion of Findings

Chronic stress and anxiety are normal correlates of the HIV-positive diagnosis, with various researchers reporting that over half of HIV-positive gay males experience elevated rates of anxiety and insomnia. While a certain amount of anxiety may be useful, in that it encourages adaptive behaviors and compliance to medical regimens, excessive anxiety can be quite harmful in several areas. For example, excessive anxiety is likely to contribute to a sense of helplessness

and to a perceived inability to do anything about the situation (Seligman's learned helplessness). One possible negative outcome is that the chronically stressed individual may continue to engage in maladaptive behaviors due to the totally external locus of control orientation.

High levels of Chronic Tension, as measured by the MBHI, are likely to be related to various psychosomatic and physical ailments (Millon et al., 1982). Individuals who have higher levels of chronic tension typically maintain a rapid pace and live under a great deal of self-imposed pressure. It is quite possible that such a lifestyle could contribute to an impaired immune system function in a person with AIDS, especially if the rapid pace is maintained over a long period of time.

In addition, higher levels of premorbid pessimism were associated with impaired immune system functioning (at 6 months). This may be related to the fact that some individuals tend to adopt a dispositional attitude characterized by feelings of helplessness. The effects on the immune system could be felt either directly or indirectly (i.e., through a mediating variable such as lower levels of compliance due to feelings of helplessness).

Also found to be significantly related to immune system variables was the level of Future Despair, which reflects the individual's willingness to plan for and look forward to the future. Individuals scoring higher on the despair scale are likely to perceive their medical condition in more negative terms, which could lead them to an more generally pessimistic outlook on the remainder of their lives.

Finally, there was also an effect of Somatic Anxiety, which measures the preoccupation and fears individuals have about their bodies. Higher scorers tend to be hypochondriacal, and may overreact to the discomforts of particular treatments.

At least two of the four psychogenic attitudes found to be significantly related to immune system functioning--tension and anxiety --are also part of an ongoing syndrome of stress. Researchers have recognized for more than a decade that there is a need for the body to regulate its internal mechanisms against the impact of external stressors. When such regulatory processes are disrupted by chronic stress, one possible result is a breakdown in the immune system. In fact, chronic stress contributes to neuroendocrine changes which can overcome the body's existing homeostasis.

Each of the four psychogenic attitudes found to be significantly related to CD4 levels--tension, pessimism (hopelessness), despair, and Somatic Anxiety--may be seen as being related to the fear of death. There is considerable variation in the way individuals experience fear of death, depending on religiosity and other variables. For example, some individuals may fear specific physiological aspects of dying (pain), while others may be more existentially concerned. However, to the degree that their stress-related symptoms affect the immune function, it is important that corrective action be taken.

PWAs are also likely to experience fear of abandonment, and this could contribute to the elements of stress and anxiety being measured in this study. Many common experiences faced by PWAs can serve to reinforce this fear; economic and housing discrimination and the

disintegration of their old social support networks are just two examples. Individuals may also experience anxiety and despair over the prospect of becoming dependent on others.

Finally, it should also be recognized that the results should be interpreted with caution, given the relatively small N. Related to this is the limitation imposed by the dropoff in available data over the period of the study. Other limitations include the need to assume that the associations observed in the study will also exist in other samples of HIV seropositive subjects and that responses to the self-report instruments utilized were valid. The use of the BDI as the primary instrument to measure depression may also limit the usefulness of the results, given the possible tendency to over-endorse somatic items on the scale.

Implications for Therapy

For the therapist working with the HIV-positive client demonstrating symptoms of anxiety, it is essential to develop strategies for reducing the client's anxiety. This may include teaching him to cope more effectively on a day-to-day basis, and using these practical "survival" skills as building blocks to reduce anxiety.

The therapist is faced with a difficult challenge in implementing anxiety-reducing techniques, in that some of the anxieties of the HIV-positive client are, inevitably, well-founded. That is, there is no guarantee that the condition will not progress to AIDS, and there is no way to predict when that event will--or will not--occur. This built-in uncertainty creates tremendous pressure, and naturally

produces a degree of anxiety. This suggests that the therapist must acknowledge the reality of the client's condition, and work in a reality-oriented, day-to-day mode.

Chronic stress and related psychogenic attitudes were found, in the current study, to be correlated with impaired immune system functioning. One aspect of treatment that is related to psychogenic attitudes is the maintenance of hope. Some researchers have reported on populations of HIV-positive men who have maintained high levels of hope and less depression.

More generally, these findings imply that the therapist should give priority to controlling stress levels. One appropriate technique is stress management using relaxation therapy, mental imagery, and meditation. Stress reduction programs should incorporate an eclectic blend of treatment modalities.

Clinicians must be aware of the changing psychological profile over the course of the disease. Individuals who are not yet symptomatic may be even more distressed (Chuang et al., 1989). The individual who is nearing death may need help in coping with the issues of mortality and the resolution of unfinished business (Kubler-Ross, 1987). Yet those who are not so progressed also face major adjustment challenges: fears of the unknown, social isolation, and high levels of stress and anxiety.

Recommendations for Future Research

Relatively few studies have specifically linked psychosocial variables to both immune system markers and physical health outcome

(Gorman & Kertzner, 1990). There are several reasons that studies applying the PNI perspective to the study of AIDS have been so scarce (Solomon & Temoshok, 1987). It is expensive to conduct adequate immunological tests, and results from the longitudinal studies already underway are just becoming available. There is also the major unresolved question of identifying the lag time between psychological factors and immune system functioning. How long, for example, does it take for depression to have an effect on the immune system? And is that effect felt directly, or is it mediated by such variables as decreased compliance or participating in high-risk activities?

While the results of the current study do not support the argument that depression has a direct effect on immune system functioning, this should be seen as inconclusive given the limits of the study. Further research is needed to clarify the effects of both depression and coping style on immune system functioning.

Future studies should also address the need for utilizing relevant psychosocial measures (Temoshok, 1990). Researchers should utilize published measures that have demonstrated acceptable reliability and validity characteristics, and that have been used in previous immunological-oriented studies. It is essential to be sensitive to the unique nature of the HIV disease, and the implications for using certain published instruments. For example, the use of standard stress surveys in assessing stress in a sample of HIV-positive gay males who have lost numerous friends to the disease may be inappropriate (Temoshok, 1990). One advantage of the current study is that it utilizes the MBHI, which was specifically designed for use with

physically ill patients and medical-behavioral decision-making issues in mind (Millon et al., 1982).

Other possible psychosocial variables that may predict immune system functioning need to be explored. For example, some researchers have suggested that there may be a "hardiness" construct--consisting of commitment, control, and challenge--that may mediate the effects on the immune system. This remains to be empirically evaluated, and represents a fruitful area for future research. Other areas in need of further study include (a) research on the relative effects of acute and chronic stress on immune system function, and (b) research on the temporal relationship among stress, immune effect, and overall physical stress.

However, the difficulties involved in conducting well-designed and comprehensive psychoneuroimmunologic studies are substantial. As Kielcolt-Glaser (1988) put it:

> ...an ideal study would include multiple immunological samples from the same subjects over a period of several days...multiple psychological measures over a two-week period or more...the subjects would have a uniform physical activity level, and they would, of course, be remarkably compliant...The most difficult subject population in this respect is HIV-seropositive individuals. (p. 76)

Nevertheless, it is essential that researchers rise to the challenge and design innovative research studies. Such studies should be longitudinal in nature, following the lead of the pioneering studies being conducted with subjects in the San Francisco Men's Health Study (Burack et al., 1992). Such studies should strive to clarify the precise nature of the complex interaction among psychological variables, immune system functioning, and medical outcome.

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APPENDICES

Appendix A

Institutional Consent Forms

Medical Center

Wadsworth and
Brentwood Divisions
Wilshire and
Sawtelle Boulevards
Los Angeles CA 90073

Veterans
Administration

In Reply Refer To: 691/151

RIGHTS OF HUMAN SUBJECTS IN MEDICAL EXPERIMENTS

Any person who is requested to consent to participate as a subject in a research study involving a medical experiment or who is requested to consent on behalf of another has the right to:

1. Be informed of the nature and purpose of the experiment.

2. Be given an explanation of the procedures to be followed in the medical experiment, and any drug or device to be utilized.

3. Be given a description of any attendant discomforts and risks reasonably to be expected from the experiment.

4. Be given an explanation of any benefits to the subject reasonably to be expected from the experiment, if applicable.

5. Be given a disclosure of any appropriate alternative procedures, drugs or devices that might be advantageous to the subject, and their relative risks and benefits.

6. Be informed of the avenues of medical treatment, if any, available to the subject after the experiment if complications should arise.

7. Be given an opportunity to ask any questions concerning the experiment or the procedure involved.

8. Be instructed that consent to participate in the medical experiment may be withdrawn at any time and the subject may discontinue participation in the medical experiment without prejudice.

9. Be given a copy of any signed and dated written consent form used in relation to the experiment.

10. Be given an opportunity to decide to consent or not to consent to a medical experiment without the intervention of any element of force, fraud, deceit, duress, coercion, or undue influence on the subject's decision.

VA MEDICAL CENTER WEST LOS ANGELES, WADSWORTH DIVISION
Wilshire & Sawtelle Blvds., Los Angeles, CA 90073

HUMAN STUDIES CONSENT FORM
(Addendum to VA Form 10-1086)

Title of Protocol: HIV-Related CNS Abnormalities

Study # 001 Add #3

Responsible Investigator: Wilfred G. Van Gorp, Ph.D. Phone # 478-3711

Co-Investigator(s): Calvin J. Frederick, Ph.D. 824-3174

W. W. Tourtellotte, M.D., Ph.D. 478-3711

a. BACKGROUND. I have been asked to participate in this study because I have been found to be HIV seropositive or seronegative (control). It is known that HIV and other viruses may cause diseases of the central nervous system in some patients. It is not understood how these affect the brain, why some patients show them and others do not, or how they might respond to various treatments. Approximately 150 patients and 50 HIV seronegative controls will be included in this study.

b. PURPOSE: The purpose of this study is to investigate the occurence and manifestations of HIV related brain abnormalities in various patients as they compare with results in HIV seronegative control subjects.

c. PROCEDURES: I will be given:

1) A neuropsychological examination which consists of an adult intelligence test, a test of memory, a test of ability to learn and recall words or pictures, tests that will require me to draw lines and simple figures, and a test requiring me to rapidly place pegs in slots. I will also complete brief paper and pencil tests measuring my mood. This examination takes approximately 2 hours.

2) I may be asked to have a spinal tap. This involves giving blood and having a small sample of spinal fluid removed from the lower back through a thin needle. This procedure is performed by a trained neurologist under sterile conditions. The skin is anesthetized with a small amount of local anesthetic, and the needle inserted into the spinal column much as the drawing of blood from a vein. Approximately one tablespoon of spinal fluid will be removed, and one ounce of blood (approximately 2-3 teaspoonfuls) will be removed from my arm vein (venipuncture). After the tap I will have to lie still for 20 minutes on my side. I may refuse the spinal tap but still participate in all other components of this study.

Date

Patient or Responsible Party

Patient's Social Security #

Witness

Investigator Initials

VA MEDICAL CENTER WEST LOS ANGELES, WADSWORTH DIVISION
Wilshire & Sawtelle Blvds., Los Angeles, CA 90073

HUMAN STUDIES CONSENT FORM
(Addendum to VA Form 10-1086)

Title of Protocol: HIV-Related CNS Abnormalities

Study # 001 Add2

Responsible Investigator: Wilfred G. Van Gorp, Ph.D. Phone # 478-3711

Co-Investigator(s): Calvin J. Frederick, Ph.D. 824-3174

W. W. Tourtellotte, M.D., Ph.D. 478-3711

3) A neurologic examination which includes a limited physical exam and questions, and paper and pencil tests to evaluate my mental state. This examination takes approximately 1 hour.

4) A Positron Emission Tomography Scan (PET): In this procedure, a small amount of radioactive sugar (18F-2-deoxyglucose, called FDG) will be injected into a vein in my arm. The maximum amount of radioactivity will be an amount equal to chest x-rays. I will lie in a comfortable bed while pictures are being made of the radioactivity in my brain by a special radioisotope scanner. The entire study will take approximately 2 hours. The radioactivity will disappear from my body rapidly, one-half in less than 2 hours, and all within 12 hours. This procedure will be repeated every 6 months for 3 years.

5) A Magnetic Resonance Imaging Scan (MRI): This is performed with a powerful magnet. This technique provides a detailed picture or map of the brain, without using x-ray radiation or radioactive material. A specially designed computer converts information from the magnetic resonance scan into a visible image. No preparation is needed and the scan can be performed in about an hour. This will be given to me only once at most.

d. RISKS AND/OR INCONVENIENCES: The neuropsychological testing will take approximately 2 hours and may be somewhat tiring. The risks from the blood drawing are: (1) pain from insertion of the needle; (2) light headedness; (3) fainting; (4) hematoma (like a bruise) at the site of needle insertion.

Date: Patient or Responsible Party

Patient's Social Security # Witness

Investigator, Physician

VA MEDICAL CENTER WEST LOS ANGELES, WADSWORTH DIVISION
Wilshire & Sawtelle Blvds., Los Angeles, CA 90073

HUMAN STUDIES CONSENT FORM
(Addendum to VA Form 10-1086)

Title of Protocol: HIV-Related CNS Abnormalities

Study # 001 Add 3

Responsible Investigator: Wilfred G. Van Gorp, Ph.D. Phone # 478-3711

Co-Investigator(s): Calvin J. Frederick, Ph.D. 824-3174

W. W. Tourtellotte, M.D., Ph.D. 478-3711

e. BENEFITS: There may be no immediate or direct benefit to me from participating in this phase of the study. However, if symptoms suggesting early neurologic disorders are detected, this might allow for earlier treatment. Also, knowledge gathered from this study may help us understand more about how and when HIV can affect the nervous system. This may have implications for the treatment of HIV-related nervous system disorders in the figure. Further, I will be informed as to any medical or laboratory findings that are important to my health care and my doctor will also be notified. I also understand that in spite of my participation in this study, my condition may become worse.

f. ALTERNATIVES: I may decline participation in this study.

g. Any questions I have concerning my participation in this study will be answered by: Wifred G. Van Gorp, Ph.D..

Date _____ Patient or Responsible Party _____

Soc. Security # _____ Witness _____

Investigator/Physician _____

VA MEDICAL CENTER WEST LOS ANGELES, WADSWORTH DIVISION
Wilshire & Sawtelle Blvds., Los Angeles, CA 90073

HUMAN STUDIES CONSENT FORM
(Addendum to VA Form 10-1086)

Title of Protocol: HIV-Related CNS Abnormalities

Study # 001 Add

Responsible Investigator: Wilfred G. Van Gorp, Ph.D. Phone # 478-3711

Co-Investigator(s): Calvin J. Frederick, Ph.D. 824-3174

W. W. Tourtellotte, M.D., Ph.D. 478-3711

h. The results of this study may be published, but my name or identity will not be revealed.

i. I will not be prevented from receiving established forms of therapy while on this study. If more than trivial side effects directly attributable to the study occur, the study will be discontinued and routine treatment continued.

j. **NON-VETERANS:** In the event physical injury is sustained as a result of participating in this program, non-veterans may receive emergency medical care and, in appropriate cases, may be entitled to compensation under the provisions of the Federal Tort Claims Act (28 U.S.C. 1346(b), 2671-2680).

k. **VETERANS:** Continuing medical care will be provided eligible veterans in the event physical injury is sustained as a result of participation in this program. Aditionally, compensation may be payable to eligible veterans and 38 U.S.C. 351, or in appropriate cases, under the provisions of Federal Tort Claims Act (28 U.S.C. 1346(B), 2671-2680.

For clarification of these laws, contact the VA District Counsel (213) 209-7379.

l. If I have complaints about the procedure or study I may express them to Drs. Van Gorp, Frederick, or Tourtellotte (investigators) or the Committee on Human Studies, or to the Associate Chief of Staff for Research and Development, Wadsworth Division, VA Medical Center West Los Angeles, (213) 478-3711, extension 4224, or the Associate Chief of Staff, Research and Development, Brentwood Division, VAMC West Los Angeles, (213) 824-3180.

m. I have received a copy of this consent form and my file copy of the the "Rights of Human Subjects in Medical Experiments."

Date

Patient or Responsible Party

Soc. Security #

Witness

Investigator/Physician

VA MEDICAL CENTER WEST LOS ANGELES, WADSWORTH DIVISION
Wilshire & Sawtelle Blvds., Los Angeles, CA 90073

HUMAN STUDIES CONSENT FORM
(Addendum to VA Form 10-1086)

Title of Protocol: HIV-Related CNS Abnormalities

Study # 001 Add #

Responsible Investigator: Wilfred G. Van Gorp, Ph.D. Phone # 478-3711

Co-Investigator(s): Calvin J. Frederick, Ph.D. 824-3174

W. W. Tourtellotte, M.D., Ph.D. 478-3711

The risks from the spinal tap are: (1) an allergic reaction to the iodine used to cleanse the skin manifested by itching, a skin rash, or blisters; Rarely the rash or blisters may occur throughout the body surface; (2) an allergic reaction to the local anesthetic manifested by difficulty breathing, sweating, coldness, or light headedness. If severe, the reaction can cause a drop in blood pressure which can very rarely cause death; (3) Pain in the lower back or tingling down one leg if the needle touches a nerve; (5) Headache for hours or days after the spinal tap. This occurs about 10% of the time and is rarely severe. Rarely infection at the needle site or meningitis.

If I am given a PET scan, FDG is not metabolized normally by the body, but in this study only minute doses will be administered, less than one-ten-thousandth of the amount required to show pharmacologic or behavioral effect. It will be excreted in my urine after administration within 12 hours. The whole body radiation dose for each study is less than 1/15 of the annual permitted dose to radiation workers, and is less than that received should I have an x-ray examination of my spine or gastro-intestinal tract. The amount I will receive is well within those limits that have been accepted by the Nuclear Regulatory Commission for radiation workers. These are limits which have been developed and carefully reviewed by nationally and internationally recognized groups of scientists.

There are no known risks associated with the MRI scan, except possibly for fatigue or boredom.

Date _____ Patient or Responsible Party _____

Soc. Security # _____ Witness _____

Investigator Physician _____

___ LOW RISK

X MEDIUM TO HIGH RISK

STUDY No. 0001 Add #3

PART I-AGREEMENT TO PARTICIPATE IN RESEARCH BY OR UNDER THE DIRECTION OF THE VETERANS ADMINISTRATION

DATE

I, _____, voluntarily consent to participate as a subject
(Type or print subject's name)

in the investigation entitled HIV Related CNS Abnormalities
(Title of study)

2. I have signed one or more information sheets with this title to show that I have read the description including the purpose and nature of the investigation, the procedures to be used, the risks, inconveniences, side effects and benefits to be expected, as well as other courses of action open to me and my right to withdraw from the investigation at any time. Each of these areas has been explained to me by the investigator in the presence of a witness. The investigator has answered my questions concerning the investigation and I believe I understand what is intended.

3. I understand that no guarantees or assurances have been given me since the results and risks of an investigation are not always known beforehand. I have been told that this investigation has been carefully planned, that the plan has been reviewed by knowledgeable people, and that every reasonable precaution will be taken to protect my well-being.

4. In the event I sustain physical injury as a result of participation in this investigation, if I am eligible for medical care as a veteran, all necessary and appropriate care will be provided. If I am not eligible for medical care as a veteran, humanitarian emergency care will nevertheless be provided.

5. I realize I have not released this institution from liability for negligence. Compensation may or may not be payable, in the event of physical injury arising from such research, under applicable federal laws.

6. I understand that all information obtained about me during the course of this study will be made available only to doctors who are taking care of me and to qualified investigators and their assistants where their access to this information is appropriate and authorized. They will be bound by the same requirements to maintain my privacy and anonymity as apply to all medical personnel within the Veterans Administration.

7. I further understand that, where required by law, the appropriate federal officer or agency will have free access to information obtained in this study should it become necessary. Generally, I may expect the same respect for my privacy and anonymity from these agencies as is afforded by the Veterans Administration and its employees. The provisions of the Privacy Act apply to all agencies.

8. In the event that research in which I participate involves certain new drugs, information concerning any response to the drug(s) will be supplied to the sponsoring pharmaceutical house(s) that made the drug(s) available. This information will be given to them in such a way that I cannot be identified.

I _____
NAME OF VOLUNTEER

HAVE READ THIS CONSENT FORM. ALL MY QUESTIONS HAVE BEEN ANSWERED, AND I FREELY AND VOLUNTARILY CHOOSE TO PARTICIPATE. I UNDERSTAND THAT MY RIGHTS AND PRIVACY WILL BE MAINTAINED. I AGREE TO PARTICIPATE AS A VOLUNTEER IN THIS PROGRAM.

9. Nevertheless, I wish to limit my participation in the investigation as follows:

PLEASE SEE ATTACHED ADDENDA TO THIS VA FORM 10-1086

5 (No. of addendums attached)

VA FACILITY	SUBJECT'S SIGNATURE
691 VAMC West Los Angeles, Wadsworth Div.	
WITNESS'S NAME AND ADDRESS (Type or print)	WITNESS'S SIGNATURE
INVESTIGATOR'S NAME (Type or print)	INVESTIGATOR'S SIGNATURE

☐ Expand abbreviations shown underlined ☐ Expand abbreviations shown overstrike or

SUBJECT'S IDENTIFICATION (I.D. plate or give name + last four digits)

SUBJECT'S I.D. NO. | GROUP

AGREEMENT TO PARTICIPATE IN RESEARCH BY OR UNDER THE DIRECTION OF THE VETERANS ADMINISTRATION

VA FORM OCT 1980 10-1086 PREVIOUS EDITIONS OF FORM 10-1086 ARE OBSOLETE AND MAY NOT BE USED.

PART II - AGREEMENT BY SUBJECT'S REPRESENTATIVE TO ALLOW SUBJECT TO PARTICIPATE IN RESEARCH BY OR UNDER THE DIRECTION OF VETERANS ADMINISTRATION

DATE

1. I, ______________________________ am authorized to give consent
(Type or print name of subject's representative)

for ______________________________ by virtue of ______________________________
(Type or print subject's name) (Relationship, legal appointment, etc.)

I voluntarily consent for this person to participate as a subject in the investigation entitled ______________________________
(Title of study)

2. I have signed one or more information sheets with this title to show that I have read the description including the purpose and nature of the investigation, the procedures to be used, the risks, inconveniences, side effects, and benefits to be expected, as well as other courses of action open to me and my right to withdraw the subject from the investigation at any time. Each of these items has been explained to me by the investigator in the presence of a witness. The investigator has answered my questions concerning the investigation and I believe that I understand what is intended.

3. I understand that no guarantees or assurances have been given me since the results and risks of an investigation are not always known beforehand. I have been told this investigation has been carefully planned, that the plan has been reviewed by knowledgeable people, and that every reasonable precaution will be taken to protect the well-being of the subject.

4. In the event the subject sustains physical injury as a result of participation in this investigation, if the subject is eligible for medical care as a veteran, all necessary and appropriate care will be provided. If the subject is not eligible for medical care as a veteran, humanitarian emergency care will nevertheless be provided.

5. I realize I have not released this institution from liability for negligence. Compensation may or may not be payable, in the event of physical injury arising from such research, under applicable federal laws.

6. I understand that all information obtained about the subject during the course of this study will be made available only to doctors who are taking care of the subject and to qualified investigators and their assistants where their access to this information is appropriate and authorized. They will be bound by the same requirements to maintain the subject's privacy and anonymity as apply to all medical personnel within the Veterans Administration.

7. I further understand that, where required by law, the appropriate federal officer or agency will have free access to information obtained in this study should it become necessary. Generally, I may expect the same respect for the subject's privacy and anonymity from these agencies as is afforded by the Veterans Administration and its employees. The provisions of the Privacy Act apply to all agencies.

8. In the event that research in which the subject participates involves certain new drugs, information concerning the subject's response to the drug(s) will be supplied to the sponsoring pharmaceutical house(s) that made the drug(s) available. This information will be given to them in such a way that the subject cannot be identified.

I ______________________________
NAME OF SUBJECT'S REPRESENTATIVE

HAVE READ THIS CONSENT FORM. ALL MY QUESTIONS HAVE BEEN ANSWERED, AND I FREELY AND VOLUNTARILY CHOOSE THAT THE SUBJECT PARTICIPATE. I UNDERSTAND THAT THE SUBJECT'S RIGHTS AND PRIVACY WILL BE MAINTAINED. I AGREE TO THE SUBJECT'S PARTICIPATION AS A VOLUNTEER IN THIS PROGRAM.

9. Nevertheless, my consent for the subject's participation in the investigation is limited as follows:

ADDRESS OF SUBJECT'S REPRESENTATIVE (Print or type)	SIGNATURE OF SUBJECT'S REPRESENTATIVE
WITNESS'S NAME AND ADDRESS (Print or type)	WITNESS'S SIGNATURE
SUBJECT'S NAME (Print or type)	SUBJECT IS NOW A PATIENT AT (Name of VA Facility)
INVESTIGATOR'S NAME (Print or type)	INVESTIGATOR'S SIGNATURE

☐ Signed information sheets attached. ☐ Signed information sheets available at:

SUBJECT'S IDENTIFICATION (I.D. plate or print name - last, first, middle)

SUBJECT'S I.D. NO.	AGE	WARD

AGREEMENT BY SUBJECT'S REPRESENTATIVE TO PARTICIPATE IN RESEARCH BY OR UNDER THE DIRECTION OF THE VETERANS ADMINISTRATION

Appendix B

Millon Behavioral Health Inventory

Name: _____
Date: _____

MILLON BEHAVIORAL HEALTH INVENTORY

INSTRUCTIONS: The following are statements that people use to describe themselves, their feelings and attitudes about health. Try to be as honest as you can in marking the statements. A few of the statements may seem unusual to you, but do not be concerned; they are included to describe a wide range of people with many types of problems. When you agree or mostly agree with a statement or decide that it describes you, circle True. If you disagree with a statement or decide that it does not describe you, circle False. Try to mark every statement even if you are not sure of your choice. If you have tried your best and still cannot decide, mark False.

T F 1. I have always been able to overcome the problems I've had.

T F 2. Lately, life has been going along as usual, with no special things happening.

T F 3. When I was a young child, my parents felt very proud of me.

T F 4. I have almost never been sick.

T F 5. I have friends who will listen to any problems I have.

T F 6. I like to be the one in authority to take charge of things.

T F 7. If I were very sick, I'm sure that everything would work out well.

T F 8. I always take the medicine a doctor tells me to even if I don't think it is working.

T F 9. I am very pleased with all the things I have done up to now.

T F 10. I almost never feel pressure in the work I do.

T F 11. I get very frightened when I think of being all alone in the world.

T F 12. I am ready to attack anyone who tries to say terrible things about me.

T F 13. I have a feeling that things in my life just go from bad to worse.

T F 14. All my life I have had to "blow up" every now and then.

T F 15. This year I was successful at something that was very important to me.

T F 16. I am in better health than most of my friends.

T F 17. A quiet hobby is more fun for me than a party.

T F 18. Most people wouldn't care much if I were very sick.

T F 19. I often say things that I regret having said.

T F 20. I have lots of plans of what I'd like to be doing ten years from now.

T F 21. I have a lot of faith that doctors can cure any sickness.

T F 22. People can influence me quite easily.

T F 23. I often find time to take it easy and do nothing.

T F 24. Even in difficult times, I always try to be cheerful.

T F 25. I don't mind that other people are not interested in my friendship.

T F 26. I've had serious money problems this year.

T F 27. I almost always have medical problems.

T F 28. I often feel that others do not want to be friendly to me.

T F 29. If I became ill, I wouldn't have much help from my family.

T F 30. In many ways I feel very superior to most people.

T F 31. If I ever got a serious illness, I think it would be the end of me.

T F 32. No matter what, seeing a doctor can make me feel better.

T F 33. So little of what I have done has been appreciated by others.

T F 34. Keeping to a time schedule is not important to me.

T F 35. I've done most things in my life very well.

T F 36. When I think about the past, I remember mostly the good things.

T F 37. I make nasty remarks to people if they deserve it.

T F 38. I have had more than my share of troubles in the past.

T F 39. It is good to have a regular way of doing things to avoid mistakes.

T F 40. Many people have been spying into my private life for years.

T F 41. I almost never worry about my health.

T F 42. If I thought I had a serious sickness, I would quickly talk it over with my family.

T F 43. There are always a number of reasons why most problems can't be solved.

T F 44. I look forward to the future with lots of hope.

T F 45. I do my best to get along with others by being pleasant and agreeable.

T F 46. All doctors care about is my money.

T F 47. I get upset when things I don't expect happen to me.

T F 48. I often get angry with people who do things slowly.

T F 49. I don't depend much on other people for friendship.

T F 50. I feel pretty upset about most things in my life.

T F 51. It is very difficult for me to stop feelings from coming out.

T F 52. My family has had really bad problems in the past year.

T F 53. I can stand a lot of pain.

T F 54. I like to flirt a lot.

T F 55. In time of trouble, there are several friends that I can depend on.

T F 56. Most people can be trusted to be kind and thoughtful.

T F 57. Even if I were very sick, I'd keep fighting and never give up

T F 58. I sometimes feel I am in this world all alone.

T F 59. I feel that the doctors I have seen are not interested in my problems.

T F 60. I am a dramatic and showy sort of person.

T F 61. I can't stand people who are late for appointments.

T F 62. I do my best to stop anyone from trying to boss me.

T F 63. I often think about unhappy things that have happened to me.

T F 64. I often do things for no reason other than it might be fun.

T F 65. During the past year, someone close to me has been very ill.

T F 66. I guess I'm a complainer who expects the worst to happen.

T F 67. It is not unusual to feel lonely and unwanted.

T F 68. I worry a lot about my health.

T F 69. Lots of people would care about me if I became very sick.

T F 70. I would much rather follow someone than be the leader.

T F 71. If I had a very serious sickness, I think I would fall apart mentally.

T F 72. To get ahead in this world I'm willing to push people who get in my way.

T F 73. Doctors have always been helpful to me.

T F 74. I find it hard to feel sorry for people who are always worried about things.

T F 75. I seem to fit in right away with any groups of people I meet.

T F 76. I like being in a crowd just to be with lots of people.

T F 77. Most of my problems just go on and on.

T F 78. I guess I depend too much on others to be helpful to me.

T F 79. I moved during the past year.

T F 80. I have always felt some kind of problem between me and the opposite sex.

T F 81. I get frightened when I think I have a medical problem.

T F 82. Punishment never stopped me from doing whatever I wanted.

T F 83. I would have lots of visitors if I were in the hospital.

T F 84. Among the most important things a person can have are a strong will and the drive to get ahead.

T F 85. I would never let a serious sickness stop me from working toward the future.

T F 86. I often feel so angry that I want to throw and break things.

T F 87. I dislike going to doctors; and do so only after trying everything myself.

T F 88. I really hate to have my work pile up.

T F 89. I find it hard to take my mind off my work even when I'm supposed to be relaxing.

T F 90. I have not seen a car in the last ten years.

T F 91. I very often think I am not wanted by others in a group.

T F 92. Even when things seem to be going well, I expect that they'll soon get worse.

T F 93. I would rather be direct with people than avoid telling them something they don't like.

T F 94. Many important things have happened in my life this past year.

T F 95. What this country really needs are more serious and devoted citizens.

T F 96. At no time in my life have I had any hair on my head or my body.

T F 97. TV programs about illness make me very upset.

T F 98. Ever since I was a child I have been losing touch with the real world.

T F 99. I cannot depend on my family when I need them.

T F 100. I like to tell others about the things I have done well.

T F 101. I'd rather be dead than have a very serious sickness.

T F 102. I usually let other people have their own way.

T F 103. I usually won't take any medicines, even if a doctor tells me to.

T F 104. I wish the people around me would move faster and get more things done.

T F 105. I often feel that there is nothing I can do to make my life easier.

T F 106. I have very few close personal ties with others.

T F 107. This past year has been one of the most difficult ones in my life.

T F 108. If I thought I were getting sick, I would quickly call a doctor.

T F 109. I have a strong desire to win any game I play with others.

T F 110. Nobody really cares about my state of health.

T F 111. I have faith that human nature is good.

T F 112. I haven't thought much about what I'll be doing a year from now.

T F 113. All my life I have had the feeling that I have done something terribly wrong or evil.

T F 114. When someone hurts me, I try to forget it.

T F 115. Hospitals are frightening and lonely places to be in.

T F 116. My work makes me tense almost all the time.

T F 117. I have flown across the Atlantic 30 times last year.

T F 118. In this world you either push or get shoved.

T F 119. If I were young again, I would do things very differently.

T F 120. It is very important that children learn to obey their elders.

T F 121. I've had a lot of shocks and disappointments this past year.

T F 122. Rather than demand things, people can get what they want by being gentle and thoughtful.

T F 123. I get very upset when I feel pain in any part of my body.

T F 124. I can see more sides of a problem better than others can.

T F 125. If I were getting sick, I wouldn't waste my time telling anyone in my family.

T F 126. I am more worried about finishing things that I start than most people.

T F 127. For me, the future looks like it will be full of trouble and problems.

T F 128. I do my best not to hurt people's feelings.

T F 129. I have never felt much life in me.

T F 130. I would rather be in pain than take any medicines.

T F 131. I often doubt whether people are really interested in what I am saying to them.

T F 132. It is very easy for me to relax and slow down.

T F 133. I don't know what I want out of life.

T F 134. Life has never gone well for me.

T F 135. I've been touchy or tearful about everything most of my life.

T F 136. I am very uneasy when I have to tell people what to do.

T F 137. I am too rushed and busy to take the vacations I should.

T F 138. There has recently been an important change in my job.

T F 139. I like to follow instructions and do what others expect of me.

T F 140. I often think that I have a serious illness.

T F 141. I am a quiet and cooperative person.

T F 142. I'd be a pretty lonely person if I ever were hospitalized.

T F 143. I become very excited or upset once a week or more.

T F 144. I always try to do what is proper.

T F 145. I don't think I would want to go on living if my body was marked up a lot in a serious operation.

T F 146. I get so touchy that I can't talk about certain things.

T F 147. From things I hear about them, I don't trust the people who work in hospitals.

T F 148. I have a strong need to feel like an important person.

T F 149. My day is filled with pressures and responsibilities.

T F 150. I like to arrange things down to the last detail.

Appendix C

Beck Depression Inventory

BECK INVENTORY
(rev 10/73)

Name: _____

Date: _____

INSTRUCTIONS: On this questionnaire are groups of statements. Please read each group of statements carefully. Then pick out the one statement in each group that best describes the way you have been feeling the PAST WEEK, INCLUDING TODAY. Circle the number beside the statement you picked. If several statements in the group seem to apply equally well, circle each one. Be sure to read all the statements in each group before making your choice.

1. ( ) 0 I do not feel sad.
   1 I feel sad.
   2 I am sad all the time and I can't snap out of it.
   3 I am so sad or unhappy that I can't stand it.

2. ( ) 0 I am not particularly discouraged about the future.
   1 I feel discouraged about the future.
   2 I feel I have nothing to look forward to.
   3 I feel that the future is hopeless and that things cannot improve.

3. ( ) 0 I do not feel like a failure.
   1 I feel I have failed more than the average person.
   2 As I look back on my life, all I can see is a lot of failures.
   3 I feel I am a complete failure as a person.

4. ( ) 0 I get as much satisfaction out of things as I used to.
   1 I don't enjoy things the way I used to.
   2 I don't get real satisfaction out of anything anymore.
   3 I am dissatisfied or bored with everything.

5. ( ) 0 I don't feel particularly guilty.
   1 I feel guilty a good part of the time.
   2 I feel quite guilty most of the time.
   3 I feel guilty all the time.

6. ( ) 0 I don't feel I am being punished.
   1 I feel I may be punished.
   2 I expect to be punished.
   3 I feel I am being punished.

7. ( ) 0 I don't feel disappointed in myself.
   1 I am disappointed in myself.
   2 I am disgusted with myself.
   3 I hate myself.

8. ( ) 0 I don't feel I am any worse than anybody else.
   1 I am critical of myself for my weaknesses or mistakes.
   2 I blame myself all the time for my faults.
   3 I blame myself for everything bad that happens.

9. ( ) 0 I don't have any thoughts of killing myself.
   1 I have thoughts of killing myself, but I would not carry them out.
   2 I would like to kill myself.
   3 I would kill myself if I had the chance.

10. ( ) 0 I don't cry any more than usual.
    1 I cry more than I used to.
    2 I cry all the time now.
    3 I used to be able to cry, but now I can't even though I want to.

11. ( ) 0 I am no more irritated now than I ever am.
1 I get annoyed or irritated more easily than I used to.
2 I feel irritated all the time now.
3 I don't get irritated at all by the things that used to irritate me.

12. ( ) 0 I have not lost interest in other people.
1 I am less interested in other people than I used to be.
2 I have lost most of my interest in other people.
3 I have lost all of my interest in other people.

13. ( ) 0 I make decisions about as well as I ever could.
1 I put off making decisions more than I used to.
2 I have greater difficulty in making decisions than before.
3 I can't make decisions at all anymore.

14. ( ) 0 I don't feel I look any worse than I used to.
1 I am worried that I am looking old or unattractive.
2 I feel that there are permanent changes in my appearance that make me look unattractive.
3 I believe that I look ugly.

15. ( ) 0 I can work about as well as before.
1 It takes an extra effort to get started at doing something.
2 I have to push myself very hard to do anything.
3 I can't do any work at all.

16. ( ) 0 I can sleep as well as usual.
1 I don't sleep as well as I used to.
2 I wake up 1-2 hours earlier than usual and find it hard to get back to sleep.
3 I wake up several hours earlier than I used to and cannot get back to sleep.

17. ( ) 0 I don't get more tired than usual.
1 I get tired more easily than I used to.
2 I get tired from doing almost anything.
3 I am too tired to do anything.

18. ( ) 0 My appetite is no worse than usual.
1 My appetite is not as good as it used to be.
2 My appetite is much worse now.
3 I have no appetite at all anymore.

19. ( ) 0 I haven't lost much weight, if any, lately.
1 I have lost more than 5 pounds.
2 I have lost more than 10 pounds.
3 I have lost more than 15 pounds.

I am purposely trying to lose weight by eating less: YES NO

20. ( ) 0 I am no more worried about my health than usual.
1 I am worried about my physical problems such as aches and pains; or upset stomach; or constipation.
2 I am very worried about my physical problems and it's hard to think of much else.
3 I am so worried about my physical problems, that I cannot think about anything else.

21. ( ) 0 I have not noticed any recent change in my interest in sex.
1 I am less interested in sex than I used to be.
2 I am much less interested in sex now.
3 I have lost interest in sex completely.

VITA

GARY K. RICHEY

DISSERTATION

The Impact of Psychosocial Variables on Immune System Functioning in a Sample of HIV-Positive Males

EDUCATION

Ph.D. (Psychology) Utah State University, Logan, Utah (1992)

M.A. (Psychology) Loyola College, Baltimore, Maryland (1982)

B.S. (Psychology) Virginia Commonwealth University, Richmond, Virginia (1975)

PROFESSIONAL EXPERIENCE

1992	Chemical Dependency Counselor, St. John's Hospital, Santa Monica, California.
1991-1992	Behavioral Specialist, Kedren Community Mental Health Center, Los Angeles, California.
1990-1991	Psychology Intern, West Los Angeles Veteran's Affairs Medical Center, Brentwood Division, Los Angeles, California.
1988-1990	Psychometrician, The Open Quest Institute, Pasadena, California.
1987-1988	Therapist, Cedar House, Long Beach, California.
1986-1987	Therapist, Penny Lane, Sepulveda, California.
1985-1986	Teaching Assistant, Department of Psychology, Utah State University, Logan Utah.
1984	Children Services Worker, Los Angeles County Department of Public Social Services (DPSS), Long Beach, California.
1982-1983	Psychiatric Assistant, Del Amo Hospital, Torrance, California.

VITA (continued)

1980-1982 Substance Abuse Counselor, Human Resources, Inc., Richmond, Virginia.

1978-1980 Vocational Evaluator, Sinai Hospital, Baltimore, Maryland.

1976-1977 Rehabilitation Specialist, Human Resources Industry, Inc., Columbia, Maryland.

1975-1976 Behavioral Specialist, Baltimore City Public Schools, Baltimore, Maryland.

PROFESSIONAL AFFILIATIONS

American Psychological Association, California Psychological Association, Los Angeles County Psychological Association.

PERSONAL DATA

Parents: Charles Abner and Violet Nichols Richey. Married: April 14, 1973 to Susan Wood Montgomery. Children: Keegan Charles and Grayson Nash Richey.