PREDICTORS OF HIV-RELATED NEUROCOGNITIVE IMPAIRMENT IN AN HIV/AIDS POPULATION

Tara C. Steinberg, B.A., M.A..

Dissertation Prepared for the Degree of

DOCTOR OF PHILOSOPHY

UNIVERSITY OF NORTH TEXAS

August 2012

APPROVED:

Mark Vosvick, Major Professor
Timothy Lane, Committee Member
Charles Guarnaccia, Committee Member
Vicki Campbell, Chair of the Department of Psychology
Mark Wardell, Dean of the Toulouse Graduate School

Steinberg, Tara, C. Predictors of HIV-related neurocognitive impairment in an HIV/AIDS population. Doctor of Philosophy (Health Psychology and Behavioral Medicine), August 2012, 119 pp., 5 tables, references, 350 titles.

Although, in the United States HIV infectivity has increased, survival rates have also improved due to highly active antiretroviral therapies (HAART). Adherence to HAART successfully prevents the progression of AIDS and AIDS-related morbidity for many living with HIV. Unfortunately, HAART's permeability into the central nervous system (CNS) is limited; thus, the prevalence of HIV-associated neurocognitive disorders (HAND) still persists. The health belief model (HBM) is the theory often used to explain and predict behavior in relation to chronic illness. This model incorporates perceptions of susceptibility, vulnerability, and severity towards a particular illness, and beliefs regarding perceived efficacy and benefits of treatment. This study expands the existing model. Many who live with HIV have a long history of negative experiences, such as stigmatization, traumatic events, and discrimination. I examined supplementary psychosocial and physiological predictor variables, such as stigma, trauma, ethnicity, general medical conditions, HIV-opportunistic infections, and falls; all relevant to disease progression in HIV. Previous researchers found links between stigma and immune function, trauma and memory, ethnicity and neuropsychological impairment, and symptom load and CNS-related alterations. Therefore, this study examined how these different psychosocial predictor variables are associated with HIV-related neurocognitive impairment. My model explained 38.6% of the variance in the outcome variable, and I

found that trauma (*B* = -.15, *OR* = .87; CI 95% = .75, 1.0, *p* = .05), ethnicity (*B* = 2.2, *OR* = 9.0, CI 95% = 1.68, 48.48, *p* =.01), general medical conditions (*B* = .30, *OR* = 1.34; CI 95% = 1.0, 1.81, *p* = .05), and falls (*B* = 2.0, *OR* = 7.2; CI 95% = 1.1, 47.0, *p* = .04), were all significant predictors of HIV-related neurocognitive impairment. However, contrary to my hypothesis, HIV-related opportunistic infections and HIV-related stigma were not significant predictors of HIV-related neurocognitive impairment. I hope that my results will contribute to revisions of older health models as well as suggest avenues for primary and secondary prevention and intervention to address those living with HIV/AIDS.

ACKNOWLEDGEMENTS

I would like to offer a special thank you to Dr. Mark Vosvick for all of your support and guidance throughout the dissertation process, and for allowing me to pursue my research interests. I am appreciative of all of the effort and time you spent on completing this project. Your mentorship has provided me with the skill-set necessary to progress in this field of research.

Committee members, thank you for providing me with constructive feedback and recommendations. Your insightful comments evoked new research topics that I plan to pursue in the future.

Thank you to the HIV/AIDS community of the Dallas Metroplex. I appreciate your willingness to participate in this study for multiple appointments.

Members of the Center for Psychosocial Health Research (CPHR), thank you for all of the time and dedication spent on recruitment, survey and test administration, and data entry. Your contributions to Project Heart were invaluable, as you facilitated the process and helped me to complete data collection for my dissertation.

Lastly, thank you to my loving family. Your encouraging words keep me motivated and your support provides me with the confidence and passion necessary to make a difference in the world.

TABLE OF CONTENTS

Page

ACKNOWLEDGEMENTS III

LIST OF TABLES VI

CHAPTER 1 INTRODUCTION 1

Epidemiology and Demographic Information 1

Health Belief Model 2

HIV-Associated Neurocognitive Disorders 5

History 5

Diagnosis 6

Neuropathology of HIV 8

Course of Cognitive Impairment in HIV 9

Evidence of Cognitive Impairment in HIV 10

Negative Affect 15

Rationale for Proposed Study 29

CHAPTER 2 METHOD 31

Participants 31

Procedures 32

Measures 34

Health History Questionnaire (HHQ; Vosvick & Colleagues, 2009) 34

International HIV-Dementia Scale (IHDS; Sacktor, 2005) 34

HIV Stigma Scale (HSS; Berger, Ferrans & Lashley, 2001) 35

Trauma History Questionnaire (THQ; Green, 1996) 36

Data Analyses 37

CHAPTER 3 RESULTS 40

Missing Data 40

Univariate and Bivariate Analyses 40

Multivariate Analyses 41
Exploratory Analyses 42

CHAPTER 4 DISCUSSION 44
Stigma 44
Trauma 46
Ethnicity 51
Symptom Load 52
General Medical Conditions 52
HIV-Related Opportunistic Infections 54
Falls 55
Education 55
Gender 56
Exploratory Analyses 58

CHAPTER 5 LIMITATIONS 59

CHAPTER 6 CLINICAL IMPLICATIONS 61

CHAPTER 7 FUTURE RESEARCH 66

APPENDIX A INFORMED CONSENT 72

APPENDIX B RECRUITMENT FLYERS 76

APPENDIX C ELECTRONIC RECRUITMENT 80

APPENDIX D EMPLOYMENT/INCOME 82

APPENDIX E MENTAL HEALTH CONTACTS 84

APPENDIX F INSTITUTIONAL REVIEW BOARD (IRB) APPROVAL 87

REFERENCES 89

LIST OF TABLES

Page

Table 1 Demographic Summary (n = 70) 69

Table 2 Mean, Standard Deviation, Range and Reliability Statistics (n = 70) 70

Table 3 Crosstabulation of Age and CVD Risk Factors (n = 70) 70

Table 4 Correlation Matrix 71

Table 5 Multivariate Statistics for Logistic Regression (HIV-related neurocognitive impairment; Nagelkerke R^2 = .368, p < .01) 71

CHAPTER 1

INTRODUCTION

Epidemiology and Demographic Information

According to the Centers for Disease Control and Prevention (CDC, 2008), more than one million individuals in the United States are infected with the human immunodeficiency virus (HIV) and approximately 470,902 live with acquired immunodeficiency syndrome (AIDS; CDC, 2009). An estimated 56,000 new HIV infections occur each year in the US (CDC, 2009); however, approximately 21% of those infected are unaware of their HIV status. Furthermore, HIV diagnoses are distributed amongst various ethnic groups: 45% African-American, 36% European-American, 17% Latino(a), and 2% other (US; CDC, 2008). Recently, the rate of infection decreased for certain socio-demographic groups, such as substance users and homosexual men (CDC, 2008). Conversely, for other groups, rates of infection increased, with heterosexual females at highest risk (CDC, 2008). Presently, new reports suggest a 12% increase of new cases of HIV among African-American females and 5% increase among Latinas in the United States (CDC, 2009).

Although rates of HIV infection increased in the US (CDC, 2008), survival rates also improved dramatically due to highly active antiretroviral therapies (HAART; Kaul, 2009). Sometimes known as a drug cocktail, HAART is used to reduce viral load, decrease opportunistic infections, increase CD4-cell count, and improve overall quality of life (Powderly, 2002). Despite the remarkable improvements made in immune function

and health outcomes linked to HAART, HIV-associated neurocognitive disorders (HAND) continue to remain a concern (Woods et al., 2009). Many of the neurological manifestations once seen in HIV have decreased; nevertheless, neuropsychological impairments are still prevalent (Robinson-Papp et al., 2008). Although, neurocognitive symptoms vary across those infected with HIV, 30% of those with asymptomatic HIV and 50% of those with AIDS display signs and symptoms of mild neurologic disease (Heaton et al., 1995; Woods et al., 2009). The most severe form of HAND, HIV-associated dementia (HAD), is mostly eliminated due to HAART; however, the incidence and prevalence of milder forms of HAND are still widespread (McArthur, 2004; Sacktor et al., 2001). Thus, this study focuses on psychosocial predictors of HIV-related neurocognitive impairment that exist, but that are more mild and do not meet diagnostic criteria for HAD.

Health Belief Model

The health belief model (HBM) is the theory commonly used to explain and predict behavior in relation to disease and chronic illness (Glanz, Rimer & Lewis, 2002). The HBM is a socio-cognitive model (Rosenstock, 1974) that postulates that health behavior (s) can be understood based on health values/concerns, beliefs about vulnerability to a particular disease/disorder, and beliefs about the consequences of that disease/disorder (Mcdermott, 1998). Furthermore, HBM is useful in understanding how perceptions of susceptibility, vulnerability, and severity towards a particular illness, and beliefs on perceived efficacy and benefits of treatment interact (Stiles & Kaplan, 2004).

Specifically, the HBM predicts behavior (s) associated with illness based on one's perception of threat (Rosenstock, Strecher & Becker, 1994). This model proposes that in order for an individual to be proactive and avoid negative health-related consequences, he/she must contemplate the perceived susceptibility (i.e. perceived risk of a health condition) and perceived severity (perceived seriousness of contracting a medical illness or leaving it untreated; Rosenstock, Strecher & Becker, 1994). The HBM also attempts to uncover barriers and benefits in regards to taking health actions that may be physical, psychological and/or financial in nature (Rosenstock, Strecher & Becker, 1994).

The HBM attempts to identify motivations that prompt particular health-related behaviors. Bandura's concept of self-efficacy, the belief in one's ability to successfully execute behaviors necessary for a particular outcome, extends the HBM (Rosenstock, Strecher & Becker, 1994; Bandura, 1977). Moreover, Rosenstock, Strecher and Becker (1988), proposed that self-efficacy is associated with the expectation of a person's ability to influence outcome. Self-efficacy can predict whether an individual initiates or eliminates maladaptive and risky health behaviors (National Institute of Mental Health, 2001; Norman & Brain, 2005).

The HBM was originally designed as a cognitive model of behavior change, and posits that behaviors are based on two different consequences, either health enhancing or health threatening (Neff & Crawford, 1998). Additionally, the HBM is related to the value expectancy theory, which argues that each individual is a rational 'actor' who contemplates costs and benefits in order to maximize gains (Neff & Crawford, 1998). The HBM maintains that particular cues (internal or external) are necessary for the

initiation of health-related behavior modifications (Neff & Crawford, 1998). Furthermore, the HBM is used to predict and explain factors associated with medical and mental health service utilization (Henshaw & Freedman-Doan, 2009). The HBM also includes barriers associated with healthcare services and the likelihood of successful outcomes (Constantine, Myers, Kindaichi & Moore, 2004). Researchers examined the HBM cross-culturally to document the beliefs associated with the cause(s) of a disease, the appraisal of a particular disease (Chadda et al., 2001), the different perceptions that exists within diverse communities (e.g. stigma towards mental healthcare; Chadda et al., 2001), and the cultural mistrust or perceived cultural insensitivity that may be a barrier to treatment (Poston, Craine & Atkinson, 1991). Revised versions of the HBM incorporate demographic variables, such as age, gender, socioeconomic status, education and knowledge to predict behaviors and/or illnesses (Janz, Champion & Stretcher, 2002).

The HBM is used to understand health behaviors and circumstances in which health behaviors change (Taylor, 1999). Accordingly, the HBM is employed in a variety of healthcare settings and research. For example, research with the HBM examined the relationship between beliefs and health behaviors among people with sleep apnea (Ali, 2002), cardiovascular disease (Ali, 2002), stroke (Sullivan et al., 2008), coronary heart disease (Ali, 2002), breast cancer (Norman & Brain, 2005), tuberculosis (Barnhoorn & Adriaanse, 1992), diabetes (Wdowik et al., 2001) and hepatitis C (Ali, 2002). Sullivan and Waugh (2007) examined the role of the HBM in stroke prevention and found that beliefs and attitudes toward cerebrovascular disease were significant predictors of healthier outcomes in participants at risk for stroke. In HIV/AIDS psychosocial research,

the HBM is used to understand sexual risk taking and decision making in medication adherence and condom use (Rosenstock et al., 1994).

This study expands on the HBM because I included several psychosocial variables not extensively studied in published literature. I examined supplementary predictor variables, such as stigma, trauma, ethnicity, falls, general medical conditions and HIV-opportunistic infections, which are relevant in disease progression, particularly HIV-associated neurocognitive impairment, in a sample of people living with HIV/AIDS.

HIV-Associated Neurocognitive Disorders

History

Early in the HIV/AIDS epidemic, researchers and clinicians detected organic symptoms in a substantial amount of people with advanced stages of the disease (Perry & Jacobsen, 1986). Snider et al. (1983), hypothesized that the virus affected the central nervous system (CNS), which would account for the many neurological and psychological symptoms exhibited. Researchers also found that many cases of dementia were comorbid with opportunistic infections linked to HIV (Navia et al., 1986). HAD, a neurocognitive disease that is directly attributed to HIV, was controversial until 1987 when Grant and colleagues published the first comprehensive study of HIV-associated neurocognitive deficits. Grant et al. (1987), found objective data to suggest neurocognitive impairment across all stages of HIV disease, such as executive functioning, episodic memory, motor skills, attention/working memory, language, sensory perception, and information processing speed (Grant et al., 1987). The AIDS

Task Force of the American Academy of Neurology (AAN, 1991), outlined diagnostic guidelines for the classification of HIV-associated cognitive impairments. From this classification two types of impairment were distinguished: HIV-associated dementia (HAD) with motor, behavioral/psychosocial or combined features, and minor cognitive motor disorder (MCMD). To meet criteria for a diagnosis of HAD, at least two neurocognitive domains must be impaired, as well as evidence of impaired motor functioning, neuropsychiatric or psychosocial functioning (e.g., motivation, lability, and social behavior; AAN, 1991). When HIV-associated neurocognitive impairments do not meet criteria for HAD, but impairment exists in at least two neurocognitive or behavioral areas, a diagnosis of MCMD is appropriate. In 1995, Grant and Atkinson expanded the AAN system to include an additional diagnosis of subsyndromic neurocognitive impairment, for those who demonstrated mild neurocognitive deficits that do not noticeably interfere with work or daily living. Though the AAN's criteria to distinguish cognitive impairments were fairly accurate; the advances in the field (e.g. HAART) rendered this classification system inadequate (Antinori et al. 2007). Currently, despite advances from HAART, new yearly incidence of HAD is 10–25%, which suggests that immunology, genetics, and psychosocial variables continue to play a role in predicting cognitive impairment in those with HIV/AIDS (Robertson et al., 2007).

Diagnosis

The National Institutes of Mental Health changed the criteria for a diagnosis of HAND (Antinori et al., 2007; McGuire, 2003). HIV infection can induce several

neurocognitive impairments that result in three HAND diagnoses: asymptomatic neurocognitive impairment (ANI), mild neurocognitive disorder (MND), and HIV-associated dementia (HAD).

- Asymptomatic neurocognitive impairment (ANI): To meet criteria for this diagnosis, individuals exhibit impairments in ≥ 2 ability domains, such as attention, working memory, verbal, abstract reasoning, executive functioning, memory, processing speed or visuospational processing. Moreover, individuals must score ≥ 1 standard deviation below the mean for age/ education on standardized neuropsychological tests. According to this diagnosis, impairments do not interfere with daily functioning.
- Mild neurocognitive disorder (MND): To meet criteria for this diagnosis, individuals score ≥ 1 standard deviations below the normative mean on neuropsychological measures and display ≥ 2 impairments in ability domains, resulting in mild changes of daily functioning, inefficiency in work, homemaking or social functioning.
- HIV-associated dementia (HAD): To meet criteria for this diagnosis, individuals must exhibit impairments in ≥ 2 ability domains, particularly in learning information, processing speed, and attention/concentration, and score ≥ 2 standard deviations below the mean on neuropsychological measures. Individuals must also display significant impairment in daily functioning.

These domains of cognitive functioning are assessed using performance-based neurocognitive batteries and interpreted using demographically-appropriate normative data (Antinori et al., 2007). To determine criteria for a diagnosis, practitioners often

conduct a mental status exam, such as the International HIV-Dementia Scale (IHDS), which is a highly reliable and valid screening tool (Morgan et al., 2008; Sacktor et al., 2005). To make an accurate diagnosis, neurocognitive impairment due to comorbid conditions and other preexisting causes for dementia must be excluded, such as Alzheimer's disease, neurological disease, or substance abuse (Antinori et al., 2007).

Neuropathology of HIV

The CNS is a major target for HIV infection. Many CD4-cells, such as T-cells and monocytes, are infected by HIV and consequently circulate in the blood and cross the blood-brain-barrier to transmit the infection (Haase, 1986). HIV targets the CNS because neurons in the brain express chemokine receptors (e.g., CXCR4 and CCR5) that are necessary for HIV's entry into cells (Hult et al., 2008). As a result, HIV disrupts the fronto-striato-thalamo-cortical loop circuitry (Hult et al., 2008). Unlike other neurodegenerative disorders that have affinity for a specific brain region, HIV affects the structures and functions of the brain in a pervasive manner (Thompson et al., 2005). The HIV virus appears most densely located in the basal ganglia, subcortical regions, and frontal cortex (Thompson et al., 2005). Similarly, the virus will cause pathological changes in the deep gray and white matter regions of the brain. Neurons are not directly infected with HIV; rather the neuron is damaged from proinflammatory neurotoxins that circulate in the CNS (Sahai-Srivastava, 2008). Once HIV infects the brain, severe pathology ensues, such as HIV encephalitis and chronic inflammation. Neural injury may be amplified by comorbidities, such as substance use, medical diseases (e.g. hepatitis C),

and normal aging (Foley et al., 2008; Woods et al., 2009). Furthermore, the presence of viral proteins such as gp41 and gp120 in the basal ganglia and frontal lobes, are significantly correlated with the severity of HAND (Mukhtar et al., 2002).

The neuropathology of HIV infection changed due to the improved antiretroviral therapies (Kaul, 2009). Despite HAART's benefits, HIV-positive individuals may experience neural damage, particularly neuronal cell death. Though, HAART successfully prevents many of the end stage complications associated with AIDS, HAART cannot fully eliminate the prevalence of minor HIV-associated neurocognitive impairment (Ghafouri et al., 2006). Similarly, HAART cannot prevent the deterioration or enable the restoration of cognitive functions due to its limited access into the CNS (Liner, Hall & Robertson, 2008). Ghafouri et al. (2006) suggest that even in those treated with HAART, HAD and other CNS opportunistic infections are still common. Moreover, the use of HAART in the long term, poses a potential toxicological risk that may affect neurocognitive performance (Letendre et al., 2008). Fortunately, with the prolonged use of HAART, people living with HIV are living longer lives; but, are also at increased risk of CNS infectious diseases, substance use disorders, and medical conditions that are side-effects of the disease and medications (Letendre et al., 2008).

Course of Cognitive Impairment in HIV

Unlike other neurocognitive disorders, such as Alzheimer's and Huntington's diseases, HAND is not necessarily a progressive disorder (Cole et al., 2007). Some individuals may show recovery of certain neurocognitive functions when highly adherent

to HAART regimens (Cole et al., 2007). Robertson et al. (2007) suggest that a significant proportion of individuals with HAND improve. In a recent study, 56% of participants with HAND at baseline remained impaired at follow-up, whereas 44% no longer met criteria at follow-up (Robertson et al., 2007). Furthermore, McArthur (2004) proposed that approximately 21% of people living with HIV can transition from mildly impairment to unimpaired, and 23% can transition from unimpaired to mildly neurocognitively impaired.

Evidence of Cognitive Impairment in HIV

Neuropathological, neuropsychological and in-vivo imaging studies, find evidence of HAD and HAND despite adherence to HAART (Brew et al., 2009). Moreover, there are commonalities between the development of HAND/HAD and other neurodegenerative diseases, such as Alzheimer's and Parkinson's disease (Brew et al., 2009; Khanlou, Moore & Chana, 2008). The presence of HIV may accelerate normal aging and facilitate the development of neurodegenerative diseases before the manifestation of HAD (Brew et al., 2009; Esiri, Biddolph & Morriss, 1998). Imaging studies found that HIV affects the frontal and parietal cortex and subcortical structures (Castelo et al., 2006; Chang et al., 2001; Ernst et al., 2002), which results in decreased brain activation (Chang et al., 2004). HIV is also related to tissue loss in the frontal and parietal areas (Thompson et al., 2005) and in the caudate nucleus (Ances et al., 2006). In people living with HIV/AIDS, alterations in brain activity is documented even when performance on neuropsychological tests are within normal limits (Castelo et al., 2006;

Ernst et al., 2002). Moreover, motor speed, information processing, working memory, learning, memory recall, executive functioning, visuoperception and attention span are most typically impaired (Courtney, 2004; Marcotte et al., 2006; Postle, 2006).

Motor Skill, Information Processing and Psychomotor Speed

People living with HIV may experience movement disorders such as chorea and dyskinesia (Mirsattari et al. 1999). In Valcour et al. (2008), older adults living with HIV were disproportionately more impaired on the Unified Parkinson's Disease Rating Scale, which particularly measures bradykinesia, tremors, and manual dexterity (Carey et al., 2004). Furthermore, rapid alternating movements and finger contractions are increasingly difficult in those with HIV based on electrophysiological assessments (Arendt et al., 1990). According to Hardy and Hinkin (2002), bradykinesia (motor retardation) and bradyphrenia (decreased information processing) are the most prominent features of people living with HIV (Hardy & Hinkin, 2002). Similarly, decreased information processing is observed on tasks involving motor (e.g., Trail-Making) and non-motor (e.g., Stroop Color-Word Test) demands. Both cognitive and motor speed (i.e. psychomotor speed) are further compromised on tasks requiring a combination of such processes (Martin et al., 1999).

Learning and Memory Recall

Approximately 40-60% of people living with HIV/AIDS experience deficits in episodic memory (Rippeth et al., 2004). These deficits are seen on verbal and visual tasks

(Rippeth et al., 2004). Apart from impaired psychomotor speed, people living with HIV experience changes in episodic memory (Carey et al., 2004), and episodic memory steadily decreases with progression of the disease (Reger et al., 2002). Episodic memory deficits are associated with difficulties in encoding and retrieving information (Delis et al., 2000) and difficulties with immediate and delayed recall (Woods et al., 2005). Free recall deficits are associated with impaired usage of strategic encoding, such as semantic clustering and increased perseveration (Carey et al., 2006; Peavy et al., 1994), which may exacerbate other neurocognitive impairments associated with HIV (Gongvatana et al., 2007). Furthermore, prospective-memory is typically impaired in people living with HIV, particularly in regards to daily functioning, such as self-care, employment and medical compliance (Woods et al., 2007; 2008).

Attention and Working Memory

Attention and working memory impairments are correlated with HIV progression (Reger et al., 2002). Early after infection with HIV, people may experience minimal alterations in attention and concentration; however, with the onset of AIDS, mild to moderate deficits emerge (Brew, 2004; Reger et al., 2002). Attentional impairments are related to increased frontoparietal activation (e.g., Chang et al., 2004), that results in difficulties with orientation (Martin et al., 1995), and response inhibition (Hinkin et al., 1999). Furthermore, Hinkin et al. (2000), found that in people living with HIV/AIDS, complex tasks (i.e. those requiring both auditory and visual performance) were more impaired than single tasks. Moreover, Bassel et al. (2002) found that attention and

working memory impairments combined with depression were responsible for the majority of cognitive complaints in people living with HIV. Other studies found that attention and concentration impairments were associated with nonadherence to HAART (Levine, 2008), and often result in decreased daily functioning (Marcotte et al., 2006).

Executive Functioning

Impaired executive functioning (e.g. judgment, problem solving, goal-directed behavior and cognitive flexibility), is associated with HIV, especially in later stages of the disease (Dawes et al., 2008; Mega & Cummings, 1994). People living with HIV typically score lower on tasks, such as the Stroop Color Word Test (Tozzi et al., 1999), the Trail Making Test B (Reger et al., 2002), and the Wisconsin Card Sorting Test (Carter et al., 2003), all which require cognitive shifting and response inhibition. Similarly, impaired executive functioning is also seen on measures of judgment and impulsivity (Benedict et al., 2000), which is especially noteworthy since approximately one-third of people living with HIV continue to engage in risky sexual behaviors after diagnosis (Janssen et al., 2001; Martin et al., 2004).

Visuoperception

Visual-spatial cognition is the ability to detect, understand and manipulate visual information in a given context. Olesen et al. (2007) found that HIV-positive participants displayed more difficulty with mental rotation of hands and 3D objects, than HIV-negative participants. Furthermore, Hardy et al. (2004) suggest that though only minor

deficits exist in people living with HIV, these deficits may ultimately increase the risk of negative life events, such as car-related accidents (Marcotte et al., 2006).

Speech and Language

People living with HIV typically do not suffer from aphasia; however, the presence of CNS opportunistic infections increases the likelihood of speech related impairments (Robertson, Liner & Heaton, 2009). For instance, Lopez et al. (1994) found evidence of mild dysarthria and Mathew and Bhat (2008) found the presence of lower speech volume and vocal tremors in a sample of HIV-positive participants. Likewise, communication deficits may be evident, such as inappropriate interruptions during conversations, poor lexical decision making and reduced fluency of speech (McCabe et al., 2007). Approximately 40% of people living with HIV experience clinically significant impairments in verbal fluency (Rippeth et al., 2004). Verbal fluency deficiency is associated with the neurodegeneration of the frontal cortex and caudate nucleus (Hestad et al., 1993). In Woods et al. (2004), HIV-positive participants with verbal fluency deficiency could not fully search for and/or retrieve lexical information. Furthermore, approximately 91% of HIV-positive participants displayed phonemic intrusion errors (Woods et al., 2004). Similarly, in Millikin, Trepanier & Rourke (2004), people living with AIDS displayed greater difficulty with letter and category fluency than difficulty with semantic clustering.

Negative Affect

The prevalence of depression in people with HIV is significantly higher than in the general public (Ciesla & Roberts, 2001). Approximately 10% meet criteria for a diagnosis of current major depressive disorder and 36% meet criteria for a recent major depressive disorder (Bing et al., 2001; Cruess et al., 2003). In people living with HIV, depression is linked to negative health outcomes, such as poor treatment adherence (Kemppainen, 2001), immune compromise and disease progression (Ickovics, Hamburger & Vlahov, 2001). Individuals who are HIV-positive and are symptomatic tend to exhibit more neuropsychological deficits than those who are asymptomatic (Carter et al., 2003). Particularly, mood disturbances and comorbid physical symptomatology are associated with neurocognitive impairment (Rourke, Halman & Bassel, 1999). For example, in Chan et al. (2007), depression was a significant predictor of subjective memory complaints. Furthermore, participants who were dissatisfied with their social networks and relationships were more likely to endorse memory deficits, such as forgetfulness, poor concentration and mental and motor slowing (Chan et al., 2007).

Andersson and Bergedalen (2002) proposed that apathy, a component of depression, is a significant variable associated with cognitive compromise and alterations of the CNS. In people living with HIV, apathy is correlated with decreased working memory and impairment in frontal-subcortical circuitry (Castellon et al., 1998). Furthermore, Castellon et al. (2006), found that elevated Beck Depression Inventory (BDI) scores were associated with neurocognitive performance, particularly deficits in executive functioning, verbal memory and motor performance. In McAllister-Williams,

Ferrier and Young (1998), researchers hypothesized that the neurochemical and neuroanatomical mechanisms involved in negative affective states are similar to those in dementia. This neural dysregulation may be related to alterations of the hypothalamic-pituitary-adrenal (HPA) axis, such as the increase of cortisol and changes in dopamine levels (Berger & Arendt, 2000). Additionally, depression and negative affect are associated with frontal hypometabolism and psychomotor slowing (Mayberg, 1997; Millikin, Trépanier, & Rourke, 2004). Although the existing literature that links mood to dementia is somewhat controversial (Henderson et al., 1997), sufficient evidence exists that suggests depression plays a vital role for those with mild cognitive impairment in the progression of dementia. Lu and Scicutella, (2009), suggest that every point increase on the BDI is associated with a 3% higher risk of developing Alzheimer's disease. Thus, negative affect may be a risk factor associated with the onset of dementia and future research is needed to examine this relationship (Jorm, 2008).

Many people living with HIV have a long history of negative experiences, such as stigmatization, traumatic events, and discrimination (Whetten et al., 2008). These negative events may exacerbate an already previous history of mental illness and/or manifest into heightened levels of anxiety, anger and depression. Negative affect is associated with poorer medication adherence, disease progression and overall lower quality of life (Whetten et al., 2008).

Stigma

Since the onset of the HIV/AIDS epidemic, people living with the disease

reported stigmatization and oppression (Collani, Grumm & Streitcher, 2010). HIV-related stigma is typically layered onto other stigmas related to homosexuality, prostitution and/or substance use (Herek, 1999). According to Herek (1999), stigma occurs when a person with HIV or those associated with that individual, are discounted, discriminated and discredited (Snyder, Omoto & Crain, 1999). Likewise, people living with HIV are sometimes viewed as invaders of an otherwise healthy society and are considered deviations and a liability (Bunting, 1996). Some believe that HIV-related stigma exists because the threat of infectivity and transmission produces an intolerable fear (Berrenberg et al., 2002). Connors and Hely (2007), suggest that HIV-related stigma stems from homophobic ideologies, the fear of contamination and mortality, and false beliefs regarding transmission (Dias, Matos & Goncalves, 2006). Unfortunately, the avoidance of people living with HIV to alleviate such irrational fears causes segregation and isolation of individuals who are in need of help and support (Varas-Diaz et al., 2005). Rush (1998) maintains that personal control over infectivity, reduces stigmatization and lessens the likelihood that empathy from others will be received. In a study conducted by the CDC (2000), 1 in 5 participants believed that people infected with AIDS through sex or drugs 'got what they deserved.'

In the United States, HIV-related stigma may manifest as anger and negativity towards people living with the virus/disease (Herek, Capitanio & Widaman, 2002). Consequently, many internalize this stigma and perceive themselves as devalued members of society (Crocker, Major & Steele, 1998; Kang, Rapkin & DeAlmeida, 2006). HIV-related stigma is linked to negative self-image (Emlet, 2007). Many people living

with HIV are unable to meet occupational, familial and social requirements due to reduced health and disease progression; thus, negative self-evaluation is further exacerbated. Consequently, people who experience stigma may inevitably suffer from clinical levels of depression, and experience hostility and anxiety (Jones et al., 1984; Leary & Schreindorfer, 1998; Swendeman, et al., 2002). Stigma is both directly and indirectly related to stress (Heatherton et al., 2000). Lee, Kochman and Sikkema (2002), found a correlation between internalized stigma and clinical symptoms of anxiety, depression and hopelessness. Moreover, Clark et al. (2003), confirm that stigma predicts poor psychological adjustment. The feeling of alienation coupled with shame, guilt, fear and a loss of social support, all have a great impact on HIV/AIDS prognosis and morbidity (Leary & Schreindorfer, 1998). HIV/AIDS-associated discrimination creates a barrier to resources, such as healthcare, employment, housing and education (Heatherton, et al., 2000). Furthermore, the stigma associated with the physical changes that accompany the disease (e.g. disfigurement and disability) contribute to chronic stress (Heatherton et al., 2000). Consequently, people living with HIV not only suffer from their illnesses, but from these additional burdens and daily stressors (Allison, 1998).

Recently, researchers linked ethnicity with stigma. For example, in Emlet (2007), African-Americans reported significantly higher scores on stigma than other participants. Furthermore, in the last decade, HIV-related stigma has become a concern in Latino(a) communities (Kaiser Family Foundation, 2001). Familismo, described as family support, can also be a source of stigma for people with HIV (Ortiz-Torres, Serrano-García & Torres-Burgos, 2000). Due to the tight bond within the family system, many find it

impossible to detach from their family; thus, stigma is frequently relived (Varas-Diaz, Serrano-Garcia & Toro-Alfonso, 2004).

HIV-related stigma also exists in healthcare settings. In a study conducted by the National AIDS Research Institute (NARI), clinicians made several judgments of their patients based on perceptions of immoral behavior associated with HIV (NARI, 2002-2003). They found that some clinicians expressed a fear of touching a patient with HIV, and some considered treating a patient with HIV as a 'special duty' (NARI, 2002-2003). Consequently, stigma (e.g. the fear of being rejected or 'outed') may be a deterrent to medical and/or psychological care (Chesney & Smith, 1999). This in turn may delay HIV testing and diagnosis (Mawar et al., 2005). In Mawar et al. (2005), HIV-positive women hesitated to seek psychological treatment due to fear that their diagnosis would be disclosed. Similarly, they found that women resisted HIV medications due to the fear that these medications could potentially 'out' their diagnosis (Fife & Wright, 2000). Stigma may also interfere with adherence to treatment regimens and the implementation of adaptive health behaviors (Chesney & Smith, 1999). Given these associations, stigma is related to long term health outcomes and immune compromise (Ickovics, Hamburger & Vlahov, 2001; Leonard, 2000).

Trauma

People living with HIV/AIDS often endure a history of trauma (Whetten et al., 2006). In a study of 357 HIV-positive participants, 45% (68% female and 35% male) reported an episode of sexual abuse before age 15, and among those 80% were assaulted

at least twice with an average of ten times (Kalichman, 2002). Furthermore, 34% of females and 27% of males disclosed a history of childhood physical abuse (Kalichman, 2002). Researchers did not identify differences in history of trauma based on race/ethnicity, age, or education; however, HIV-positive men that were gay or bisexual experienced the most abuse (Kalichman, 2002). Additionally, in Whetten et al. (2006), women and gay or bisexual men experienced and reported more abuse than others, such that the majority reported at least one traumatic event in his/her lifetime (Pence et al., 2007). Zierler et al. (2000) suggest that approximately 21% of women and 12% of MSM (men who have sex with men) living with HIV/AIDS have experienced physical abuse and violence since diagnosis. In Leserman et al. (2007), researchers followed HIV-positive men and women for approximately 40 months and found that participants with more traumatic events were at an increased risk of HIV-specific morbidity (Leserman et al., 2007). In this study, trauma was associated with increased bodily pain, somatic symptoms, disability and use of healthcare services (Leserman et al., 2005; Leserman et al., 2007). Unfortunately, traumatic experiences do not dissipate with a diagnosis of HIV. In the National HIV Costs and Service Utilization Study (HCSUS), 20.5% of females, 11.5% of MSM and 7.5% heterosexual males, reported physical abuse by an intimate partner or a close friend or relative since diagnosis (Zierler, 2000). Moreover, Kimerling et al. (1999) found that participants with a history of at least three crime related traumas had a higher incidence of HIV-related physical symptoms than those with two or less.

Trauma has persistent effects on health and health behaviors in people living with HIV/AIDS (Whetten et al., 2008). Trauma, particularly physical and/or sexual abuse,

predicts higher levels of anxiety, depression, and post traumatic stress disorder (PTSD), etc. (Kalichman et al., 2002; Leserman et al., 2005). Additionally, trauma is associated with poor medication adherence and increased HIV risk behavior (Kalichman et al., 2002). Leserman and colleagues found that higher averages of cumulative traumatic events predicted faster progression from HIV to AIDS during 5.5, 7.5 and 9 year follow-up periods (Leserman et al., 1999; Leserman et al., 2000; Leserman et al., 2002). Also, for every increase in traumatic events, the risk of developing AIDS doubled (Leserman et al., 2000). Moreover, Kimberling et al. (1999), conducted a year-long follow-up study with 618 HIV-positive participants and found that trauma (e.g. sexual/physical abuse), coupled with a diagnosis of PTSD, was associated with greatest decrease of CD4 cell counts during that one year period. Other researchers confirm a direct association between stressful life events (e.g. trauma) and HIV disease progression and AIDS-related morbidity (Leserman et al., 2008; Patterson et al., 1996). Trauma is associated with faster development of opportunistic infections (Mugavero et al., 2007) and greater increase in viral load (Ironson et al., 2005). In comparison to those with HIV/AIDS and no history of trauma, exposure to traumatic episodes is associated with poorer romantic relationships, impaired relational and social roles, increased disability, increased emergency room visits and more frequent hospitalizations (Leserman et al., 2005). The emotional sequelae that are a consequence of trauma may negatively affect health-protective behaviors related to HIV/AIDS morbidity and prognosis (Liebschutz et al., 2000).

A history of traumatic experiences can have a profound effect on memory and memory recall (Bremner & Narayan, 1998). During a traumatic experience, such as rape,

an individual may feel trapped in the situation, thus, the fight or flight response is rendered inoperable. As autonomic arousal increases, the individual becomes numb due to the infiltration of opiates in the CNS, which causes a dissociation in processing and encoding (Bremner, Southwick & Charney, 1999). When the traumatic event is over, the individual is stuck in between a state of dissociation and fight or flight. With time, the individual adapts to this state of 'in-between' and memory is compromised (Burgess & Phillips, 2009). Shannon et al. (2009) suggests that childhood trauma is associated with cognitive impairment and a reduction of hippocampal volume in adulthood.

Approximately 16-54% of people living with HIV/AIDS also suffer from PTSD symptomatology, exceeding rates of the general population (Boarts et al., 2006; Pence et al., 2007). PTSD is related to severe alternations in memory (Yaffe et al., 2010). A significant correlation exists between veterans exposed to trauma in war, the manifestation of PTSD, and the later onset of dementia (Yaffe et al., 2010). Furthermore, Yaffe and colleagues found that 17.2 % of veterans with a previous diagnosis of PTSD, later developed dementia in a seven year follow-up study. The veterans with PTSD had a 10.6% risk of developing dementia in comparison to those who did not, who only had a 6.6% risk. The association between chronic stress and trauma may indirectly predict dementia, due to profound hippocampal damage, altered neurotransmission and hormone production associated with stress (Yaffe et al., 2010). Moreover, those with PTSD have decreased frontal lobe volume, which leads to deficits in psychomotor speed and frontal lobe processing (Hart et al., 2008; Koenen et al., 2001). Alterations in memory are a part of the clinical presentation of people with PTSD. Individuals diagnosed with PTSD often

experience deficits in declarative memories, fragmented memory and dissociative amnesia (Bremner, Southwick & Charney, 1999).

Ethnicity

Ethnic differences exist on neuropsychological test performances (Tang et al., 2001). Poor test performance may be related to lower levels of education and lower socioeconomic status (SES; Tang et al., 2001). However, in Manly et al. (1998; 2002), African-American participants scored lower on verbal and non-verbal neuropsychological tests, even when age, gender, education, disease stage and SES were matched to European-American participants. In Mehta et al. (2003), Latino participants received lower mental status exam scores, in comparison to other ethnicities, which predicted increased neuropathology and cognitive decline. Similarly, in Ramani et al. (2001), ethnicity was associated with poor performance on neuropsychological assessments for dementia. Ramani and colleagues found that African-American women performed poorly on tests of verbal memory and motor speed, whereas, Latinas had more difficulty with tasks of psychomotor speed, when compared to European-American female participants. Although education was matched in this sample, Ramani et al. (2001) believe that ethnic women may perform poorer on neuropsychological tests due to decreased quality of education.

Researchers hypothesize that neuropsychological assessments may overdiagnose cognitive impairment in African-Americans, Latinos and other minority ethnic groups (Mast et al., 2001). Future research is needed to address ethnic discrepancies and separate

norms are needed for a better comparison. Similarly, many assessments are limited since they do not account for acculturation, which results in measurement bias and poor diagnostic specificity in cross-cultural test-taking settings (Mahurin, Espino & Holifield, 1992; Marcopulos, 1997). Robertson et al. (2007), contend that normative data for Latino(a) individuals is especially crucial due to the under development and lack of appropriate protocols that are still used in neuropsychological testing.

Currently, African-American and Latino(a) communities are disproportionately affected by HIV/AIDS (CDC, 2008). Ethnicity may be associated with the onset and progression of HAD and other neurocognitive impairments (Manly et al., 1998). In, Mason et al. (1998), researchers found that ethnic differences, primarily among African-Americans, only were seen once HIV progressed into AIDS. Mehta et al. (2003), believe that ethnic minorities are more susceptible to developing dementia due to the fact that they experience more medical symptoms, deteriorated health status and chronic stress that negatively affects cognition (Mehta et al., 2003). Although, to date there are no known genetic factors associated with greater prevalence of dementia among ethnic individuals; medical conditions such as hypertension, coronary heart disease, and diabetes are known risk factors and are more relevant in ethnic communities. The impact of these conditions on the incidence of cognitive impairment and on the progression of dementia is well documented (Brancati, Kao & Folsom, 2000; Sundquist, Winkleby & Pudaric, 2001). Vascular dementia is a type of dementia linked to heart disease and stroke. Although, this diagnosis exists on its own, it can also occur simultaneously with other forms of dementia such as HAD and Alzheimer's type (Yale University, 2001).

Symptom Load

The mechanism by which HIV enters the CNS is associated with the infiltration of microglia and macrophages (Ghafouri, 2006). HIV produces aggregate proteins in the brain and lowered hemoglobin blood levels (Stern et al., 2001). HIV is a viral agent that attacks the CNS; however, other pathogens (e.g. opportunistic infections, bacteria, cancers) that cause neuronal injury and death are responsible for the deterioration of neurocognitive functioning (Gonzalez-Duarte, Cikurel & Simpson, 2004). HIV primarily affects white matter, the basal ganglia, hippocampus and spinal cord. HIV-related illnesses, such as opportunistic infections, will compromise the immune system; thus, lowered immunity coupled with high viral loads may predict mental status changes and CNS-related diseases (Foltzer, 2005).

In people living with HIV, the degeneration of CD4 cells causes a dysregulation of macrophages and increased production of proinflammatory cytokines and chemokines (Griffin, 1997). Furthermore, systemic infections in the blood and abnormalities of cerebrospinal fluid are common in people with HAD (Simpson & Tagliati, 1994). Brew et al. (1990), suggest that cerebrospinal fluid, microglobulin and neopterin levels, which are all indicators of inflammation, are significantly correlated with the severity of HAD. Similarly, tumor necrosis factor is also associated with cognitive impairment and HAD (Choi, 1988).

HIV yields neurological symptoms and immunodeficiency, which often results in opportunistic infections and comorbid medical diseases (McArthur, 2003). Infectious diseases that infiltrate the CNS may ultimately predict dementia. These infections are

caused by a variety of sources such as abnormal protein, bacteria, and parasites (Almeida & Lautenschlager, 2005). Furthermore, approximately 16% of people living with HIV are also infected with hepatitis C (Radkowski, Wilkinson & Nowicki, 2002). The combination of both HIV and hepatitis C often causes encephalopathy. Radkowski, Wilkinson and Nowicki (2002), suggest that hepatitis C has profound effects on the CNS, and may predict cognitive dysfunction. hepatitis C is associated with impairments in attention, learning, psychomotor speed and mental flexibility (Hilsabeck et al., 2002; Kramer, Bauer & Funk, 2002). Apart from hepatitis C, other common co-infections include herpes simplex virus type I, Varicella virus, cytomegalovirus, Epstein-Barr virus and encephalitis (McArthur, 2003).

HAD is also linked to drug addiction, hydrocephalus, traumatic brain injury and brain tumors (Hulse et al., 2005; Starkstein & Jorge, 2005). Tumors most often seen in HAD include CNS lymphomas and metastatic disease (Starkstein & Jorge, 2005). Moreover, B12 deficiency, thyroid disease and Addison's disease are also typical in those with HAD (Tedaldi et al., 2003). People living with HIV often suffer from comorbidities associated with liver disease, such as anemia, lipodystrophy and hepatocelluar carcinoma (Tedaldi et al., 2003).

In people living with HIV, cardiovascular disease is the fourth leading cause of death (Grinspoon & Carr, 2005). HAART is linked to cardiovascular disease (Friis-Moller et al., 2007) and atherosclerosis (Hsue et al., 2004) among people living with HIV/AIDS. Moreover, the coupling of HIV and HAART causes endothelial injury, increased vascular inflammation and carotid atherosclerosis (Stein, Klein & Bellehumeur,

2001). In a large epidemiological study (D:A:D; 2003), researchers found that HIV-positive individuals on HAART were at higher risk of myocardial infarction and cardiovascular disease (Friis-Moller et al., 2007). In a joint study conducted by the American Heart Association and the American Academy of HIV Medicine (Grinspoon, Grunfeld & Kotler, 2008), researchers found the risk of heart attack was 70%-80%, and people living with HIV are more than three times more likely to develop coronary artery disease, than the general public not taking antiretroviral therapies (Vittecoq, Escaut & Chironi, 2003).

Protease inhibitors increase LDL cholesterol, triglycerides, blood pressure, and risk for diabetes (Lee et al., 2004). Nucleoside analogs also increase risk for diabetes and insulin resistance (Tien et al., 2007). Thus, antiretroviral therapies cause metabolic disorders (Eron, Yeni & Gathe, 2006) and dyslipidemia (Grinspoon & Carr, 2005), which together increase the risk of cardiovascular disease. Furthermore, people living with HIV have increased c-reactive protein (CRP) concentrations in the blood (Hsue, Lo & Franklin, 2005). Higher concentrations of CRP, a cardiovascular inflammatory biomarker; are associated with HIV disease progression, regardless of CD4- cell count and/or viral load (Hsue, Lo & Franklin, 2005). Thus, cardiovascular risk factors are seen more than not in people living with HIV/AIDS. Though, participants with a diagnosed heart condition were excluded from this study (see method); cumulative cardiovascular risk factors were examined as part of symptom (described as general medical conditions).

Hypertension, dyslipidemia, obesity, and diabetes mellitus, are all associated with an increased risk of cognitive decline and dementia (Ligthart, Moll van Charante, Van

Gool & Ricard, 2010). Magnetic resonance imaging (MRI) and computed tomography (CT) scans of patients with vascular risks demonstrate deficits in white matter lesions in the CNS (Erkinjuntti, 2005). Foley et al. (2010) found that cardiovascular risk factors were associated with declines in processing speed in participants with HIV, even when controlling for age. Vascular risk factors also predict cognitive decline in HIV (Foley et al.., 2010). McMurtray, Nakamoto, Shikuma and Valcour (2008) suggest that white matter lesions caused by small-vessel ischemic disease, were more common in people living with HIV with higher systolic blood pressure, than those without. Also, aging individuals living with HIV were more at risk for fronto-subcortico neurocognitive dysfunction, subcortical pathology, and frontal lobe impairments, than younger individuals (Sacktor et al., 2007). Along with sensorimotor changes, vascular risks are linked to cortical cognitive impairments, such as aphasia, apraxia, and agnosia (Erkinjuntti, 1999), decreased executive functioning (Erkinjuntti, 1999), and structural changes in the thalamus, basal forebrain, and caudate nucleus (Erkinjuntti 1999; O'Brien, Erkinjuntti, Reisberg, 2003).

Falls

Syncope (a temporary loss of posture and/or consciousness) is a symptom of cardiovascular disease, particularly due to aortic stenosis, cardiomyopathy, carotid sinus syndrome, ventricular tachycardia and arrhythmia (Colledge, 2000). Syncope is caused by orthostatic hypotension, which is exacerbated by medication use, diabetes, neuropathy and Addison's disease. Syncope is positively correlated with falls and loss of

consciousness (Colledge, 2000). Moreover, individuals with claudication and lower systolic blood pressure experience more falls (Gardner & Montgomery, 2001; Puisieux, Bulckaen, Fuachais, Granier & Dewailly, 2000). Head injuries, often a consequence of falls, can lead to neurocognitive decline (Luukinen, Viramo, Koski, Laippala & Kivela, 1999). In Luukinen, Koski, Honkanen and Kivela (1995), fall-related brain injury was associated with mild to moderate dementia. In a sample of participants who experienced brain trauma from a fall, participants were at increased risk of dementia, especially those carrying the apolipoprotein E (ApoE) e4 allele that alters brain activity, decreases neural repair and impairs cell growth (Luukinen et al., 2005; 2008). ApoE e4 allele increases the risk of cognitive impairment and lowers cognitive performance, particularly in older individuals (Kutner, Erlanger, Tsai, Jordan & Belkin, 2000). Consequently, ApoE e4 potentiates the risk of dementia after a fall has occurred (Mayeux, Ottman & Maestre, 1995).

Rationale for Proposed Study

The primary objective of this study is to relate several psychosocial predictors to neurocognitive impairment in people living with HIV/AIDS. The predictors were selected based upon the existing theories that explain the relationship between health/illness and behavior/lifestyle. Thus, my study builds on the HBM.

While the HBM is used to predict health outcomes in other clinical populations (e.g. coronary heart disease; Ali, 2002), there is insufficient research on its relevance to neurocognitive impairment in HIV/AIDS populations. This study examines several

under-studied predictors of HIV-related neurocognitive impairment, such as stigma, trauma, ethnicity, HIV-opportunistic infections, general medical conditions, and falls. I hope that my results will contribute to revisions of older health models as well as suggest avenues for prevention and remediation for people living with HIV.

1) I hypothesized that increased stigma is positively associated with neurocognitive impairment, particularly motor speed, psychomotor speed and memory recall. I believe that with increased stigma, participants would experience more HIV-related neurocognitive impairment. 2) I also hypothesized, that trauma is positively correlated with neurocognitive impairment. Trauma history, such as the exposure to physical or sexual abuse and/or crime and natural disaster (s) would be positively associated with HIV-related neurocognitive impairment. For instance, the more one has experienced trauma, the more one will experience difficulties with motor speed, psychomotor speed and memory recall. 3) I also hypothesized that ethnicity is a significant predictor of neurocognitive functioning. I believe that ethnic individuals are at increased risk for developing HIV-related neurocognitive impairment due to many psychosocial variables and socioeconomic conditions (e.g. trauma history and lower levels of education) that increase vulnerability. 4) Moreover, I hypothesized that increased symptom load, particularly due to general medical conditions, HIV-opportunistic infections, and increased number of falls, are positively associated with HIV-related neurocognitive impairment. 5) Lastly, I hypothesized that all of the predictor variables combined explain a significant proportion of the variance accounted for in HIV-related neurocognitive impairment (motor speed, psychomotor speed and memory recall).

CHAPTER 2

METHOD

Participants

Data were obtained from a larger study entitled Project Heart (Vosvick & Colleagues, 2009). I recruited 120 participants for the first phase of the study. From these, 71 participants who were categorized in the "high" self-reported HIV stigma group, based on a cutoff score of ≥ 99 on the HIV Stigma Scale (HSS), were asked to return for the second phase of the study. I used a sample of participants recruited from HIV/AIDS service providers in the Dallas/Fort Worth Metroplex area, such as the Resource Center in Dallas, AIDS Service of North Texas, and the AIDS Outreach Center in Fort Worth. Other recruitment venues included health centers and community-based support groups. Additionally, fliers were distributed at different HIV/AIDS community events and online advertisements were placed weekly on Craigslist, a free online medium for recruitment (see Appendix B & C). This study was approved by the University of North Texas' IRB committee (see Appendix G).

Only participants from Phase II of the study, who were categorized in the "high" HIV-related stigma group, were examined in my analyses. The gender-balanced sample of participants (male = 46.5% and female = 53.5%), ranged in age from 29 to 66 (M = 47.9; SD=9.0), with an average of 12.0 (SD = 2.6) years of education. Moreover, the sample was ethnically diverse (69.0% African-American, 23.9% European-American, 2.8% Latino (a), and 4.3% other). Of the participants surveyed, 29.7% were

unemployed14.1% self-reported they were engaged in a professional career and 14.1% reported they were homemakers. Furthermore, 81.3% reported their source of income as governmental assistance (SSI/SSDI/TANF/Food Stamps) and 59.4% reported a household income before taxes of less than $10,000, and 15.6% reporting earnings between $10,000 and $14,999 (see Appendix E). The majority of participants reported either Medicare (39.1%) or Medicaid (32.8%) as their primary source of health insurance. Similarly, participants reported they received care at county clinics (40.6%), HIV/AIDS service organizations (46.6%), community clinics (21.9%) and from family physicians (17.2%). HIV-related medical disorders were common with 29.7% of participants reporting hepatitis C, followed by 18.8% reporting pneumocystis pneumonia, 14.1% genital herpes, 12.5% tuberculosis, 12.5% syphilis, and 10.5% HIV-dementia complex. Furthermore, 42.2% reported hypertension, 23.4% high cholesterol, 20.3% arthritis, 20.3% seasonal allergies, 18.8% asthma, 18.8% chronic bronchitis, 12.5% anemia, 12.5% liver disease, and 10.9% type II diabetes.

Procedures

I conducted telephone screenings to determine eligibility for participation. Inclusion criteria included (1) an HIV-positive status (regardless of mode of transmission), (2) at least 18 years of age, (3) sufficient English language proficiency for reading and completion of surveys/assessments, (4) willingness to participate in multiple sessions, and (4) willingness to sign informed consent (see Appendix A). Exclusion criteria included (1) a diagnosed heart condition (2) cardiac arrhythmia (3) a pace maker

and (4) allergies to adhesives. These exclusionary criteria relate to aspects of the larger study that were not measured or included in this study.

Data collection occurred in two phases. In Phase I, I administered self-report questionnaires using a computerized survey protocol. I collected demographic, psychosocial, behavioral and medical data. Participants completed self-report measures independently, unless assistance was required. Upon request for assistance, trained researchers administered the survey in an interview format. Survey completion took approximately 1.5 hours, and participants received $10 as incentive to complete Phase I

During Phase II, I conducted psychophysiological assessments using a digital blood pressure monitor, a cold compress device and Infiniti biofeedback equipment. I conducted a series of visualization exercises and guided imagery. Following the psychophysiological assessment, I administered a brief cognitive measure and additional computer-delivered surveys. Participants were debriefed upon completion of this phase and received information about local mental health contacts (see Appendix F). Phase II took approximately 1.5 hours to complete and participants received $25 as incentive to complete Phase II.

To maintain confidentiality of the information provided, all data was secured in a locked office in the Center for Psychosocial Health Research. Once enrolled in the study, participants were assigned a unique participant code number that was used in all of the data collected. Participant codes were used to coordinate Phase I and Phase II data. No identifiable information was associated with a participant's code and only the principal investigator and senior researchers had access to these codes.

Measures

Health History Questionnaire (HHQ; Vosvick & Colleagues, 2009)

The HHQ includes a series of items on childhood illness, surgeries, known allergies, blood transfusions, and health behaviors (diet, exercise, caffeine, substance and alcohol use). Additionally, this questionnaire includes items on medical health history, mental health/psychiatric history, gender specific health illnesses, cardiovascular, pulmonary, renal/metabolic, neurological/nervous system, and cancerous diseases.

International HIV-Dementia Scale (IHDS; Sacktor, 2005)

The IHDS is a cognitive screening tool designed for international HIV populations. The IHDS consists of three subtests; a timed fingertapping, a timed alternating hand sequence, and a memory recall task. The timed fingertapping task requires the participant to open and close fingers on the non-dominant hand as widely as possible over a five second period. Normal performance on fingertapping task is ≥ 15 taps/5 seconds. The hand sequence tasks requires the participant to perform the following movements with the non-dominant hand as quickly as possible over a 10 second period: (1) clench the hand in a fist on a flat surface; (2) put the hand flat on the surface with the palm down; and (3) put the hand perpendicular to the flat surface on the side of the fifth digit. The number of sequences correctly performed within 10 seconds, up to a maximum number of 4 sequences, is scored. A participant unable to perform the alternating hand sequence is assigned a score of zero. Memory recall is measured by reciting four words to the participant and then asking him/her to repeat them immediately. The words are

repeated by the examiner until the participant can repeat all four words correctly. The participant is then asked to recall the four words after the timed fingertapping and alternating hand sequence tests. The number of items recalled is scored out of 4. If the words are not recalled, the participant is prompted with a semantic and a half-point is assigned for each correct word recalled after prompted. The maximum possible score is 12 points; thus, a score of ≤ 10 is evidence of probable dementia. This cut-off score has been recommended to minimize false positives errors (Sacktor, 2005). Therefore, I dichotomized HIV dementia scores so that participants, who scored ≥ 11 or more, received a zero (0), and participants who scored ≤ 10 received a one (1). Furthermore, the IHDS has a positive predictive value of 91% and a negative predictive value of 93% in detecting HAND. Using a cut-off of ≤ 10, the sensitivity and specificity for HIV dementia with the IHDS were 80% and 57% respectively in a US sample, and 80% and 55% respectively in a Ugandan sample. Also, the IHDS is useful for HIV-positive individuals with or without a high school education (Sacktor, 2005).

HIV Stigma Scale (HSS; Berger, Ferrans & Lashley, 2001)

The HSS assesses perceived stigma in people living with HIV. The measure consists of 40 likert-type items with potential responses ranging from 1 *(strongly disagree)* to 4 *(strongly agree)*. The HSS consists of four subscales including personalized stigma, disclosure concerns, negative self-image, and concern with public attitudes towards people living with HIV/AIDS. Example items include "Telling someone I have HIV is risky," "Since learning I have HIV, I feel set apart and isolated

from the rest of the world," and "Since learning I have HIV, I worry about people discriminating against me." I used a cutoff score of ≥ 99 on the HSS to distinguish between "high" vs. "low" HIV-related stigma. The HSS has high internal consistency ranging between $r = .90$ to $r = .93$, for the different subscales, and .96 for the overall instrument. Furthermore, the HSS demonstrates construct validity and is associated with other measures of self-esteem, depression, social support and social conflict. The HSS has high external validity for stigma for ethnic minority individuals living with HIV/AIDS (Berger, Ferrens, & Lashley, 2001).

Trauma History Questionnaire (THQ; Green, 1996)

The THQ is a 24-item self-report measure that examines serious or traumatic life events, such as crime (e.g. robbery and mugging), general disaster (e.g. injury and witnessing death), sexual and physical abuse. Example questions include, "Has anyone ever tried to take something from you by using force or the threat of force, such as a stick-up or mugging?" and "Has anyone ever made you have intercourse, oral or anal sex against your will? If yes, please indicate nature of relationship with person (e.g., stranger, friend, relative, parent, and/or sibling)." Items on the THQ are summed to yield subscale scores as well as an overall summed score, with higher scores reflecting more self-reported trauma.

The THQ has high test-rest reliability, ranging from $r = .60$ to $r = 1.00$, depending on the subscale and a reported Cronbach's alpha for the entire instrument was .85 (Green, 1996). The THQ has been validated on different clinical and nonclinical samples, such as

holocaust survivors, cancer patients, and those with severe mental illnesses and non-epileptic crises.

Data Analyses

Data were entered into the Questionnaire Development System (QDS), a computer-based survey program that saves all participant data, exports data into SPSS and other statistical software, and merges participant data from different collection points. Assumptions of normality were tested using the Shapiro-Wilk test, a powerful normality test that is able to detect small departures from normality. I used the Levene's test to test assumptions of equal variances for variables of interest. Though not needed for my analysis, I intended on remedying violations of assumptions by transforming data with either log or the Box-Cox power transformations.

I interpolated missing data if a participant answered at least 80 percent of the items for any particular scale/subscale. In these cases the individual's mean of that subscale was substituted for the missing responses. I examined participants with missing data to determine if missing data were due to random or systematic error. Chi square-tests for categorical variables and *t*-tests for continuous variables were conducted to determine if any differences exist between the sample of participants used in the analysis and those excluded. A power analysis was conducted for the logistic regression model to determine the sample required to achieve 80% power at a 0.05 significance level, in a design that uses a binary response variable on continuous predictor variables. I used G*Power (a program for calculating power; Faul & Erdfelder, 1992) to conduct this analysis and

found a sample of 56 participants was required for this study. Moreover, I adhered to Cohen's article (1988), such that a medium effect size ranges between .18 and .48.

Univariate statistics were calculated for all variables to describe the sample. Univariate statistics included means, standard deviations, actual and possible ranges, and percentages when applicable. I computed Cronbach alpha statistics as a measure of internal consistency for the psychometric instruments used in the analyses, such as the HSS, IHDS, and THQ.

Bivariate analyses were conducted for all predictor and dependent variables to identify significant correlations. I conducted chi square-tests and *t*-tests to determine if gender and ethnic differences existed on variables such as, stigma, trauma, general medical conditions, HIV-opportunistic infections, falls, and HIV-related neurocognitive impairment.

A binary logistic regression analysis was conducted on the categorical dependent variable, HIV-related neurocognitive impairment. Predictor variables included trauma, stigma, ethnicity, falls, general medical conditions and HIV-opportunistic infections. I used a 95% confidence interval and $p < .05$, to determine significant odds ratios (Exp (B)); the odds of a participants with or without HIV-related neurocognitive impairment. I used the Hosmer-Lemeshow goodness of fit to test the null hypothesis, hypothesizing that a linear relationship existed between the predictor variables and the logit of the criterion variable. The Hosmer-Lemeshow goodness of fit test computed a chi square statistic. A nonsignificant chi-square statistic ($p > .05$) indicated that the data fits my model. I also computed a Nagelkerke R^2 value to determine goodness of fit. Additionally,

multicollinearity diagnostics were conducted by examining tolerance and variance inflation factor (VIF) scores.

Lastly, I conducted an exploratory analysis to determine if differences existed between participants included (Phase II) vs. those excluded (Phase I). I conducted chi square-tests for categorical variables and *t*-tests for continuous variables to determine if any differences exist between the sample of participants used in the analysis (Phase II) and those excluded (Phase I).

CHAPTER 3

RESULTS

Missing Data

Of the 120 participants who completed the survey, 71 participants who scored "high" on HIV-related stigmatizing events, returned for the second phase of the study. Missing data were interpolated using subscale means if participants answered at least 80 percent of the items on that particular subscale. Due to missing data, one participant (n = 1) was eliminated from the analysis rendering a sample size of 70 participants (n = 70).

Univariate and Bivariate Analyses

Table 2 presents means, standard deviations, ranges, and internal-consistency reliabilities for variables of interest. An independent samples t-test revealed that African-American participants reported more overall trauma on the Trauma History Questionnaire (THQ) compared to other ethnic groups (t (2, 68) =.93, p = .02), particularly on self-reported physical and sexual violence, crime and natural disaster. Chi-square analyses revealed that African-Americans were more likely to exhibit neurocognitive impairment based on the International HIV-Dementia Scale (IHDS; χ^2 (1, n = 70) = 6.43, p = .01), and European-Americans were least likely to exhibit neurocognitive impairment (χ^2 (1, n = 70) = 6.79, p < .01).

I used Pearson product moment correlation coefficients to determine significant relationships between all variables in my model (Table 4). In this study, ethnicity was

negatively associated with HIV-related stigma (r = -.34, p < .01). Furthermore, ethnicity and number of lifetime falls, were positively associated with neurocognitive impairment (r = .31, p < .01; r = .32, p < .01), and years of education was negatively associated with neurocognitive impairment (r = -.27, p < .05). Moreover, HIV-related conditions, general medical conditions and education, were positively associated with trauma (r = .35, p < .01; r = .30, p < .05; r = .27, p < .05). Interestingly, gender (female) was negatively associated with education (r = -.37, p < .01) and positively associated with general medical conditions (r = .34, p < .01).

Multivariate Analyses

I conducted a binary logistic regression analysis to test variance in cognitive impairment based on overall scores on the International HIV-Dementia Scale (IHDS). Consistent with acceptable cutoff scores proposed by Sacktor et al. (2007), my dependent variable was dichotomized so that participants who scored ≤ 10 received a 1 (impaired), and participants who scored ≥ 11 or more, received a 0 (not impaired).

The regression model used to predict HIV-related neurocognitive impairment, was statistically significant (χ^2 (8, n = 70) = 6.54, p = .59). My model explained 36.8% (Nagelkerke R^2) of the variance in the outcome variable. I found that self-reported trauma was a significant predictor of HIV-related neurocognitive impairment; however these variables were inversely related. Participants with more lifetime trauma scored better on the on the IHDS (B = -.15, OR = .87; CI 95% = .75, 1.0, p = .05). Ethnicity was a significant predictor of HIV-related neurocognitive impairment, such that ethnic

participants scored worse on the IHDS than European-Americans (B = 2.2, OR = 9.0; CI 95% = 1.68, 48.48, p = .01). Furthermore, as participants reported more general medical conditions they also exhibited more HIV-related neurocognitive impairment (B = .30, OR = 1.34; CI 95% = 1.0, 1.81, p = .05), and participants who experienced more falls in their lifetime also experienced more HIV-related neurocognitive impairment (B = 2.0, OR = 7.2; CI 95% = 1.1, 47.0, p = .04). Please see Table 5 for full results.

Exploratory Analyses

In Phase II, researchers administered the IHDS, the outcome variable of interest. Participants from Phase I who did not return (due to low scores on the HIV Stigma Scale; HSS) were excluded from my analyses. Chi-square analyses and t-tests reveal that certain socio-demographic variables, such as ethnicity, age, gender, education, current employment, occupation, household income, household living arrangement, and type of health insurance, did not significantly differ between participants that were included vs. those excluded from the study (i.e. those in Phase I vs. Phase II). Furthermore, I found no significant differences in the type of HIV-related healthcare provider/facility that participants use for treatment (e.g. family physicians, free clinic, AIDS service organizations, community clinic, etc.).

I found no significant differences in HIV status (diagnosed with HIV vs. AIDS) and recent CD4-cell count; however, I found significant differences in recent viral load (t (2, 118) = .49, p < .01), between those included in Phase II (M = 484.17, SD = 1871.63) vs. those excluded from Phase I (M = 746.30, SD = 3676.84). Furthermore, I found

differences in self-reported HIV-related conditions, such as hepatitis B (χ^2 (1, n = 70) = 4.25, p =.04) and human papilloma virus (HPV; χ^2 (1, n = 70) = 5.10, p =.02, and general medical conditions including hypertension (χ^2 (1, n = 70) = 4.25, p = .04). Of those who were excluded from the study, 68.8% self-reported hepatitis B (in comparison to 31.3% that were included), 72.7% HPV (in comparison to 27.3%), and 28.9% hypertension (in comparison to 71.1%). Furthermore, I found no significant differences in the number of self-reported falls participants experienced.

I found no differences on responses on the Trauma History Questionnaire (THQ); however, as expected, I did find differences on the HSS (t (2, 118) = -1.02, p < .01), between participants included and excluded in the study. Included participants scored higher on the HSS; thus, were asked to come back and participate in Phase II of the study.

CHAPTER 4

DISCUSSION

Stigma

Contrary to my hypothesis, stigma was not significantly associated with HIV-related neurocognitive impairment. HIV-related stigma is seen as the devaluation, oppression and social inequality of people living with HIV/AIDS (Hamra, Ross, Karuri, Orrs, & D'Agostino, 2005). Cognitive appraisals predict the stress and behavioral response associated with stigma. When faced with threat, an individual undergoes a specific cognitive process of appraisal (Dickerson & Kemeny, 2004). Based on the appraisal, the individual must determine if he/she has the resources necessary to meet the demands of the situation and overcome the threat. Thus, stigma may be more related to locus of control and self-efficacy that allow the individual to move past the negative event (Calhoun, Cann, Tedeschi, & McMillan, 2000; Pat-Horenczyk & Brom, 2007). Furthermore, social support may be a mediating variable. Social support facilitates coping with HIV (McDowell & Serovich, 2007), and acts as a buffer to stress-related diseases (Johnson et al., 2001), disease progression (Ross et al., 2005), and enhances neuropsychological wellbeing (Serovich, Kimberly, Mosack, & Lewis, 2001). Similarly, AIDS service organizations and community based organizations that provide free or low costing resources provide additional support to people living with HIV/AIDS. These organizations supply comprehensive case management, support services, psychoeducation, and financial assistance (Rhodes, 2004).

Alternatively, I found ethnicity was negatively correlated with HIV-related stigma ($r = .36$, $p < .01$). Though, previous studies report mixed results (Foster & Gaskins, 2009), my findings may reveal ethnic differences in labeling sexual orientation and HIV disclosure. European-American men, who have sex with men (MSM), are more likely to identify as homosexual, in comparison to Latino and African-American MSM who more likely self- identify as bisexual or heterosexual (Agronick et al., 2004). In traditional African-American and Latino (a) communities, masculinity and procreation are necessary components of male identity. Consequently, sexual orientation and HIV disclosure may be limited due to fear of negative social reactions (Harawa et al., 2004), and stigma and shame associated with homosexuality in ethnic communities (Lichenstein, 2000). To avoid or lessen the possibility of stigma, individuals may conceal sexual orientation to ‘‘pass’’ as normal (Galvan, Davis, Banks & Bing, 2008). Herek and Capitanio (1999) conducted a survey to examine the effects of layered stigma that exist in HIV/AIDS. Researchers analyzed variables, such as race, sexual orientation, route of transmission and gender, and found that homosexuality was the most negative attitude/belief held towards people living with HIV/AIDS. Thus, European-Americans who self-identify as homosexual may experience more stigma than those who do not. Moreover, social support, more often reported in ethnic minority individuals, may be a buffer to HIV-related stigma. In Heckman et al. (2000), HIV-positive African-Americans reported more social support from family and AIDS service organizations, than European-Americans. African-Americans also reported increased spirituality and religious affiliation, and

described their beliefs as a form of coping that allowed them to find purpose and meaning despite stigma (Vyavaharkar et al., 2007).

Trauma

In this study I found that trauma was positively related to neurocognitive impairment. The more self-reported trauma the higher participants scored on the International HIV-Dementia Scale (IHDS). My finding is inconsistent with previous findings that trauma affects one's ability to attend to information and concentrate (Dyregrov, 2004), and reduces test performance (Streeck-Fischer & van der Kolk, 2000). In children, trauma is linked to poor academic achievement, limited self-regulation, depression, and defiant or oppositional behavior (Hart, 2009). In adulthood, trauma can alter information processing, and the intrusive thoughts associated with trauma interfere with memory, attention and concentration (Dyregrov, 2004). However, in line with the health belief model (HBM), after a traumatic event, some adapt and move forward if they possess resilience (Alim, Feder & Graves, 2008; Collishaw, Pickles & Messer, 2007). Resilience occurs when one adjusts to trauma despite adverse consequences (Yehuda, Flory, Southwick & Charney, 2006). Resilient individuals experience more positive affect and optimism (Boardman, Blalock & Button, 2008), use more active coping strategies, are more socially competent, use social support networks, and are less likely to experience post traumatic stress disorder (PTSD) following the trauma (Yi, Smith & Vitaliano, 2005). Resilience is also associated with increased white matter and myelination of the prefrontral cortex and synaptic proliferation (Barnea-Goraly, Menan &

Eckert, 2005; Katz, Liu & Schaer, 2009). Resilience is considered "bouncing back" to psychological functioning that existed prior to the traumatic event; however, some individuals may "bounce forward" in response to trauma (Walsh, 2002). Post traumatic growth (PTG) is a term coined to describe positive adaptation to life circumstances after trauma (Calhoun, Cann, Tedeschi, & McMillan, 2000). PTG is also associated with meaning making, such that after trauma one may find meaning or purpose in the event that helps to recover and move forward. Furthermore, PTG leads to less guilt or shame associated with trauma and higher self-efficacy (Pat-Horenczyk, R., & Brom, D. (2007). Thus, resilience and PTG may mediate the relationship between trauma and cognitive impairment (Pat-Horenczyk & Brom, 2007).

Furthermore, I found that with more years of education, participants also reported more trauma. Though this finding is inconsistent with the existing literature (trauma is linked to fewer years of education; Ullman & Brecklin, 2002), perhaps my finding is more related to the disclosure of trauma rather than actual trauma. Negative social reactions and stigma associated with disclosing trauma are more common in disenfranchised and urban communities (Lott, 2002). Oppression and shame create more self-blame and external locus of control, which deter the victim from labeling the incident as abuse/rape and reporting the event (Frazier & Seales, 1997; Ullman & Brecklin, 2002). Furthermore, education, a significant component of socioeconomic status (SES), may lead to increased reports of trauma because individuals with more education have greater access to support networks (Brewin, Andrews, & Valentine, 2000), hospital and emergency care, and psychological services. In Leserman et al. (2005), 611 HIV-positive

adults, with an average of 12 years of education (80%), who also experienced lifetime trauma, were more likely to seek overnight hospital services, emergency room visits, and on average four HIV-related outpatient visits within the past 9 months. Moreover, after a traumatic event, individuals who experience PTSD show disrupted cognitive processing and dissociation, and become increasingly confused and disorientated (Ehlers & Clark, 2000). For example, following trauma, recollection about characteristics of the perpetrator and surrounding environment are then organized into an autobiographical narrative. Unfortunately, individuals who experience trauma may dissociate and are unable to recollect details of the event. Thus, reporting trauma may be more closely related to whether or not the individual experiences PTSD and dissociation. Premorbid cognition is a predisposing factor in the development of trauma-related symptomatology (Kessler et al., 1995). In Macklin et al. (1998) veterans were assessed pre and post deployment and found that those who developed PTSD as a result of trauma, post combat, had lower intelligence quotients (IQ) and cognitive functioning at baseline. Similarly, veterans who developed PTSD had lower performances on the Wechler Memory Scale-Revised (WMS-R; $M = 96.1$), in comparison to veterans who did not develop PTSD ($M = 113.5$; Gilbertson et al., 2001). Therefore, higher IQ at baseline may in fact be a protective factor for the development of trauma-related symptomatology (De Bellis et al., 1999). In sum, individuals with higher education may be more likely to report a traumatic event, than those with less education who dissociate and use avoidance coping to manage the traumatic event.

In this study, African-American participants experienced more trauma than other

ethnic participants. Consistent with other studies, my findings suggest that African-Americans, particularly those of lower socioeconomic status, are at higher risk of increased number of lifetime traumatic events (Davis, Ressler, Schwartz, Stephens & Bradley, 2008; Liebschutz et al., 2007). Alim et al. (2006), found that out of 617 African-American primary-care patients, 65% experienced trauma and 33% received a diagnosis of PTSD in their lifetime (Alim et al., 2006). African-Americans also have a higher incidence of physical abuse in childhood (Kalof, 2000) and sexual abuse in adulthood (Ullman & Filipas, 2005), than European-Americans. Trauma in the African-American community is transmitted from one generation to another. Intergenerational trauma is a product of historical oppression and discrimination from the time of slavery. Consequently, post traumatic slave syndrome (PTSS) is used to describe the presence of lower self-esteem, sensitivity to disrespect, and "shame proness" often seen in the African-American community (Gump, 2010; Leary, 2005). Ethnic differences in trauma may also be related to control-related beliefs, such that African-Americans have more of an external locus of control than European-Americans (Shaw & Krause, 2001). Control-related beliefs may dictate the severity of traumatic experiences. Higher rates of trauma in the African-American community are attributed to heightened media attention and increased likelihood of self-reports. Unfortunately, though African-Americans self-report more trauma than other ethnic minority groups, they seek less mental health services (Snowden & Pingitore, 2002). According to Hines-Martin, Malone, Kim and Brown-Piper, (2003), African-Americans are more likely to seek emergency care after trauma, but less likely to seek therapy due to the perceived stigma, lack of education and

resources, and the cultural shame associated with physical and sexual abuse.

Furthermore, the more participants reported HIV-opportunistic infections and general medical conditions, the more likely they experienced a traumatic event in their lifetime. Psychological trauma, associated with physical and sexual abuse, is common in people living with HIV/AIDS (Mugavero et al., 2007). Furthermore, trauma is linked to increased number of opportunistic infections, and the additive effect of multiple traumas increases the odds of negative health outcomes in HIV (Mugavero et al., 2007). For example, in Kimerling et al. (1999), 88 HIV-positive African-American females, who experienced 3 or more traumatic events, had a higher incidence of HIV-related medical conditions, than did participants with two or less traumatic experiences. Trauma is also linked to risky sexual behaviors (Bensley, Van Eenwyk, & Simmons, 2000; Wu, El-Bassel, Witte, Gilbert & Chang, 2003), and maladaptive health behaviors, such as smoking, unhealthy diet and physical inactivity, in people living with HIV/AIDS (Felitti et al., 1998). Moreover, trauma is associated with unprotected sex, anonymous sex, sex with multiple partners, prostitution (Johnsen & Harlow, 1996; Kalichman, Sikkema, DiFonzo, Luke & Austin, 2002), and intravenous drug use (Lodico & DiClemente, 1994). Thus, engaging in such maladaptive behaviors can also result in sexually transmitted diseases and HIV co-occurring infections such as hepatitis C (Rotheram-Borus, Murphy, Reid & Coleman, 1996), and reinfection with different strains or drug resistant strains of HIV (Halkitis, 1998). Increased lifetime trauma is also associated with nonadherence to highly active antiretroviral therapies (HAART). In Mugavero et al., (2006), nonadherence to HAART was found in 9.5% of participants without a history of trauma, in comparison

to 22.4% who experienced three traumatic events and 34% who experienced more than five traumatic events in their lifetime. A history of trauma and HIV/AIDS is associated with less self-care, less medical consultation (Cohen, Brom & Dasberg, 2001), increased somatization and negative health perceptions (Miranda, Meyerson, Marx and Tucker, 2000). Similarly, in Smith, Egert, Winker and Jacobson (2002), HIV-positive participants, who also experienced trauma, self-reported more pain and worse perceptions of health status. Trauma-related symptomatology is associated with heart disease, stroke, cancer, emphysema, hepatitis, diabetes, arthritis, and irritable bowel syndrome (Felitti et al., 1998; Golding, 1994; Leserman et al., 1996; Schnurr, Sprio & Paris, 2000).

Ethnicity

In this study I found ethnic minority differences on the IHDS (χ^2 (1, n = 70) = 6.79, p = .01), such that of the 45% of participants who scored in the impaired range, 84% were African-American, and African-Americans also scored in the most impaired range on the IHDS (χ^2 (1, n = 70) = 6.43, p = .01). Statistically, African-Americans are disproportionately more affected by HIV than any other ethnic groups (CDC, 2009) and HIV can lead to several forms of HIV-associated neurocognitive disorders (HAND), particularly mild neurocognitive impairment (MNI) and minor cognitive motor disorder (MCMD). African-Americans are twice as likely to develop Alzheimer's disease and other dementias, than European-Americans (Potter et al., 2009; Tang et al., 2001). Similarly, Lopez et al. (2003) found that African-Americans were 4.4 times at greater risk of mild cognitive impairment than other ethnic minority groups. Though, these higher

prevalence rates are often associated with Alzheimer's disease and vascular dementia, less is known about ethnic difference with other dementias and neurocognitive disorders. The increased incidence and prevalence of dementia and cognitive impairment seen in African-Americans is often explained by education, SES, genetic predisposition and measurement bias in neuropsychological assessments (Fitzpatrick et al., 2004; Plassman et al., 2007). African-American performance on neuropsychological tests of reading (Boekamp, Strauss & Adams, 1995), naming (Lichtenberg, Ross & Christensen, 1994), and nonverbal abilities (Campbell et al., 1996), is consistently lower than European-Americans' performance (Manly, Jacobs, Touraji, Small & Stern, 2002).

Symptom Load

General Medical Conditions

I found that as participants self-reported more general medical conditions, they experienced more neurocognitive impairment. The cumulative effect of numerous medical conditions significantly predicted lower scores on the IHDS. People living with HIV/AIDS are disproportionately at higher risk of cardiovascular risk factors such as diabetes, hypertension and obesity (Adeyemi, 2007; Hsue et al., 2004). For example, Triant, Lee, Hadigan and Grinspoon (2007), found that 11.3% of participants with HIV experienced myocardial infarction (MI), in comparison to 6.98% of seronegative patients. Furthermore, Magalhaes, Greenberg, Hansen, Odont and Glick (2007) found increased hypertension and Hsue et al. (2004) found higher rates of coronary artery disease in HIV-positive participants than negative controls. HIV directly exacerbates medical conditions,

such as hypertension (Magalhaes et al., 2007), atherosclerosis (Lorenz et al., 2008), and diabetes (Triant et al., 2007). Research substantiates the association between hypertension and cognitive disorders (Kivipelto, Helkala & Laakso, 2002; Schmidt, Schmidt & Fazekas, 2000). Hypertension causes an increase in neurofibrilliary tangles and amyloid plaques commonly seen in individuals with Alzheimer's disease and vascular dementia (Jellinger, 2002). As such, antihypertensive treatments are deemed successful at decreasing the risk for dementing disorders (Qiu, Strauss & Fastbom, 2003). In the Systolic Hypertension Europe trial (Syst-Eur), antihypertensives reduced the risk of Alzheimer's disease and vascular dementia by approximately 50% (Forette, Seux & Staessen, 1998).

Metabolic syndrome is also linked to dementia (Muller, Tang & Schupf, 2007; Yaffe, 2007). Metabolic syndrome, mainly heightened glucose levels, and high (> 29) and low (< 21) BMIs, are associated with cognitive decline (Berg, Biessels & Craen, 2007; Whitmer, Gunderson & Barratt-Connor, 2005). Thus, vascular risk factors contribute to cognitive changes in the brain (Erkinjuntti, 2005; Gregg et al., 2000; Gunstad et al., 2007), particularly deficits in executive function, processing speed, motor function, and memory (Sacktor et al., 2007). The link between cardiovascular disease and HIV may be related to pharmacotherapy. HAART is thought to cause lipid abnormalities and metabolic changes that may lead to atherosclerosis and cardiovascular disease (Bergersen, 2006; Mary-Krause et al., 2003). In the D:A:D study (2003), participants on HAART experienced a 23% increase in myocardial infarction, and participants on protease inhibitors were twice as likely to experience myocardial infarction, in

comparison to participants not taking those drugs. Moreover, due to increased longevity for individuals on HAART, age is also a risk factor for cardiovascular disease in HIV (Bergersen, 2006; Mary-Krause et al., 2003). Mary-Kause et al. (2003) found a 43% increased risk of myocardial infarction for every 10 year increase in age. Consequently, older adults or those with numerous medical conditions are at greater risk for cognitive dysfunction (Uchiyama, Mitrushina, Satz, & Schall, 1996). Backman et al. (2004) found that diabetes mellitus and vascular risks were linked to aphasia and impaired verbal fluency, and Brady et al. (2001) found similar findings even in younger HIV-positive participants. In my sample, 39% of participants were age 50 and older, and these participants were more likely to experience hypertension (χ^2 (1, n = 70) = 18.85, $p < .01$), and hypercholesterolemia (χ^2 (1, n = 70) = 7.10, $p = .01$; Table 3).

HIV-Related Opportunistic Infections

Contrary to my initial hypothesis, HIV-related conditions were not significantly related to neurocognitive impairment. With the progression of HIV, opportunistic infections are common in the central nervous system (CNS; Mamidi, DeSimone, & Pomerantz 2002), mainly when CD4- cells drop ($<200mm^3$)[1]. Bacteria and viruses such as cryptococcus meningitis, cytomegalovirus (CMV), herpes simplex virus, toxoplasmosis, human papilloma virus (HPV), and Epstein Barr, are typically diagnosed in people living with HIV/AIDS (Anthony & Bell, 2008). The abovementioned opportunistic infections are responsible for demyelination of axons in the CNS and

[1] In our study this is particularly true for tuberculosis (r = -.24, $p < .05$) and human papilloma virus (r = -.27, $p < .05$).

neuronal loss. Consequently, subcortical dementia affects 10–20% of people living with HIV/AIDS (Anthony & Bell, 2008). However, drug cocktails or combined antiretroviral therapies are effective at suppressing opportunistic infections that attack the immune system and brain. Since the advent of HAART, CNS opportunistic infections have decreased substantially (Anthony & Bell, 2005; Gray & Keohane, 2003; Langford, Letendre, Larrea & Masliah, 2003; Maschke et al., 2000; Masliah, De Teresa, Mallory, & Hansen, 2000). Thus, participants adherent to medications may suppress opportunistic infections, resulting in fewer neurocognitive changes.

Falls

I found that participants who self-reported more falls also experienced more neurocognitive impairment. Individuals who fall are more susceptible to a host of other medical conditions including cognitive impairment, disorientation and deteriorated physical and mental health (Endres et al., 2006; Kallin, Jenson, Olsson, Nyberg & Gustafson, 2004). Falls are not only associated with the onset of cognitive dysfunction, but are also a consequence of changes in neurological functioning (Kallin et al., 2004). Increased falling is associated with impaired orientation and judgment, gait, and visuospatial processing (Van Doorn, et al., 2003). Falls are particularly associated with brain-related injuries, which exacerbate dementia risk (Kallin et al., 2004).

Education

I found that education is significantly related to neurocognitive impairment.

Participants with less formal education experienced more cognitive dysfunction. Education is a protective resource against dementia, because more education is thought to stimulate dendritic growth and number of synapses that stall neuropathological changes (Shadlen et al., 2006; Stern, Albert & Tang, 1999). Formal education helps with performance on neuropsychological and cognitive measures, because individuals are more familiar with testing demands and utilize advanced test-taking skills that are taught in school. In a study conducted by Mehta, Simonsick and Rooks (2004), researchers found that education, literacy and income accounted for 86% of the variance in mental status scores between participants. Conversely, according to Lopez, Wess, Sanchez, Dew and Becker (2007), the association between dementia and education may be more complex than once assumed. Researchers believe that higher education is protective against dementia only if one pursues active or intellectual careers. Individuals with high education but less challenging careers may be at similar risk for dementia as individuals with less education, based on the idea if you don't use it you lose it (McDowell & Serovich, 2007).

Gender

My findings reveal that female participants had fewer years of formal education and self-reported more general medical conditions than men. Though, little research has examined biomedical and psychological gender differences in HIV/AIDS (Faílde-Garrido, Alvarez & Simón-López, 2008), women living with HIV come from lower socioeconomic backgrounds, are more frequently diagnosed with alcohol and substance

abuse disorders, experience higher mortality rates, and report lower levels of educational attainment (Faílde-Garrido, Alvarez & Simón-López, 2008). Along these lines, HIV-positive women also self-report worse health status, more psychiatric disturbances, and more utilization of healthcare services than men (Leserman et al., 2005). Moore, Mocroft and Madge (2001) found that women living with HIV/AIDS had faster immunological compromise and experienced more side effects than men. Furthermore, female participants in Weiss et al. (2010) had more HIV-related medical comorbidities than men, perhaps due to the tendency for women to wait until they are more immune compromised to seek emergency care. For example, Hendricks and Gorbach, (2009) found that in a sample of 400 ill HIV-positive adults in New York City, women were less likely to use AIDS service organizations and primary-care facilities and were more likely to use the emergency room than men. Furthermore, other studies found that HIV-positive have less access to HAART than men (Dray-Spira & Lert, 2003). In a study conducted at a John's Hopkins HIV Clinic, researchers found that only 36% of HIV-positive female patients sought gynecological care and only 55% general medical care (Tello et al., 2008). In this study, Tello et al. (2008) found that female participants reported weather, discomfort/pain, forgetfulness and fear of a bad diagnosis as the primary reasons for missing or not scheduling medical appointments. Moreover, low self-esteem, and denial were reported barriers to accessing healthcare in female participants (Andrasik, Rose, Pereira & Antoni, 2008). Waiting to seek services and less access to medical care may be related to lower education, substance abuse, and lack of knowledge of resources and AIDS service organizations available.

Exploratory Analyses

In my study, I found significant differences between participants who were included (Phase II) vs. excluded (Phase I) from the analyses. Excluded participants had higher viral loads, evidence of greater disease progression, than those included. Participants who were excluded from the analysis also self-reported "low" HIV-related stigma. In line with the HBM (Rosenstock, 1974), participants with high stigma, discrimination and oppression, but also high internal locus of control and self-efficacy (Bandura, 1977), may use their negative experiences as motivation to overcome barriers interfering with medical and psychological health. Thus, with social support and optimistic cognitive appraisals (Stiles & Kaplan, 2004), participants who experience more stigma may in turn become more proactive about health, resulting in better health outcomes. Similarly, included participants had less HIV-related co-infections and sexually transmitted diseases, particularly hepatitis B and HPV, which is consistent with my finding on viral load. The HBM suggests that self-efficacy and internal locus of control also dictates condom use and condom negotiation (Rosenstock et al., 1994). Included participants, who experienced more stigma, may also be hypervigilant and cautious during sex. However, increased awareness of the environment or hyperarousal may explain why included participants self-reported more hypertension, than those excluded. Accordingly, individuals who experience chronic stress (i.e. depression and anxiety) are susceptible to chronic diseases, such as hypertension, and cardiovascular disease (Taylor, Washington, Artinian & Lichtenberg, 2008).

CHAPTER 5

LIMITATIONS

Despite multiple strengths, my study also had limitations. I obtained the majority of the data through self-report measures, and as a result the data may be subject to inaccurate recall or concealment of important information. Furthermore, I recruited participants from one geographic region; thus, generalizability is limited. Since my sample was recruited exclusively from AIDS service organizations, my findings may not generalize to other people living with HIV/AIDS that are not treated in community clinics, or those who receive private healthcare services.

The International HIV-Dementia Scale (IHDS) neurocognitive screener used in this study may not fully account for differences in educational quality and culture. These differences can potentially result in misdiagnoses in ethnic minority participants and participants of lower socioeconomic backgrounds (Manly, 2006). On the other hand, using the current cut off score of < 10 may yield false negatives (Carey et al., 2004; Smith et al., 2003) and milder forms of HIV-related neurocognitive impairment may be overlooked and undiagnosed. Impaired scores on the IHDS must be interpreted with caution as they do not necessarily indicate the presence of a dementing disorder. Ideally, additional measures should be used as a diagnostic screening tool; however, to date, no other reliable and valid measure is widely accepted in the HIV/AIDS literature. Also, it is difficult to determine if the neurocognitive impairment seen in my participants are attributed to an HIV/AIDS diagnosis or due to the overlap with negative side-effects of

HAART, or the comorbidity of other medical conditions associated with HIV, such as hypertension and diabetes (Stoff, 2004). Furthermore, this study employed a cross-sectional, correlational design, which prevents me from inferring causality.

CHAPTER 6

CLINICAL IMPLICATIONS

People living with HIV/AIDS require a plethora of healthcare services. According to the HIV research network, most patients seek at least 10.7 outpatient visits annually (Bodenlos, et al., 2004). As hypothesized by the health belief model (HBM), stigma, trauma and negative affect create barriers for treatment. However, self-efficacy, a component of the HBM (Bandura, 1977), suggests that the individual will be proactive about health if they believe they can overcome obstacles and have resources necessary to comply with treatment. Similarly, the HBM proposes that individuals will adhere to medications if they believe they can do so effectively. Clinicians should emphasize the importance of adhering to highly active antiretroviral therapies (HAART), but also stress the impact of physical inactivity, poor nutrition, and cigarette smoke on disease progression. Clinicians should provide psychoeducation to increase adaptive health behaviors, and utilize motivational interviewing so that patients believe they can adhere at home. The mutual collaboration between the patient and healthcare provider is correlated with medication adherence (Miller, Murphy, Clark, Hamburger & Moore, 2004), appointment attendance (Volpicelli, Pettinati, Monterosso, & Lipkin, 2003), following medical advice (Zrinyi & Horvath, 2003), diet, exercise, and other health behaviors (Bodenlos et al., 2004). Therefore, consistent with the HBM, people living with HIV/AIDS with higher self-efficacy have greater health outcomes; thus less susceptible to HIV-related neurocognitive impairment.

Unfortunately, some patients are viewed as more deserving of treatment than others. Surprisingly, HIV-related stigma and ageism in HIV is not only held by the public, but by healthcare providers as well (Kahana & Kahana, 2001). Clinicians must facilitate rapport, and provide empathy and unconditional regard to increase compliance and therapeutic gain. Clinicians in both medical and psychological settings should focus on creating a reciprocal environment where patients are involved in setting goals and finding ways to realize such goals (Sprouse et al., 2005).

Although, drug cocktails were developed to increase survival and quality of life; the impact of HAART on cognition is two-fold. While HIV-positive individuals who adhere to medications are less likely to experience co-occurring infections, they are also more susceptible to increased body mass index, hypertension and hypercholesterolemia (Bergersen, 2006). Likewise, HIV is directly linked to vascular risk factors; such as enhanced c-reactive protein (CRP), an immunological cardiovascular biomarker. Thus, it is nearly impossible to tease out vascular risks while forming an HIV-associated neurocognitive disorder (HAND) diagnosis. Clinicians must adopt a systems approach to evaluate patients and recognize competing factors that contribute to neurocognitive impairment. According to a proposal submitted by Jeste et al. (2010), the *Diagnostic and Statistical Manual-5 (DSM-V)* will reflect changes to the current classification of dementia due to HIV. A new diagnosis, minor neurocognitive disorder (MND), will be added to classify individuals who experience mild cognitive deficits, but can function independently (Woods, Moore, Weber & Grant, 2009). Apart from the addition of MND, this study reveals that clinicians should also examine the need for a separate diagnosis to

reflect HIV-related neurocognitive impairment associated with vascular risk factors. Currently, cardiovascular disease is exclusionary from a diagnosis of HAND; however, due to the effects of HIV and HAART on the immune system and the heart, people living with HIV/AIDS are inevitably plagued by multiple vascular risk factors. The clinical health and neuropsychology fields need a more organized and delineated classification system for diagnosing individuals with HIV-related neurocognitive impairment with comorbid medical and psychiatric conditions.

People living with HIV/AIDS may also confront a variety of psychosocial stressors. Affective and cognitive changes in mood are linked to opportunistic infections, medical conditions and medication use (Simoni, Pantalone, Plummer & Huang, 2007). Depression is the most frequently diagnosed psychiatric disorder in HIV-positive individuals (Treisman, Angelino & Hutton, 2001), with rates two to four times higher than those in the general public (Ciesla & Roberts, 2001; Morrison et al., 2002). Depression in HIV/AIDS is linked to negative health outcomes, such as disease progression, immunosuppression, and shorter survival time (Ironson et al., 2005). Anxiety is also common in people living with HIV/AIDS (Moskowitz & Wrubel, 2005). In the HIV Cost and Services Utilization Study (HCSUS), Bing et al. (2001) found 16% of people living with HIV/AIDS had generalized anxiety disorder, versus 2.1% of the general public. Anxiety can accelerate HIV disease progression primarily because of cortisol release which causes immunosuppression (McCain et al., 2003). Emotional dysregulation causes changes in memory and cognition (Berger & Arendt, 2000). Thus, clinicians are encouraged to administer assessments, such as the Beck Depression

Inventory (BDI-2) and the Geriatric Depression Scale (GDS), to determine if changes in memory, attention and concentration are due to mood or due to a dementing disorder.

When working with people living with HIV/AIDS, clinicians should focus on interventions that teach practical approaches to managing daily tasks (Buckingham & Shernoff, 1998). Memory deficits are managed with the use of calendars, appointment books, journals, and tape recorders to track thoughts, concerns, and emotions. Therapists should encourage patients to maintain structure at home, take time to complete tasks to reduce the likelihood of forgetting details, and utilize their social resources (e.g., family and friends) to provide assistance. Moreover, psychotherapy should focus on enhancing and preserving skills that are still intact. Clinicians should use positive reinforcement to praise strengths and minimize conversations on patients' weaknesses (Farber & McDaniel, 1999). Using a cognitive-behavioral approach will help memory because homework assignments and handouts consolidate lessons learned in session (Mohlman et al., 2003; Stanley, Diefenbach, & Hopko, 2004).

Lastly, clinicians who use the International HIV-Dementia Scale (IHDS) should be cautious of classification and interpretation of results. The IHDS was developed to reform many of the limitations seen in the original HIV-Dementia Scale (HDS; Carey et al., 2004; Power, Selnes, Grim & McArthur, 1995). Though the IHDS' sensitivity and specificity is superior, clinicians may overlook individuals with mild or asymptomatic neurocognitive impairment. False negatives are worrisome because individuals, who need additional neuropsychological testing, evaluation and medical treatment, are not identified or seen for follow-up visits. Misdiagnoses can be reduced by supplementing

the screener with additional tests, such as the Hopkins Verbal Learning Test–Revised (HVLT-R; Benedict, Schretlen, Groninger, & Brandt, 1998), and providing a referral for an extensive neuropsychological assessment and medical evaluation.

CHAPTER 7

FUTURE RESEARCH

In the future, more research is needed on sociodemographic variables that contribute to skewed misclassifications of neurocognitive impairment on the International HIV-Dementia Scale (IHDS). Neuropsychological measures are scored based on education level, which depends on type of schooling, method of instruction, teacher qualities, cohort, presence of special education, length of education, and days of attendance, etc. (Manly et al., 2002). Thus, number of school years reported does not necessarily mean similar quality of education, and 12 years of completed education for two individuals may yield different standardized scores. The individual with the same amount of education but lower quality of education may appear more cognitively impaired based on the normative data. Similarly, the individual with higher quality of education may still score within normal limits despite mild neurocognitive impairments (Rohit, 2007). In a study conducted by Ryan et al. (2005), 200 HIV-positive individuals (51% African-American; 24% Latino), were assessed using neuropsychological measures of HIV-associated neurocognitive disorders (HAND). Ethnic minority participants had a larger discrepancy between the number of years of education reported and reading grade level. When researchers used reading grade level instead of years of education, cutoff scores for cognitive impairment fell substantially, particularly for ethnic minority participants. Therefore, standardized scores used to diagnose dementias may lack sensitivity or specificity for ethnic minority populations (Ryan et al., 2005) and may

result in misdiagnoses of cognitive impairment (Fillenbaum, 2001). Furthermore, though the IHDS has excellent specificity, it may lack sensitivity in detecting milder forms of impairment (Carey et al., 2004; Smith, et al., 2003). Future research should focus on reforming the screener to delineate those with milder neuropsychological impairments that are more commonly seen in the era of Highly active antiretroviral therapies (HAART).

In my study, greater symptom load due to medical conditions, contributed more to neurocognitive impairment, than HIV-opportunistic infections. Past researchers found that HIV-related infections lead to cortical atrophy, enlarged ventricles and white matter abnormalities (Portegies, Enting & de Gans, 1993). However, my findings reflect new correlates of impairment in the era of HAART. This study uncovered a topic not addressed in the existing literature: Are the cumulative effects of medical conditions more associated with the occurrence of HIV-related neurocognive impairment, than the toxic impact of HIV-opportunistic infections?

Furthermore, many people living with HIV/AIDS also have a current or past lifetime history of alcohol and drug abuse (Collins et al., 2010). The long term use of toxins have deleterious effects on neuropsychiatric and behavioral functioning. Drug use must be ruled out to diagnose a patient with HAND; yet insufficient research delineates areas of the brain and cognition affected by substance abuse vs. HAND. Future research is needed to address the impact of long term drug use on the brain and immunology of people living with HIV/AIDS.

Lastly, in the era of HAART, people are living longer lives. The number of older

individuals living with HIV in the United States has drastically increased (CDC, 2009). In 2009, approximately 6,990 individuals over the age of 50 were newly diagnosed with HIV and 8,030 diagnosed with AIDS. In 2005, the number of individuals aged 50 and older comprised more than 15% of all new HIV/AIDS diagnoses; 24% of people living with HIV; 29% of people living with AIDS; and 35% of AIDS-related deaths (CDC, 2005). Consequently a new subgroup of the HIV populations is geriatric. With increased age comes a host of physical and psychological change. Older people living with HIV/AIDS are uncertain if their symptoms are attributed to their illness, normal aging, comorbid illnesses, or side-effects from medications (Siegal et al., 1999). Thus, more research is needed to understand the differences between HAND and cognitive changes that occur with the normal aging process.

Overall, my findings reveal that people living with HIV/AIDS continue to experience neurocognitive impairment, though they may not meet diagnostic criteria for HIV-associated dementia (HAD) the most severe form of HAND. Contrary to popular belief, HAND is still prevalent and causes adverse functional consequences, despite HAART's effectiveness. My findings raise a major medical and public health concern (Woods, Moore, Weber & Grant, 2009). HAND diagnoses classifications should be reformed and clinicians must be aware of competing factors that may predict or are associated with neurocognitive impairment in HIV. I believe that this study provides a stepping stone for future researchers to examine predictors of HIV-related neurocognitive impairment and associated concomitants in the new era of HAART.

Table 1

*Demographic Summary (*n = *70)*

Variables		Males (*n* = 32) *n* (%)	Females (*n* = 38) *n* (%)
Ethnicity	African American	20 (60.6)	28 (76.3)
	Latino (a)	2 (6.1)	0 (0.0)
	European American	9 (30.3)	8 (18.5)
	Other	1 (3.0)	2 (5.2)
Occupation	Professional	9 (27.3)	0 (0.0)
	None	6 (18.8)	14 (36.8)
	Labor	4 (12.1)	3 (7.9)
Income	$10,000-$14,999	8 (24.2)	2 (5.3)
	$20,000-$29,000	6 (18.2)	2 (5.3)
Health Insurance	Medicare	18 (54.5)	8 (21.1)
	Medicaid	4 (12.1)	21 (55.3)
	None	6 (18.2)	1 (2.6)
Living Arrangements	Live Alone	20 (60.6)	16 (42.1)
	Live with Spouse	4 (12.1)	6 (15.8)
	Live with Other Family	5 (15.2)	2 (5.3)
IHDS	≥11	21 (56.4)	17 (43.6)
	≤10	11 (34.4)	21 (65.6)
Treats HIV	Family Physician	6 (18.2)	6 (15.8)
	HIV/AIDS Service Org.	6 (18.2)	14 (36.8)
	Community Clinic	4 (12.1)	12 (31.6)
	County Hospital	16 (48.5)	13 (34.2)
HIV-Related Conditions	Pneumocystis pneumonia	6 (18.2)	7 (18.4)
	Tuberculosis	3 (9.1)	6 (15.8)
	Genital herpes	4 (12.1)	7 (18.4)
	Hepatitis C	9 (27.3)	12 (31.6)
General Medical Conditions	Hypertension	13 (39.4)	19 (50.0)
	Dizziness/Fainting	2 (6.1)	7 (18.4)
	Asthma	6 (18.2)	8 (21.1)
	Chronic bronchitis	3 (9.1)	12 (31.6)
	Hypercholesterolemia	9 (27.3)	9 (23.7)
	Type II Diabetes	4 (12.1)	4 (10.5)
	Arthritis	7 (21.1)	10 (26.3)
Falls	Yes	4 (12.1)	7 (18.2)

(table continues)

Table 1 *(continued).*

Variables	Males (*n* = 32) Mean (*SD*)	Females (*n* = 38) Mean (*SD*)	Range
Age (yrs)	48.6 (7.9)	47.5 (10.0)	29-66
Education (yrs)	13.0 (2.7)	11.1 (2.2)	3-18
HIV Conditions	1.8 (1.3)	1.5 (1.7)	0-17
Medical Conditions	2.2 (1.9)	3.8 (2.5)	0-24
Living with HIV (yrs)	15.9 (6.4)	13.4 (6.4)	2-31
Recent CD4 Count	517.5 (224.4)	224.4 (406.1)	0-2000

Table 2

*Mean, Standard Deviation, Range and Reliability Statistics (*n *= 70)*

Variables	Mean (SD)	Actual Range	Possible Range	Reliability
THQ	6.9 (4.8)	0-18	0-18	.71
HSS	93.6 (23.4)	45-160	40-160	.94
IHDS	10.3 (1.7)	4-12	0-12	--*

*Cannot be computed due to the fact that the scale items are discrete do not measure a similar construct (Cronbach, 1951)

Table 3

*Crosstabulation of Age and CVD Risk Factors (*n *= 70)*

Variables		Age		χ^2	*p*
		Under 50	Over 50		
Hypertension	Yes	9	31	18.85	.00
	No	23	9		
Hypercholesterolemia	Yes	5	13	7.10	.01
	No	35	18		

Table 4

Correlation Matrix

	1.	2.	3.	4.	5.	6.	7.	8.
1. IHDS	—							
2. Gender	.22	—						
3. Education	-.27*	-.37**	—					
4. Falls	.32**	.09	-.10	—				
5. HIV Conditions	-.05	-.09	.16	.11	—			
6. Med Conditions	.21	.34**	-.02	.21	.25*	—		
7. HSS	.03	.01	-.08	.16	-.03	.05	—	
8. THQ	-.21	.11	.27*	.04	.35**	.30*	.18	—
9. Ethnic Participants	.31**	.14	-.12	.06	-.08	-.12	-.34**	-.18

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

Table 5

Multivariate Statistics for Logistic Regression (HIV-related neurocognitive impairment; Nagelkerke $R^2 = .368$, $p < .01$*)*

Variables	B	OR	95% CI	*p*	Tolerance	VIF
THQ	-0.15	0.87	0.8-1.0	.05	-1.99	.05
Falls	2.00	1.97	1.1-47.0	.04	2.11	.04
Ethnic	2.20	1.90	1.7-48.4	.01	2.73	.01
Medical Conditions	0.30	1.34	1.0-1.8	.05	2.00	.05

APPENDIX A

INFORMED CONSENT

Informed Consent Form

Title of Study: Project Heart

Before agreeing to participate in this research study, it is important that you read and understand the following explanation of the purpose and benefits of the study and how it will be conducted. If you do not understand any part of the study or if you have any questions, please consult a member of the research team.

Principal Investigator: Mark Vosvick, Ph.D. Associate Professor, University of North Texas (UNT), Department of Psychology, Division of Health Psychology and Behavioral Medicine.

Co-Investigators: John Ruiz, Ph.D., Assistant Professor, Department of Psychology, Division of Health Psychology and Behavioral Medicine

Chwee Lye Chng, Ph.D., Regents Professor, Department of Kinesiology, Health Promotion and Recreation

Anthony Mazzarulli, M.D., Emergency Psychiatry and Addiction Medicine Green Oaks Hospital

Start Date of Study: Aug. 01, 2009 End Date of Study: July 31st, 2010

Purpose of the Study: You are being asked to participate in a research study. The study will consist of a survey which addresses issues such as health conditions, health-related behaviors, and various psychological and social factors affecting HIV-positive individuals. The study will also involve a physiological measure of stress and recollection of past negative events. This information will be used to better understand factors associated with stress in the HIV-positive individuals.

Description of the Study: Your participation in Phase I of the study involves completing a set of survey questions design to gather information about psychosocial factors and past trauma. Following Phase I researchers will screen for participation in the second phase of our study. If invited to return for Phase II, you will be asked to recall a past negative event and participate in a psychological assessment of stress, which will include the use of surface electrodes. Finally, you will also be asked to complete additional surveys.

Procedures: The study will be conducted in a private room at the Resource Center of Dallas, Parkland Hospital, the Center for Psychosocial Health Research in Terrill Hall, room 284, at the University of North Texas, AIDS Service of North Texas, AIDS Outreach Center, or the UNT Health Science Center in Fort Worth. Phase I of the study will take approximately 1.5 hour to complete. The survey includes questions about

health and living with HIV/AIDS. Some topics include stigma, forgiveness, and stress. Additional screening will be conducted to discern the appropriateness for the final phase of the study. Phase II of the study will take approximately 1.5 hours. This phase will involve electrodes being placed on the skin of participants to record physiological data and event recollection. We will also collect blood pressure information during this phase. Following the physiological session participants will complete additional surveys.

Foreseeable Risks: The topics addressed throughout the course of the study may prove a source of emotional discomfort. Although unlikely, survey questions administered during Phase I may trigger anxiety, stress, fear, confusion, embarrassment, depression, or guilt. During Phase II, you may experience stress associated with event recollection. You may experience minor skin irritation or redness associated with electrode placement. Upon completion of Phase II, you will be given toll-free phone numbers to crisis lines and low-cost mental health services to assist in event of distress.

Benefits to the Subjects or Others: Although no benefits are promised, you may gain some insight into your socioemotional functioning. Findings from this study will inform the development of interventions and communications to improve health-care for persons living with HIV/AIDS.

Compensation for Participants: After completing Phase I of the survey, you will be compensated $10. If invited to participate in Phase II, you will receive compensation of $25 upon completion. All participants will sign a receipt for compensation.

Confidentiality: Data collection for the survey questions will be conducted in a private area at the Resource Center of Dallas, Parkland Hospital in Dallas, at the Center for Psychosocial Health a computer laboratory in Terrill Hall, room 284, at the University of North Texas, or at the UNT Health Science Center in Fort Worth. These locations will ensure your confidentiality. Once enrolled in the study you will be assigned a participant code number that will be used in all data collection. No one outside of the PI and office manager will have access to any data or information associated with the project or participants. The data will be de-identified and also be kept on a password-protected computer by the office manager and the PI. All data will be secured and accessed only by research staff.

To maintain confidentiality of the information provided, all data will be secured in a locked office in the Center for Psychosocial Health Research at UNT. Should data pertaining to this research be published, your identity will not be revealed.

Voluntary Participation: Participation in this research study is voluntary and you are free to withdraw your consent at any time without penalty or losing benefit to which you are otherwise entitled.

Questions about the Study: If you have any questions about the study, you may contact the Center for Psychosocial Health by phone at (940) 891-6844 or contact Mark Vosvick, Ph.D., Department of Psychology at (940) 565-4715 or by email at vosvick@unt.edu.

Research Participant's Rights: Your signature below indicates that you have read or have had read to you all of the above and that you confirm that all of the following:

Dr. Vosvick or a designated research assistant from the Center for Psychosocial Health has explained the study to you and answered all of your questions. You have been told the possible benefits and the potential risks and/or discomforts of the study.

You understand that you do not have to take part in this study, and your refusal to participate or your decision to withdraw will involve no penalty or loss of rights or benefits. The study personnel may choose to stop your participation at any time.

You understand why the study is being conducted and how it will be performed.

You understand your rights as a research participant and you voluntarily consent to participate in this study.

You understand that you will receive a copy of this consent form.

_____

_____

Printed Name of Participant Date

_____

_____

Signature of Participant Date

For the Principal Investigator or Designee: I certify that I have reviewed the contents of this form with the subject signing above. I have explained the possible benefits and the potential risks and/or discomforts of the study. It is my opinion that the participant understood the explanation.

_____

_____

Signature of Principal Investigator or Designee Date

APPENDIX B

RECRUITMENT FLYERS

PROJECT HEART

R U +?

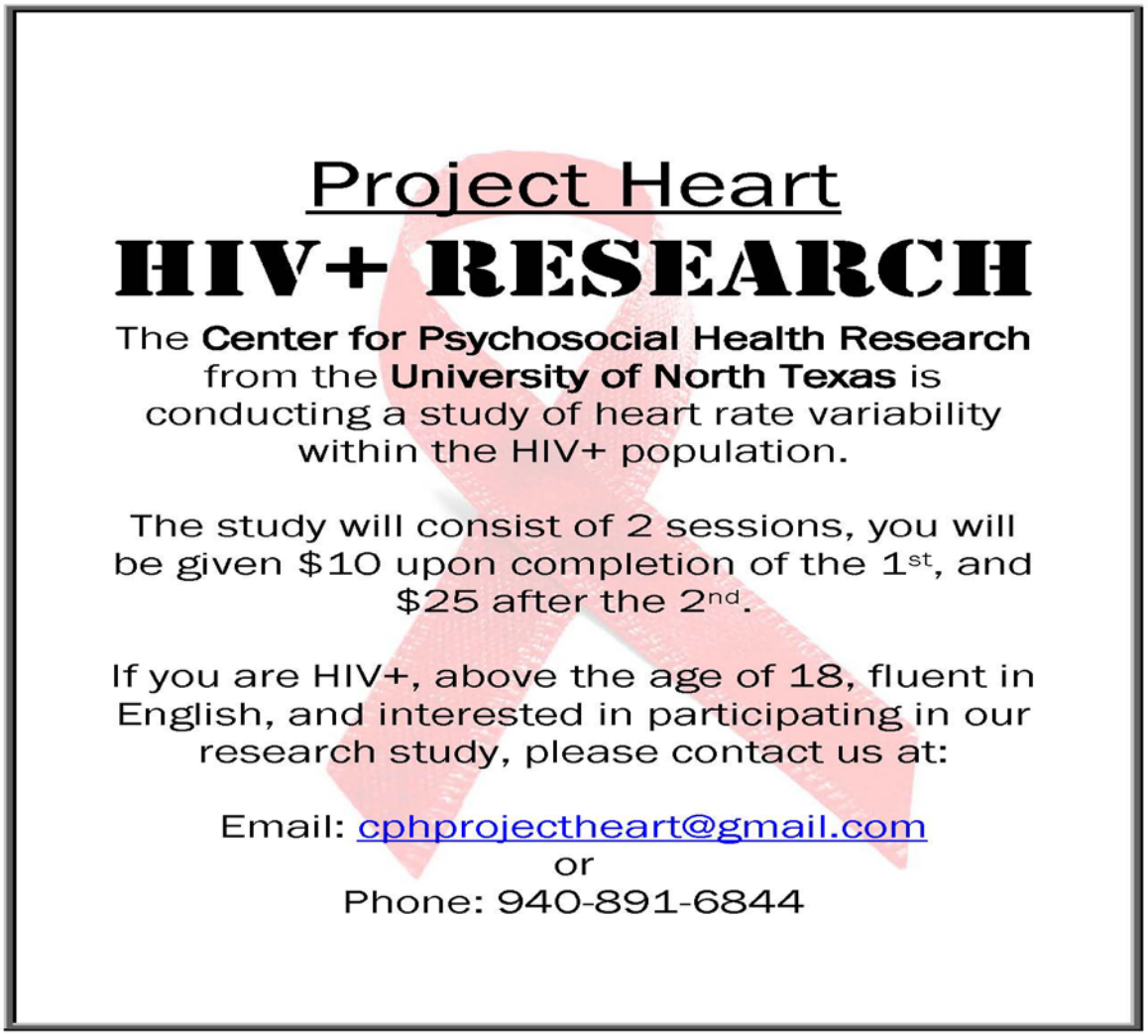
The **Center for Psychosocial Health Research** from the **University of North Texas** is conducting a study of heart rate variability within the HIV+ population.

The study will consist of 2 sessions, you will be compensated $10 upon completion of the 1st, and $25 after the 2nd.

If you are HIV+, above the age of 18, fluent in English, and interested in participating in our research study, please contact us at:

Email: cphprojectheart@gmail.com
or
Phone: 940-891-6844

Center for Psychosocial Health Research
Phone:940-891-6844
Email:cpprojectheart@gmail.com


Project Heart
HIV+ RESEARCH
The Center for Psychosocial Health Research from the University of North Texas is conducting a study of heart rate variability within the HIV+ population.
The study will consist of 2 sessions, you will be given $10 upon completion of the 1st, and $25 after the 2nd.
If you are HIV+, above the age of 18, fluent in English, and interested in participating in our research study, please contact us at:
Email: cphprojectheart@gmail.com
or
Phone: 940-891-6844

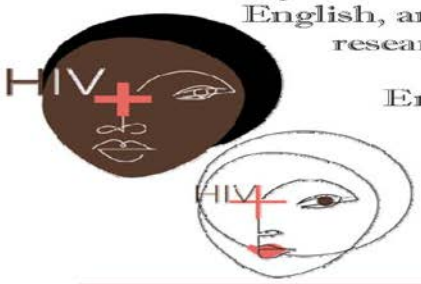
Project Heart

The **Center for Psychosocial Health Research** from the **University of North Texas** is conducting a study of heart rate variability within the HIV+ population.

The study will consist of 2 sessions, you will be given $10 upon completion of the 1st, and $25 after the 2nd.

If you are HIV+, above the age of 18, fluent in English, and interested in participating in our research study, please contact us at:

Email: cphprojectheart@gmail.com
or
Phone: 940-891-6844



Center for Psychosocial Health Research
Phone: 940-891-6844
E-mail: cphprojectheart@gmail.com

APPENDIX C

ELECTRONIC RECRUITMENT

The Center for Psychosocial Health Research at UNT is initiating a new study examining stress in HIV+ adults. We are currently actively involved in recruiting participants for a study called Project Heart. The purpose of this study is to gather information about stress and health in HIV+ adults. The study is conducted in 2 phases. In Phase I, participants are asked to complete an anonymous computer-based survey, which takes about an hour, and are compensated $10 for their time. Some participants will be schedule to come back to participate in Phase II. During this phase, we will measure physiological indicators of stress (heart rate and blood pressure), participants will be compensated $25 for this phase. Our Center is devoted to ensuring equal representation among all subgroups of the community. This study has been approvd by the Insititutional Review Board. Participants can be scheduled to complete the survey in Dallas at the Resource Center, in Denton at our Center on the UNT campus, or in Fort Worth at the UNT Health Science Center.

We are asking area groups and individuals to pass along word of our study to HIV+ individuals. Also, we are opened to any suggestions on potential recruitment venues. Our researchers are willing to speak to any group in the Dallas Fort Worth area. We are aware of the need for confidentiality and our researchers are trained to handle individuals' information with care. Please contact me with any questions or concerns, my contact information is listed below. I look forward to hearing from you.

Thank you,
Researcher
University of North Texas
Center for Psychosocial Health Research

For more information,
contact us at **cphprojectheart@gmail.com**
or visit our website at **www.unt.edu/cph**
Terrill Hall 284
940-891-6844

APPENDIX D

EMPLOYMENT/INCOME

1. Which of the following best describes your employment status?
    - Homemaker
    - Retired
    - Working full time for pay
    - Working part time for pay
    - Not currently employed looking for work for less than 3 months
    - Not currently employed looking for work for more than 3 months
    - On leave of absence
    - Disabled (not working because of temporary or permanent disability)
    - Other (please specify): _____

2. What kind of occupation do you/ did you have?
    - Professional
    - Technical
    - Clerical
    - Crafts
    - Operatives
    - Service or Domestic (Homemaker)
    - Other (please specify: _____

3. How satisfied would you say you are/ were with your work?
    - Very dissatisfied
    - Somewhat dissatisfied
    - Neither dissatisfied nor satisfied
    - Somewhat satisfied
    - Very satisfied
4. Has your heart condition caused you to be unemployed?
    - Yes
    - No
5. Which category best describes your yearly household income before taxes? Please consider all sources that contribute to your income (e.g., pay, gifts, investments, etc.)
    - Less than $5,000
    - $5,000 – $9,999
    - $10,000 – $14,999
    - $15,000 – $19,999
    - $20,000 – $29, 999
    - $30,000 – $39,999
    - $40,000 –$49,999
    - $50,000 – $69,999
    - $70,000 – $99,999
    - $100,000 – $124, 999
    - $125,000 or more

APPENDIX E

MENTAL HEALTH CONTACTS

Project Heart

You have participated in a study of stress and health. This study addressed issues such as health conditions, health-related behaviors, and various psychological and social factors affecting HIV-positive individuals. The purpose of this survey was to evaluate psychophysiological manifestations of stress associated with negative affect. To accomplish this we measured heart rate variability (HRV) associated with negative imagery. HRV is a physiological measure of stress, which is commonly used in clinical settings. If differences in HRV levels are found, this will validate the use of this physiological measure in HIV+ individuals.

Parts of this study may have been difficult, your generosity and willingness to participate in this study are greatly appreciated. Your input will help contribute to the understanding of stress and health in HIV+ individuals. Sometimes people find the subject matter of these questionnaires or imagery session disturbing. If answering any of these questions led you to feel distressed and you would like to speak to someone about your thoughts, please contact one of the following:

Dallas Metrocare Services
Phone: 214-743-1200 #0
Services: Provides adult mental health care, medication management, and counseling services
Fee: Sliding scale fee

University of North Texas - Dallas Campus Counseling Clinic
Phone: 972-780-3646

Galaxy Counseling Center
Phone: 972-272-4429
Services: all types of counseling, individual and family, also have Anger Management group
Fee: Sliding scale fee, typically ranges from $30-120 per session

Legacy Counseling Center-Dallas
Phone: 214-520-6308
Services: Individual, group, and substance abuse counseling for persons living with HIV/AIDS
Fee:
AIDS Outreach Center
Phone: 817-335-1994
Services: Counseling for persons living with HIV/AIDS
Fee: No Cost

University of North Texas Psychology Clinic
Phone: 940-565-2631 or Metro 817-267-3731 ext.2631
Services: Provides individual, group, marriage and family, and play therapy. as well as behavioral medicine health services and evaluations.
http://www.psyc.unt.edu/clinic/index.htm
Fee: Reduced - cost sliding scale

Parkland Health and Hospital System – The Psychiatric ER
Phone: 214-590-8761
Services: Provides mostly emergency care, homicidal and suicidal intentions, will refer to appropriate clinic, will provide first time medication prescriptions if needed
Fee: Cost depends on insurance, will provide North Star Insurance to those who do not have insurance

As stated earlier, your responses to all of the questionnaires will be absolutely confidential. Your name will be converted to a code number, and only people who are associated with this research will see your name or your responses.

If you have any questions about the study, you may contact the Center for Psychosocial Health by phone at (940) 891-6844 or contact Mark Vosvick, Ph.D., Department of Psychology at (940) 565-4715 or by email at vosvick@unt.edu.

APPENDIX F

INSTITUTIONAL REVIEW BOARD (IRB) APPROVAL

UNT
UNIVERSITY OF
NORTH TEXAS
Discover the power of ideas.

OFFICE OF THE VICE PRESIDENT FOR RESEARCH AND ECONOMIC DEVELOPMENT

September 11, 2009

Mark Vosvick
Department of Psychology
University of North Texas

Re: Human Subjects Application No. 09302

Dear Dr. Vosvick:

As permitted by federal law and regulations governing the use of human subjects in research projects (45 CFR 46), the UNT Institutional Review Board has reviewed your proposed project titled "Project Heart." The risks inherent in this research are minimal, and the potential benefits to the subject outweigh those risks. The submitted protocol is hereby approved for the use of human subjects in this study. **Federal Policy 45 CFR 46.109(e) stipulates that IRB approval is for one year only, September 11, 2009 to September 10, 2010.**

Enclosed is the consent document with stamped IRB approval. Please copy and **use this form only** for your study subjects.

It is your responsibility according to U.S. Department of Health and Human Services regulations to submit annual and terminal progress reports to the IRB for this project. The IRB must also review this project prior to any modifications.

Please contact Shelia Bourns, Research Compliance Administrator, or Boyd Herndon, Director of Research Compliance, at extension 3940, if you wish to make changes or need additional information.

Sincerely,

Patricia L. Kaminski, Ph.D.
Associate Professor
Chair, Institutional Review Board

PK:sb

REFERENCES

Adeyemi, O. (2007). Cardiovascular risk and risk management in HIV-infected patients. *Cardiovascular Risk*, *15*(5), 159–162.

Ali, N. S. (2002). Prediction of coronary heart disease preventative behaviors in women: A test of the health belief model. *Women & Health, 35*(1), 83-96.

Alim, T. N., Graves, E., Mellman, T. A., Aigbogun, N., Gray, E., & Lawson, W. (2006). Trauma exposure, posttraumatic stress disorder and depression in an African-American primary care population. *Journal of the National Medical Association*, *98*, 1630-1636.

Alim, T.N., Feder, A. & Graves, R.E. (2008). Trauma, resilience, and recovery in a high-risk African-American population. *American Journal of Psychiatry*, *165*, 1566-1575.

Allison, K. W. (1998). Stress and oppressed category membership. In *Prejudice: The target's perspective* (pp. 145-170). San Diego, CA: Academic.

Almeida, O.P. & Lautenschlager, N.T. (2005). Dementia associated with infectious diseases. *Int Psychogeriatr, 17*, S65-S77.

Ances, B.M., Roc, A.C. & Korczykowski, M. (2008). Combination antiretroviral therapy modulates the blood oxygen level-dependent amplitude in human immunodeficiency virus-seropositive patients. *Journal of Neurovirology, 14*, 418-424.

Andersson, S., Bergedalen, A.M. (2002). Cognitive correlates of apathy in traumatic brain injury. *Neuropsychiatry, Neuropsychology, & Behavioral Neurology*, *15*, 184-191.

Andrasik, M.P., Rose, R., Pereira, D. & Antoni, M. (2008). Barriers to cervical cancer screening among low-income HIV-positive African-American women. *Journal of Health Care for Poor and Underserved*, *19*, 912-925.

Antinori, A., Arendt, G., Becker, J.T., Brew, B.J., Byrd, D.A. & Cherner, M. (2007). Updated research nosology for HIV-associated neurocognitive disorders. *Neurology*, *69*(18), 1789-1799.

Anthony, I. C., & Bell, J. E. (2008). The neuropathology of HIV/AIDS. *International Review of Psychiatry*, *20*(1), 15-24.

Anthony, I. C., & Bell, J. E. (2005). Effects of advances in therapy on the neuropathology of HIV infection. *ACNR*, *5*, 20–22.

Arendt, G., Hefter, H., Elsing, C., Strohmeyer, G. & Freund, H. J. (1990). Motor dysfunction in HIV-infected patients without clinically detectable central-nervous deficit. *Journal of Neurology*, *237*(6), 362-368.

Agronick, G., O'Donnell, L., Stueve, A., Doval, A. S., Duran, R. & Vargo, S. (2004). Sexual behaviors and risks among bisexually- and gay-identified young Latino men. *AIDS and Behavior*, *8*, 185-197.

Backman, L., Wahlin, A., Small, B. J., Herlitz, A., Winbald, B., & Fratiglioni, L. (2004). Cognitive functioning in aging and dementia: The Kungsholmen Project. *Aging, Neuropsychology, and Cognition*, *11*(3), 212-244.

Bandura, A. (1977). Self-efficacy: Toward a unifying theory of behavioral change. *Psychological Review*, *84*, 191-215.

Barnea-Goraly, N., Menon, V. & Eckert, M. (2005). White matter development during childhood and adolescence: A cross-sectional diffusion tensor imaging study. *Cereb Cortex*, *15*, 1848-1854.

Barnhoorn, F. & Adriaanse, H. (1992). In search of factors responsible for noncompliance among tuberculosis patients in Wardha district India. *Social Science and Medicine*, *34*(3), 291-306.

Bassel, C., Rourke, S. B., Halman, M. H. & Smith, M. L. (2002). Working memory performance predicts subjective cognitive complaints in HIV infection. *Neuropsychology, 16*, 400-410.

Berg, E., Biessels, G. & Craen, A. (2007). The metabolic syndrome is associated with decelerated cognitive decline in the oldest old. *Neurology*, *69*, 979-985.

Benedict, R. H. B., Mezhir, J. J., Walsh, K. & Hewitt, R. G. (2000). Impact of human immunodeficiency virus type-1-associated cognitive dysfunction on activities of daily living and quality of life. *Archives of Clinical Neuropsychology, 15*, 535-544.

Benedict, R. H. B., Schretlen, D., Groninger, L, & Brandt, J. (1998). Hopkins Verbal Learning Test–Revised: Normative data and analysis of inter-form and test–retest reliability. *Clinical Neuropsychologist*, *12*, 43-55.

Bensley, L.S., Van Eenwyk, J., & Simmons, K.W. (2000). Self-reported childhood sexual and physical abuse and adult HIV-risk behaviors and heavy drinking. *American Journal of Preventive Medicine*, *18*, 151-158.

Berger, B.E., Ferrans, C.E. & Lashley, F.R. (2001). Measuring stigma in people with HIV: Psychometric assessment of the HIV Stigma Scale. *Research in Nursing and Health, 24*, 518-529.

Berger, J. R., & Arendt, G. (2000). HIV Dementia: The role of the basal ganglia and dopaminergic systems. *Journal of Psychopharmacology, 14,* 214-221.

Bergersen, B. M. (2006). Cardiovascular risk in patients with HIV infection: Impact of antiretroviral therapy. *Drugs, 66*(15), 1971-1987.

Bing, E.G., Burnam, M.A., Longshort, D., Fleishman, J.A., Sherbourne, C.D., London , A.S., Turner, B.J., Eggan, F., Beckman, R., Vitiello, B., Morton, S.C., Orlando, M., Bozzette, S.A., Ortiz-Barron, L. & Shapiro, M. (2001). Psychiatric disorders and drug use among human immunodeficiency virus-infected adults in the United States. *Archives of General Psychiatry, 58*, 721-728.

Boardman, J., Blalock, C. & Button, T (2008). Sex differences in the heritability of resilience. *Twin Res Hum Genet*, *11*, 12-27.

Bodenlos, J.S., Karen, M.A., Grothe, M.A., Kendra, K., Whitehead, D., Copeland, A.L., & Brantley, P.J. (2004). Attitudes toward HIV Health Care Providers Scale: Development and validation. *AIDS Patient Care and STDs*, *18* (12).

Boekamp, J.R., Strauss, M.E. & Adams, N. (1995). Estimating premorbid intelligence in African-American and white elderly veterans using the American version of the National Adult Reading Test. *Journal of Clinical and Experimental Neuropsychology*, *17*, 645-653.

Brady, C. B., Spiro, A., McGlinchey-Berroth, R., Milberg, W., & Gaziano, J. M. (2001). Stroke risk predicts verbal fluency decline in healthy older men: Evidence from the normative aging study. *Journal of Gerontology*, *56*, 340-346.

Brancati, F.L., Kao, W.H.L., Folsom, A.R. (2000). Incident type 2 diabetes mellitus in African American and white adults: The atherosclerosis risk in communities study. *Journal of the American Medical Association, 283*, 2253-2259.

Bremner , J.D. & Narayan, M. (1998). The effects of stress on memory and the hippocampus throughout the life cycle: Implications for childhood development and aging. *Developmental Psychopathology*, *10*, 871-886.

Bremner, J.D., Southwick, S.M. & Charney, D.S. (1999). The neurobiology of posttraumatic stress disorder: An integration of animal and human research. In *Posttraumatic stress disorder: A comprehensive text* (pp. 103-143). New York, NY: Allyn & Bacon.

Brew, B.J. (2004). Evidence for a change in AIDS dementia complex in the era of highly active antiretroviral therapy and the possibility of new forms of AIDS dementia complex. *AIDS*, *18* (1), 75-78.

Brew, B.J., Bhalla, R.B., Paul, M., Gallardo, H., McArthur, J.C. & Schwartz, M.K. (1990). Cerebrospinal fluid neopterin in human immunodeficiency virus type 1 infection. *Ann Neurol*, *28*, 556-60.

Brew, B.J., Crowe, S.M., Landay, A., Cysique, L.A. & Guillemin, G. (2009). Neurodegeneration and ageing in the HAART era. *Journal of Neuroimmune Pharmacol*ogy, *4*, 163-174.

Brewin, C. R., Andrews, B., & Valentine, J. D. (2000). Meta-analysis of risk factors for PTSD in trauma-exposed adults. *Journal of Consulting and Clinical Psychology*, *68,* 748-766.

Buckingham, S., & Shernoff, M. (1998). Psychosocial interventions in persons with HIV-associated neuropsychiatric compromise. In *Practitioner's guide to the neuropsychiatry of HIV/AIDS* (pp. 228-240). New York, NY: Guilford Press.

Bunting, S. M. (1996). Sources of stigma associated with women with HIV. *Advances in Nursing Science, 19* (2), 64-73.

Burgess, A.W. & Phillips, S.L. (2009). Sexual abuse, trauma and dementia in the elderly: A retrospective study of 284 cases. *Journal of the American Geriatrics Society*, *54*(7), 1154-1155.

Calhoun, L.G., Cann, A., Tedeschi, R.G., & McMillan, J. (2000). A correlational test of the relationship between posttraumatic growth, religion, and cognitive processing. *Journal of Traumatic Stress*, *13,* 521-527.

Campbell, A., Rorie, K., Dennis, G.,Wood, D., Combs, S., Hearn, L., Davis, H., Brown, A. & Weir, R. (1996). Neuropsychological assessment of African Americans: Conceptual and methodological considerations. In R. Jones (Ed.), *Handbook of tests and measurement for black populations* (pp. 75-84). Berkeley, CA: Cobb & Henry.

Carey, C. L., Woods, S. P., Rippeth, J. D., Heaton, R. K. & Grant, I. (2006). Prospective memory in HIV-1 infection. *Journal of Clinical and Experimental Neuropsychology, 28*, 536-548.

Carey, C.L., Woods, S.P., Rippeth, J.D., Moore, D.J & Marcotte, T.D. (2004). Initial validation of a screening battery for the detection of HIV-associated cognitive impairment. *Clinical Neuropsychology, 18* (2), 234-248.

Carter, S. L., Rourke, S. B., Murji, S., Shore, D., & Rourke, B. P. (2003). Cognitive complaints, depression, medical symptoms, and their association with neuropsychological functioning in HIV infection: A structural equation model analysis. *Neuropsychology, 17*, 410-419.

Castellon, S. A., Hardy, D. J., Hinkin, C. H., Satz, P., Stenquist, P. K., van Gorp, W. G., Myers, H. F. & Moore, L. (2006). Components of depression in HIV-1 infection: Neurocognitive performance. *Journal of Clinical and Experimental Neuropsychology, 28*, 420-437.

Castellon, S.A., Hinkin, C.H. & Wood, S. (1998). Apathy, depression, and cognitive performance in HIV-1 infection. *Journal of Neuropsychiatry and Clinical Neuroscience, 10*, 320-329.

Castelo, J. M., Sherman, S. J., Courtney, M. G., Melrose, R. J., & Stern, C. E. (2006). Altered hippocampal-prefrontal activation in HIV patients during episodic memory encoding. *Neurology, 66,* 1688-1695.

Centers for Disease Control and Prevention (CDC; 2007). *HIV prevention strategic plan through 2007*. http://www.cdc.gov/hiv/resources/reports/index.htm

Centers for Disease Control and Prevention (CDC; 2008). *HIV prevention strategic plan through 2008*. http://www.cdc.gov/hiv/resources/reports/index.htm

Chadda, R. K., Agarwal, V., Singh, M. C. & Raheja,D. (2001). Help seeking behaviour of psychiatric patients before seeking care at a mental hospital. *International Journal of Social Psychiatry, 47*(4), 71-78.

Chan, I., Chan, E., Au, A., Leung, P., Li., P., Lee, M. P., Chung, R. & Yu, P. (2007). Subjective memory complaints of Chinese HIV-infected patients in Hong Kong: Relationships with social support, depressive mood and medical symptoms. (2007). *AIDS Care*, *19* (9), 1149-1156.

Chang, L., Lee, P.L., Yiannoutsos C.T., Ernst, T., Marra, C.M. & Richards, T. (2004). A multicenter in vivo proton-MRS study of HIV-associated dementia and its relationship to age. *Neuroimage*, *23*(4), 1336-1347.

Chang, L., Speck, O., Miller, E. N., Braun, J., Jovicich, J. & Koch, C. (2001). Neural correlates of attention and working memory deficits in HIV patients. *Neurology, 57,* 1001-1007.

Chesney, M.A. & Smith, A.W. (1999). Critical delays in HIV testing and care: The potential role of stigma. *American Behavioral Scientist, 42*(7), 1162-1174.

Choi, D.W. (1988). Calcium-mediated neurotoxicity: Relationship to specific channel types and role in ischemic damage. *Trends Neurosci, 11*, 465-469.

Ciesla, J.A. & Roberts, J.E. (2001). A meta-analysis of risk for major depressive disorder among HIV-positive individuals. *American Journal of Psychiatry, 158*, 725-730.

Clark, H.J., Lindner, G. Armistead, L. & Austin, B. (2003). Stigma, disclosure, and psychological functioning among HIV-infected and non-infected African American women. *Women and Health, 38*, 57-71.

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences*. Hillsdale, NJ: Erlbaum.

Cohen, S. (2007). Psychological stress and disease. *Journal of the American Medical Association*, *298*(14), 1685.

Cohen, M., Brom, D., & Dasberg, H. (2001). Child survivors of the Holocaust: Symptoms and coping after 50 years. *Israel Journal of Psychiatry*, *38*, 3-13.

Cole, M. A., Margolick, J. B., Cox, C., Li, X., Selnes, O. A. & Martin, E. M. (2007). Longitudinally preserved psychomotor performance in long-term asymptomatic HIV-infected individuals. *Neurology, 69*, 2213–2220.

Colledge, N. (2000). Falls. *Reviews in Clinical Gerontology*, *7*(4), 309-315.

Collins, R.L., Taylor, S.L., Elliott, M.N., Ringel, J.S., Kanouse, D.E. & Beckman, R. (2010). Off-premise alcohol sales policies, drinking, and sexual risk among people living with HIV. *American Journal of Public Health*, *100*(10), 1890-1892.

Collishaw, S., Pickles, A. & Messer. J. (2007). Resilience to adult psychopathology following childhood maltreatment: evidence from a community sample. *Child Abuse and Neglect*, *31*, 211-229.

Collani, G. V., Grumm, M. & Steicher, K. (2010*). An investigation of the determinants of stigmatization and prejudice toward people living with HIV/AIDS*. Journal compilation: Wiley Periodicals, Inc.

Connors, J & Hely, A. (2007). Attitudes toward people living with HIV/AIDS: A model of attitudes to illness. *Journal of Applied Social Psychology*, *37*(1), 124-130.

Constantine, M. G., Myers, L. J., Kindaichi, M., & Moore, J. L. (2004). Exploring indigenous mental health practices: The roles of healers and helpers in promoting well-being in people of color. *Counseling and Values, 48*, 110-125.

Courtney, S. M. (2004). Attention and cognitive control as emergent properties of information representation in working memory. *Cognitive, Affective and Behavioral Neuroscience, 4,* 501-516.

Crocker, J., Major, B., & Steele, C. (1998). Social stigma. In D. Gilbert, S. T. Fiske, & G. Lindzey (Eds.), *The handbook of social psychology* (pp. 504-553). New York, NY: McGraw Hill.

Cronbach, L. J. (1951). Coefficient alpha and the internal structure of tests. *Psychometrika, 16*(3), 297-334.

Cruess, D., Antoni, M.H., Gonzalez, J., Fletcher, M.A., Klimas, N., Duran, R., Ironson, G. and Schneiderman, N. (2003). Sleep disturbance mediates the association between psychological distress and immune status among HIV-positive men and women on combination antiretroviral therapy. *Journal of Psychosomatic Research*, *54*, 185-189.

Davis, R. G., Ressler, K. J., Schwartz, A. C., Stephens, K., & Bradley, R. G. (2008). Treatment barriers for low-income, urban African Americans with undiagnosed posttraumatic stress disorder. *Journal of Traumatic Stress*, *21*(2), 218-222.

Dawes, S., Suarez, P., Casey, C. Y., Cherner, M., Marcotte, T. D. & Letendre, S. (2008). Variable patterns of neuropsychological performance in HIV-1 infection. *Journal of Clinical and Experimental Neuropsychology*, *30*, 613-626.

De Bellis, M. D., Keshavan, M. S., Clark, D. B., Casey, B. J., Giedd, J. N., Boring, A. M., Frustaci, K., & Ryan, N. D. (1999). Developmental traumatology part II: Brain development. *Biological Psychiatry*, *45,* 1271-1284.

Delis, D. C., Kramer, J. H. & Kaplan, E. (2000). *California Verbal Learning Test*-Second Edition (CVLT-II). San Antonio, TX: Psychological Corporation.

Dias, S.F., Matos, M.G. & Gonçalves, A.C. (2006) AIDS-related stigma and attitudes towards AIDS-infected people among adolescents**.** *AIDS Care***,** *18*(3), 208-214.

Dickerson, S.S. & Kemeny, M.E. (2004). Acute stressors and cortisol responses: a theoretical integration and synthesis of laboratory research. *Psychol Bull, 130*(3), 355-91.

Dray-Spira, R.& Lert, F. (2003). Social health inequalities during the course of chronic HIV disease in the era of highly active antiretroviral therapy. *AIDS, 17*, 283.

Dyregrov, A. (2004). Educational consequences of loss and trauma. *Educational and Child Psychology*, *21*(3), 77-84.

Ehlers, A., & Clark, D. M. (2000). A cognitive model of posttraumatic stress disorder. *Behaviour Research and Therapy, 38*, 319-345.

Emlet, C. A. (2007). A comparison of HIV stigma and disclosure patterns between older and younger adults living with HIV/AIDS. *AIDS Patient Care and STDs*, *20*(5), 350-358.

Endres, H.G., Dasch, B., Lugenhauser, M., Maier, C., Smektala, R., Trampisch, H.J. & Pientka, L. (2006). Patients with femoral or distal forearm fracture in Germany: A prospective observational study on health care situation and outcome. *BMC Publ Health*, *4*, 6-87.

Erkinjuntti, T. (1999). Cerebrovascular dementia: pathophysiology, diagnosis and treatment. *CNS Drugs*, *12*, 35-48.

Erkinjuntti, T. (2005). Cerebrovascular disease, vascular cognitive impairment and dementia. *Psychiatry, 4* (1), 48-51.

Ernst, T., Chang, L., Jovicich, J., Ames, N., & Arnold, S. (2002). Abnormal brain activation on functional MRI in cognitively asymptomatic HIV patients. *Neurology, 59*, 1343-1349.

Eron, J., Yeni, P. & Gathe, J. (2006). The KLEAN study of fosamprenavir-ritonavir versus lopinavir-ritonavir, each in combination with abacavir-lamivudine, for initial treatment of HIV infection over 48 weeks: A randomized non-inferiority trial. *Lancet, 368*, 476-482.

Esiri, M.M., Biddolph, S.C. & Morris, C.S. (1998). Prevalence of Alzheimer plaques in AIDS. *Journal of Neurological Neurosurgery Psychiatry, 65*, 29-33.

Faílde-Garrido, J. M., Alvarez, M. & Simón-López, M. A. (2008). Neuropsychological impairment and gender differences in HIV-1 infection. *Psychiatry & Clinical Neurosciences*, *62*(5), 494-502.

Farber, E. W., & McDaniel, J. S. (1999). Assessment and psychotherapy practice implications of new combination antiviral therapies for HIV disease. *Professional Psychology: Research and Practice*, *30*(2), 173-179.

Faul, F. & Erdfelder, E. (1992). *GPOWER: A priori, post-hoc, and compromise power analyses for MS-DOS.* Bonn: Universität Bonn, Fachbereich Psychologie.

Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M. & Edwards, V. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults: The Adverse Childhood Experiences (ACE) study. *American Journal of Preventive Medicine*, *14*, 245-258.

Fife, B.L. & Wright, E.R. (2000). The dimensionality of stigma: A comparison of its impact on the self of persons with HIV/AIDS and cancer. *Journal of Health and Social Behavior, 41*(1), 50-67.

Fillenbaum, G.G., Heyman, A., Huber, M.S., Ganguli, M. & Unverzagt, F.W. (2001). Performance of elderly African American and white community residents on the CERAD neuropsychological battery. *Journal of International Neuropsychological Society, 7*, 502-509.

Fitzpatrick, A.L., Kuller, L.H., Ives, D.G., Lopez, O.L., Jagust, W. & Breitner, J.C.S. (2004). Incidence and prevalence of dementia in the cardiovascular health study. *Journal of American Geriatric Society, 52*, 195-204.

Foley, J., Ettenhofer, M., Wright, M., & Hinkin, C. H. (2008). Emerging issues in the neuropsychology of HIV infection. *Current HIV/AIDS Reports*, *5*, 204-211.

Foley, J., Ettenhofer, M., Wright, M. J., Siddiqi, I., Choi, M., Thames, A. D. & Hinkin, C. H. (2010). Neurocognitive functioning in HIV-1 infection: Effects of risk factors and age. *Clinical Neuropsychologist*, *24*(2), 265-285.

Foltzer, M. (2005). *HIV medical alert for primary health care providers and health professionals*. Accessed online on 8/26/2010 from www.upstate.edu/cei

Forette, F., Seux, M. & Staessen, J. (1998). Prevention of dementia in a randomized double blind placebo controlled systolic hypertension in Europe (Syst-Eur) trial. *Lancet, 352*, 1347-1351.

Foster, P. & Gaskins, S. W. (2009). Older African Americans' management of HIV/AIDS stigma. *AIDS Care*, *21*(10), 1306-1312.

Francis, S.A. & Liverpool, J. (2008). A review of faith-based HIV prevention programs. *Journal of Religion and Health*, *48*(1), 6-15.

Frazier, P. A., & Seales, L. M. (1997). Acquaintance rape is real rape. In M. D. Schwartz (Ed.), *Researching sexual violence against women: Methodological and personal perspectives* (pp. 54-65). Thousand Oaks, CA: Sage.

Friis-Moller, N., Reiss, P., Sabin, C.A., Weber, R., Monforte, A. & El-Sadr, W. (2007). Class of antiretroviral drugs and the risk of myocardial infarction. *New England Journal of Medicine, 356*, 1723-1735.

Galvan, F.H., Davis, M., Banks, D., & Bing, E.G. (2008). HIV stigma and social support among African Americans. *AIDS Patient Care and STDs*, *22*(5), 1-14.

Gardner, A.W. & Montgomery, P.S. (2001). Impaired balance and higher prevalence of falls in subjects with intermittent claudication, *Journal of Gerontology and Biological Medical Science, 56,* 454-458.

Ghafouri, M., Shohreh, A., Khalili, K. & Sawaya, B. E. (2006). HIV-1 associated dementia: Symptoms and causes. *Retrovirology*, *2*(28).

Gilbertson, M.W., Gurvits, T.V., Lasko, N.B., Orr, S.P. & Pittman, R. K. (2001). Multivariate assessment of explicit memory function in combat veterans with posttraumatic stress disorder. *Journal of Traumatic Stress, 14*, 413-432.

Glanz, K., Lewis, F. M. & Rimer, B. K. (2002). *Health behavior and health education: Theory, research, and practice*. San Fransisco: Wiley & Sons.

Golding, J. M. (1994). Sexual assault history and physical health in randomly selected Los Angeles women. *Health Psychology, 13*, 130-138.

Gongvatana, A., Schweinsburg, B. C., Taylor, M. J., Theilmann, R. J., Letendre, S. L. & Alhassoon, O. (2007). HIV-associated white matter tract injury and neurocognitive impairment in the HAART era. *Journal of the International Neuropsychological Society*, *14*(1), 145-146.

Gonzalez-Duarte, A., Cikurel, K. & Simpson, D. M. (2004). Rapidly progressive dementia due to bilateral internal carotid artery occlusion with infarction of the total length of the corpus callosum. *Journal of Neuroimaging, 14*(2), 176-9.

Grant, I., Atkinson, J. H., Hesselink, J. R., Kennedy, C. J., Richman, D. D., Spector, S. A., & McCutchan, J. A. (1987). Evidence for early central nervous system involvement in the acquired immunodeficiency syndrome (AIDS) and other human immunodeficiency virus (HIV) infections: Studies with neuropsychologic testing and magnetic resonance imaging. *Annals of Internal Medicine*, *107*, 828-836.

Gray, F., & Keohane, C. (2003). The neuropathology of HIV infection in the era of highly active antiretroviral therapy (HAART). *Brain Pathology, 13*, 79-83.

Green, B.L. (1996). *Trauma History Questionnaire. Measurement of stress, trauma and adaptation* (pp. 366-369). Lutherville, MD.

Griffin, D.E. (1997). Cytokines in the brain during viral infection clues to HIV-associated dementia. *J Clin Invest, 100***,** 2948-2951.

Grinspoon, S. & Carr, A. (2005). Cardiovascular risk and body-fat abnormalities in HIV-infected adults. *New England Journal of Medicine*, *352*, 48-62.

Grinspoon, S.K., Grunfeld, C. & Kotler, D.P. (2008). State of the science conference: Initiative to decrease cardiovascular risk and increase quality of care for patients living with HIV/AIDS: Executive summary. *Circulation, 118*(6), 109.

Gump, J. P. (2010). Reality matters: The shadow of trauma on African American subjectivity. *Psychoanalytic Psychology*, *27*(1), 42-54.

Gunstad, J., Paul, R., Cohen, R., Tate, D., Spitznagel, M. & Gordon, E. (2007). Elevated body mass index is associated with executive dysfunction in otherwise healthy adults. *Comprehensive Psychiatry*, *48*(1), 57-61.

Haase, A.T. (1986). Pathogenesis of lentivirus infections. *Nature, 322*, 130-136.

Halkitis, P. N. (1998). Advances in the treatment of HIV disease: Complexities of adherence and complications for prevention. *Health Psychologist*, *20*(1), 6-15.

Hamra, M., Ross, M.W., Karuri, K., Orrs, M. & D'Agostino, A. (2005). The relationship between expressed HIV/AIDS-related stigma and beliefs and knowledge about care and support of people living with AIDS in families caring for HIV-infected children in Kenya. *AIDS Care*, *17*(7), 911-922.

Harawa, N. T., Greenland, S., Bingham, T. A., Johnson, D. F., Cochran, S. D. & Cunningham, W. (2004). Associations of race/ethnicity with HIV prevalence and HIV-related behaviors among young men who have sex with men in 7 urban centers in the United States. *Journal of Acquired Immune Deficiency Syndromes, 35*, 526-536.

Hardy, D. J., & Hinkin, C. H. (2002). Reaction time performance in adults with HIV/AIDS. *Journal of Clinical and Experimental Neuropsychology*, *24*, 912-929.

Hardy, D. J., Castellon, S. A. & Hinkin, C. H. (2004). Perceptual span deficits in adults with HIV. *Journal of the International Neuropsychological Society, 10*, 135-140.

Hart, J., Kimbrell, T., Fauver, P., Cherry, B. J., Pitcock, J., Booe, L. Q., Tillman, G. & Freeman, T. W. (2008). Cognitive dysfunctions associated with PTSD: Evidence from world war II prisoners of war. *Journal of Neuropsychiatry and Clinical Neurosciences*, *20*, 309-316.

Hart, R. (2009). Child refugees, trauma and education: Interactionist considerations on social and emotional needs and development. *Educational Psychology in Practice*, *25*(4), 351-368.

Heaton, R. K., Grant, I., Butters, N., White, D. A., Kirson, D. & Atkinson, J. H. (1995). The HNRC 500—Neuropsychology of HIV infection at different disease stages. *Journal of the International Neuropsychological Society*, *1*, 231-251.

Heckman, T.J., Kochman, A., Sikkema, K.J., Kalichman, S.C., Masten, J., & Goodkin, K. (2000). Late middle aged and older men living with HIV/AIDS: Race differences in coping, social support and psychological distress. *Journal of the National Medical Association, 92*, 436-444.

Hendricks, K. & Gorbach, S. (2009). Nutrition issues in chronic drug users living with HIV infection. *Addict Science and Clinical Practice*, *5*, 16-23.

Herek, G.M. (1999). AIDS and stigma in the United States. *American Behavioral Scientis*t, 42 (7).Snyder, M., Omoto, A. M., & Crain, A. L. (1999). Punished for their good deeds: Stigmatization of AIDS volunteers. *American Behavioral Scientist*, *42*, 1175-1192.

Herek, G. M., & Capitanio, J. P. (1999). AIDS stigma and sexual prejudice. *American Behavioral Scientist, 42*(7), 1130-1147.

Herek, G.M., Capitanio, J.P. & Widaman, K.F. (2002). HIV-related stigma and knowledge in the United States: Prevalence and trends, 1991-1999. *American Journal of Public Health, 92*(3), 371-377.

Hestad, K., McArthur, J. H., Dal Pan, G. J., Selnes, O. A., Nance-Sproson, T. E. & Aylward, E. (1993). Regional brain atrophy in HIV-1 infection: Association with specific neuropsychological test performance. *Acta Neurologica Scandinavica*, *88*, 112-118.

Hilsabeck, R.C., Perry, W. & Hassane, T.I. (2002). Neuropsychological impairment in patients with chronic hepatitis C. *Hepatolog. 35*, 440-446.

Hines-Martin, V., Malone, M., Kim, S. & Brown-Piper, A. (2003). Barriers to mental health care access in an African American population. *Issues in Mental Health Nursing, 24*, 237-256.

Hinkin, C. H., Castellon, S. A. & Hardy, D. J. (2000). Dual task performance in HIV-1 infection. *Journal of Clinical and Experimental Neuropsychology*, *22*, 16-24.

Hinkin, C. H., Castellon, S. A., Hardy, D. J., Granholm, E., & Siegle, G. (1999). Computerized and traditional Stroop task dysfunction in HIV-1 infection. *Neuropsychology, 13*, 306-316.

Hsue, P. Y., Lo, J. C., Franklin, A., Bolger, A. F., Martin, J. N. & Deeks, S. G. (2004). Progression of atherosclerosis as assessed by carotid intima-media thickness in patients with HIV infection. *Circulation, 109*(13), 1603-1608.

Hsue, P., Lo, J. & Franklin, A. (2005). *C-reactive protein levels in patients with HIV: A marker of cardiovascular risk or chronic infection?* Paper presented at: Conference on Retroviruses and Opportunistic Infections (CROI); Boston.

Hulse, G.K., Lautenschlager, N.T., Tait, R.T. & Almeida, O.P. Dementia associated with alcohol and other drug use. *Int Psychogeriatr, 17*, S109-S127.

Hult, B., Chana, G., Masliah, E., & Everall, I. (2008). Neurobiology of HIV. *International Review of Psychiatry*, *20*, 3-13.

Ickovics, J.R., Hamburger, M.E. & Vlahov, D. (2001). Mortality, CD4 cell count decline, and depressive symptoms among HIV-seropositive women: Longitudinal analysis from the HIV epidemiology research study. *JAMA*, *285*, 1466-1474.

Ironson, G., O'Cleirigh, C., Fletcher, M.A., Laurenceau, J.P., Balbin, E., Klimas, N., Schneiderman, N. & Solomon, G. (2005). Psychosocial factors predict CD4 and viral load change in men and women with human immunodeficiency virus in the era of highly active antiretroviral treatment. *Psychosomatic Medicine*, *67*, 1013-21.

Janz, N.K., & Becker, M.H. (1984). The health belief model: A decade later. *Health Education Quarterly*, *11*, 1-47.

Jellinger K. 2002. Alzheimer disease and cerebrovascular pathology, an update. *Journal of Neural Transmission*, *109*, 813-836.

Jeste, D. Blacker, D. Blazer, D. Ganguli, M., Grant, I. Paulsen, J., Roland, P. & Sachdev, P. (2010). *A proposal from the DSM-5 neurocognitive disorders work group: Neurocognitive disorders*. Washington, DC: American Psychiatric Association.

Johnsen, L. W. & Harlow, L. L. (1996). Childhood sexual abuse linked with adult substance use, victimization and AIDS-risk. *AIDS Education and Prevention, 8*, 44-57.

Johnson, J.G., Alloy, L.B., Panzarella, C., Metalsky, G.I., Rabkin, J.G., William & J.B.W (2001). Hopelessness as a mediator of the association between social support and depressive symptoms: Findings of a study of men with HIV. *Journal of Counseling and Clinical Psychology, 69*, 1056-1060.

Jones, E.E., Farina, A., Hastorf, A.H., Markus, H., Miller, D.T. & Scott., R. (1984). *Social stigma: The psychology of marked relationships*. New York, NY: W.H. Freeman and Co.

Jorm, F. A. (2008). History of depression as a risk factor for dementia: An updated review. *Australasian Journal on Ageing, 10*, 1111.

Kahana, E. & Kahana, B. (2001). Successful aging among people with HIV/AIDS. *Journal of clinical Epidemiology*, *54*(12), 53-56.

Kaiser Family Foundation. (2001). Latinos' view of the HIV/AIDS epidemic at 20 years: Findings from a National Survey. http://www.kff.org/hivaids/3184-index.cfm

Kalichman, S.C., Sikkema, K.J., DiFonzo, K., Luke, W. & Austin, J. (2002). Emotional adjustment in survivors of sexual assault living with HIV-AIDS. *Journal of Traumatic Stress, 15*, 289-296.

Kallin, K., Jensen, J., Olsson, L.L., Nyberg, L. & Gustafson, Y. (2004). Why the elderly fall in residential care facilities, and suggested remedies. *Journal of Family Practice*, *53*, 41-52.

Kalof, L. (2000). Ethnic differences in female sexual victimization. *Sexuality and Culture: An Interdisciplinary Quarterly, 4,* 75-97.

Kang, E., Rapkin, B.D., & DeAlmeida, C. (2006). Are psychological consequences of stigma enduring or transitory? A longitudinal study of HIV stigma and distress among Asians and Pacific Islanders living with HIV illness. *AIDS Patient Care STDS*, *20*(10), 712-723.

Katz, M., Liu, C. & Schaer, M. (2009). Prefrontal plasticity and stress inoculation-induced resilience. *Developmental Neuroscience*, *31*, 293-299.

Kaul, M., Garden, G. A. & Lipton, S.A. (2001). Pathways to neuronal injury and apoptosis in HIV-associated dementia. *Nature*, *410*, 988-994.

Kemppainen, J. K. (2001). Predictors of quality of life in AIDS patients. *Journal of the Association of Nurses in AIDS Care, 12*, 61-70.

Kessler, R. C., Sonnega, A., Bromet, E., Hughes, M. & Nelson, C. B. (1995). Posttraumatic stress disorder in the National Comorbidity Survey. *Archives of General Psychiatry*, *52*, 1048-1063.

Khanlou, N., Moore, D.J. & Chana, G. (2008). Increased frequency of alpha-synuclein in the substantia nigra in human immunodeficiency virus infection. *Journal of Neurovirology.* [Epub ahead of print].

Kimerling, R., Calhoun, K.S., Forehand, R., Armistead, L., Morse, E., Morse, P., Clark, R. & Clark, L. (1999). Traumatic stress in HIV-infected women. *AIDS Education and Prevention*, *11*, 321-30.

Kivipelto, M., Helkala, E. & Laakso, M. (2002). Apolipoprotein E e4 allele, elevated midlife total cholesterol level and high midlife systolic blood pressure are independent risk factors for late-life Alzheimer disease. *Ann Intern Med*, *137*, 149-155.

Koenen, K.C., Driver, K.L. & Oscar-Berman, M. (2001). Measures of prefrontal system dysfunction in posttraumatic stress disorder. *Brain Cognition, 45*, 64-78.

Kramer, L., Bauer, E. & Funk, G. (2002). Subclinical impairment of brain function in chronic hepatitis C infection. *J Hepatol. 37*, 349-354.

Kutner, K.C., Erlanger, D.M., Tsai, J., Jordan, B. & Relkin, N.R. (2000). Lower cognitive performance of football players possessing apolipoprotein E e4. *Neurosurgery, 47*, 651-658.

Leary, J. D. (2005). *Posttraumatic slave syndrome.* Milwaukee*,* OR: Uptone Press.

Leary, M. R. & Schreindorfer, L. S. (1998). The stigmatization of HIV and AIDS: Rubbing salt in the wound. In V. Derlega, & A. Barbee (Eds.), *HIV infection and social interaction* (pp. 12-29). Thousand Oaks, CA: Sage Publications.

Lee, R., Kochman, A. & Sikkema, K. (2002). Internalized stigma among people living with HIV/AIDS. *AIDS and Behavior*, *6*, 309-319.

Lee, G.A., Seneviratne, T., Noor, M.A., Lo, J.C., Schwarz, J.M. & Aweeka, F.T. (2004). The metabolic effects of lopinavir/ritonavir in HIV-negative men. *AIDS*, *18*, 641-649.

Leonard, B. (2000). Stress, depression and the activation of the immune system. *World Journal of Biological Psychiatry*, *1*, 17-25.

Leserman, J., Drossman, D. A., Zhiming, L., Toomey, T. C., Nachman, G. & Glogau, L. (1996). Sexual and physical abuse history in gastroenterology practice: How types of abuse impact health status. *Psychosomatic Medicine*, *58*, 4-15.

Leserman, J. (2008). Role of depression, stress and trauma in HIV disease progression. *Psychosomatic Medicine*, *70*, 539-545.

Leserman, J., Jackson, E.D., Petitto, J.M., Golden, R.N., Silva, S.G., Perkins, D.O., Cai, J., Folds, J.D. & Evans D.L. (1999). Progression to AIDS: The effects of stress, depressive symptoms, and social support. *Psychosomatic Medicine*, *61*, 397-406.

Leserman, J., Pence, B.W., Whetten, K., Mugavero, M.J., Thielman, N.M., Swartz, M.S., Stangl, D. (2007). Relation of lifetime trauma and depressive symptoms to mortality in HIV. *American Journal of Psychiatry, 164*, 1707-1713.

Leserman, J., Petitto, J.M., Golden, R.N., Gaynes, B.N., Gu, H, Perkins, D.O., Silva, S,G., Folds, J.D. & Evans, D.L. (2000). The impact of stressful life events, depression, social support, coping and cortisol on progression to AIDS. *American Journal of Psychiatry, 157*, 1221-1228.

Leserman, J., Petitto, J.M., Gu, H., Gaynes, B.N., Barroso, J., Golden, R.N., Perkins, D.O., Folds, J.D. & Evans, D.L. (2002). Progression to AIDS, a clinical AIDS condition, and mortality: Psychosocial and physiological predictors. *Psychological Medicine*, *32,* 1059-1073.

Leserman, J., Whetten, K., Lowe, K., Stangl, D., Swartz, M.S. & Thielman, N.M. (2005). How trauma, recent stressful events, and PTSD affect functional health status and health utilization in HIV-infected patients in the south. *Psychosom Med, 67*, 500-507.

Letendre, S., Marquie-Beck, J. & Capparelli, E. (2008). Validation of the CNS penetration-effectiveness rank for quantifying antiretroviral penetration into the central nervous system. *Archives of Neurology, 65*, 65-70.

Levine, A. J., Hardy, D. J., Barclay, T. R., Reinhard, M. J., Cole, M. M. & Hinkin, C. H. (2008). Elements of attention in HIV-infected adults: Evaluation of an existing model. *Journal of Clinical and Experimental Neuropsychology*, *30*, 53-62.

Lichtenstein, B. (2000). Secret encounters: Black men, bisexuality, and AIDS in Alabama. *Medical Anthropology Quarterly*, *14*, 374-393.

Lichtenberg, P.A., Ross, T., & Christensen, B. (1994). Preliminary normative data on the Boston Naming Test for an older urban population. *Clinical Neuropsychologist*, *8*, 109-111.

Liebschutz, J.M., Feinman, G., Sullivan, L., Stein, M., & Samet, J. (2000). Physical and sexual abuse in women infected with the human immunodeficiency virus: Increased illness and health care organization. *Archives of Internal Medicine*, *160*(11), 1659-1664.

Liebschutz, J., Saitz, R., Brower, V., Keane, T.M., Lloyd-Travaglini, C., Averbuch, T. (2007). PTSD in urban primary care: High prevalence and low physician recognition. *Journal of General Internal Medicine*, *22*, 719-726.

Ligthart, S. A.., Moll van Charante, E.P., Van Gool, W.A. & Richard, E. (2010). Treatment of cardiovascular risk factors to prevent cognitive decline and dementia: A systematic review. *Vascular Health Risk Management*, *6*, 775-785.

Liner, K.J., Hall, C.D. & Robertson, K.R. (2008). Effects of antiretroviral therapy on cognitive impairment. *Current HIV/AIDS Report, 5*, 64-71.

Lodico, M. A. & DiClemente, R. J. (1994). The association between childhood sexual abuse and prevalence of HIV-related risk behaviors. *Clinical Pediatrics*, *33*, 498-502.

Lopez, O. L., Becker, J. T., Dew, M. A., Banks, G., Dorst, S. K., & McNeil, M. (1994). Speech motor control disorder after HIV infection. *Neurology*, *44*, 2187-2189.

Lopez, O.L., Jagust, W.J. Dulberg, C., Becker, J.T., DeKosky, S.T. & Fitzpatrick, A. (2003). Risk factors for mild cognitive impairment in the cardiovascular health study cognition study. *Arch Neurol*, *60*, 1394-1399.

Lopez, O.L., Wess, J., Sanchez, J., Dew, M.A. & Becker, J.T. (1999). Neurological characteristics of HIV infected men and women seeking primary medical care. *European Journal of Neurology*, *6*(2), 205-209.

Lorenz, M. W., Stephan, C., Harmjanz, A., Staszewski, S., Buehler, A. & Bickel, M. (2008). Both long term HIV infection and highly active antiretroviral therapy are independent risk factors for early carotid atherosclerosis. *Atherosclerosis, 196*(2), 720-726.

Lott, B. (2002). Cognitive and behavioral distancing from the poor. *American Psychologist, 57*, 100-110.

Lu, P. & Scicutella, A. (2009). Depression in MCI patients predicts progression to Alzheimer's disease. *Neurology, 72*, 2115-2121.

Luukinen, H., Koski, K., Honkanen, R. & Kivela, S.L. (1995). Incidence of injury-causing falls among older adults by place of residence: a population-based study. *Journal of American Geriatric Society*, *43*, 871-876.

Luukinen, H. H., Jokelainen, J. J., Kervinen, K. K., Kesäniemi, Y. A., Winqvist, S. S. & Hillbom, M. M. (2008). Risk of dementia associated with the ApoE Ɛ4 allele and falls causing head injury without explicit traumatic brain injury. *Acta Neurologica Scandinavica*, *118*(3), 153-158.

Luukinen, H. H., Viramo, P. P., Herala, M. M., Kervinen, K. K., Kesäniemi, Y. A., Savola, O. O., & ... Hillbom, M. M. (2005). Fall-related brain injuries and the risk of dementia in elderly people: A population-based study. *European Journal of Neurology*, *12*(2), 86-92.

Macklin, M. L., Metzger, L. J., McNally, R. J., Litz, B. T., Lasko, N. B., Orr, S. P., & Pitman, R. K. (1998). Lower precombat intelligence is a risk factor for posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology*, *68*, 323-326.

Magalhaes, M. G., Greenberg, B., Hansen, H., Odont, C. & Glick, M. (2007). Comorbidities in older patients with HIV: A retrospective study. *JADA, 138*, 1468-1475.

Mahurin, R. K., Espino, D. V. & Holifield, E. B. (1992). Mental status testing in elderly Hispanic populations: Special concerns. *Psychopharmacology Bulletin*, *28*(4), 391-399.

Mamidi, A., DeSimone, J. A., & Pomerantz, R. J. (2002). Central nervous system infections in individuals with HIV-1 infection. *Journal of Neurovirology, 8*, 158-167.

Manly, J.J. (2006). Deconstructing race and ethnicity: Implications for measurement of health outcomes. *Medical Care*, *44*(11), 10-16.

Manly, J.J., Jacobs, D.M., Sano, M., Bell, K., Merchant, C.A., Small, S.A., & Stern, Y. (1998). African American acculturation and neuropsychological test performance among nondemented community elders. *Journal of the International Neuropsychological Society*, *4*, 77.

Manly, J.J., Jacobs, D.M., Touradji, P., Small, S.A., & Stern, Y. (2002). Reading Level attenuates differences in neuropsychological test performance between African American and White elders. *Journal of the International Neuropsychological Society*, *8*, 341-348.

Marcopulos, B.A., Mclain, C.A. & Giuliano, A.J. (1997). Cognitive impairment or inadequate norms? A study of healthy, rural, older adults with limited education. *Clinical Neuropsychologist*, *11*, 1111-1131.

Marcotte, T. D., Lazzaretto, D., Scott, J. C., Roberts, E., Woods, S. P., & Letendre, S. (2006). Visual attention deficits are associated with driving accidents in cognitively-impaired HIV-infected individuals. *Journal of Clinical and Experimental Neuropsychology, 28,* 13-28.

Martin, E. M., Pitrak, D. L., Novak, R. M., Pursell, K. J., & Mullane, K. M. (1999). Reaction times are faster in HIV-seropositive patients on antiretroviral therapy: A preliminary report. *Journal of Clinical and Experimental Neuropsychology*, *21*, 730-735.

Martin, E. M., Pitrak, D. L., Robertson, L. C., Novak, R. M., Mullane, K. M., & Pursell, K. J. (1995). Global-local analysis in HIV-1 infection. *Neuropsychology, 9,* 102-109.

Mary-Krause, M., Cotte, L., Simon, A., Partisani, M., Costagliola, D. (2003). Clinical epidemiology group from the French hospital database. Increased risk of

myocardial infarction with duration of protease inhibitor therapy in HIV-infected men. *AIDS*, *17* (17), 2479-2486.

Mason, C.J., Kitsiripornchai, S. & Markowitz, L. E. (1998). Nationwide surveillance of HIV-1 prevalence and subtype in young Thai men. *Journal of Acquired Immune Deficiency Syndrome Human Retrovirus, 19*, 165-73.

Mast, B.T., Fitzgerald, J., Steinberg, J., MacNeill, S.E., Lichtenberg, P.A**.** (2001). Effective screening for Alzheimer's disease among older African Americans. *Clinical Neuropsychologist, 15,* 196-202.

Mathew, M. & Bhat, J. (2007). Voice disorders in HIV-infected individuals: A preliminary study. *International Journal of STD & AIDS, 18*, 732-735.

Mawar, N., Sahay, S., Pandit, A. & Mahajan, U. (2005). The third phase of HIV pandemic: Social consequences of HIV/AIDS stigma and discrimination and future needs. *Indian Journal of Medical Research*, *122*, 471-484.

Mayberg, H.S. (1997). Limbic-cortical dysregulation: A proposed model of depression. *Journal of Neuropsychological Clinical Neuroscience*, *9*, 471-481.

Mayeux, R., Ottman, R. & Maestre, G. (1995). Synergistic effect of traumatic head injury and apolipoprotein-epsilon 4 in patients with Alzheimers disease. *Neurology, 45*, 555-557.

McAllister-Williams, R.H., Ferrier, I.N. & Young, A.H. (1998). Mood and neuropsychological function in depression: The role of corticosteroids and serotonin. *Psychological Medicine, 28*, 573-584.

McArthur, J. C. (2004). HIV dementia: An evolving disease. *Journal of Neuroimmunology*, *157*, 3-10.

McArthur, J. C. Haughey, N., Gartner, S., Conant, K., Pardo, C, Nath, A., & Sacktor, N. (2003). Human immunodeficiency virus–associated dementia: An evolving disease. *Journal of NeuroVirology*, *9*, 205-221.

McCabe, P., Sheard, C. & Code, C. (2007). Pragmatic skills in people with HIV/AIDS. *Disability and Rehabilitation*, 29, 1251-1260.

McCain, N. L., Munjas, B. A., Munro, C. L., Elswick, R. K., Robins, J. L., Ferrerira-Gonzalez, A., Baliko, B., Kaplowitz, L. G., Fisher, E. J., Garrett, C. T., Brigle, K. E., Kendall, L. C., Lucas, V. & Cochran, K. L. (2003). Effects of stress management on PNI-based outcomes in persons with HIV disease. *Research Nurses Health, 26,* 102-117.

Mcdermott, R. (1998). Adolescent HIV prevention and intervention: A prospect theory analysis. *Psychology, Health & Medicine*, *3*(4).

McDowell, T.L. & Serovich, J.M. (2007). The effect of perceived and actual social support on the mental health of HIV-positive persons. *AIDS Care*, *19*, 1223-1229.

McGuire D. (2003). Neurologic manifestations of HIV. In *HIV in site knowledge base.* San Francisco: UCSF Center for HIV Information.

McMurtray, A., Nakamoto, B., Shikuma, C. & Valcour, V. (2008). Cortical atrophy and white matter hyperintensities in HIV: The Hawaii aging with HIV cohort study. *Journal of Stroke and Cerebrovascular Disease, 17*(4), 212-217.

Mega, M. S. & Cummings, J. L. (1994). Frontal-subcortical circuits and neuropsychiatric disorders. *Journal of Neuropsychiatry and Clinical Neuroscience, 6,* 358-370.

Mehta, K.M. Yaffe, K., Pérez-Stable, E.J., Stewart, A., Barnes, P., Kurland, B.F. & Miller, B. L. (2003). Race/ethnic differences in Alzheimer disease survival in US Alzheimer disease centers. *Journal of NeuroVirology*, *9*, 205-221.

Mehta, K.M., Simonsick, E.M. & Rooks, R. (2004). Black and white differences in cognitive function test scores: What explains the difference? *Journal of American Geriatric Society*, *52*, 2120-2127.

Miller, L.C. Murphy, S.T., Clark, L.F., Hamburger, M., & Moore, J. (2004). Hierarchical messages for introducing multiple HIV prevention options: Promise and pitfalls. *AIDS Education and Prevention*, *16* (6), 509-525.

Millikin, C.P., Trepanier, L.L. & Rourke, S. B. (2004). Verbal fluency component analysis in adults with HIV/AIDS. *Journal of Clinical Experimental Neuropsychology*, *26*(7), 933-942.

Miranda, R., Meyerson, L. A., Marx, B. P., & Tucker, P. (2000). Civilian-based posttraumatic stress disorder and physical complaints: an evaluation of depression as a mediator. *Journal of Traumatic Stress*, *15*, 297-301.

Mirsattari, S. M., Berry, M. E., Holden, J. K., Ni, W., Nath, A. & Power, C. (1999). Paroxysmal dyskinesias in patients with HIV infection. *Neurology, 52*, 109-114.

Mohlman, J. Gorenstein, E., Kleber, M. De Jesus, M. Gorman, J., & Papp, L. (2003). Standard and enhanced cognitive-behavior therapy for late-life generalized anxiety disorder: Two pilot investigations. *American Journal of Geriatric Psychiatry*, *11*(1), 24-32.

Moore, A.L., Mocroft, A. & Madge, S. (2001). Gender differences in virologic response to treatment in an HIV positive population: A cohort study. *Journal of Acquired Immune Deficiency Syndrome*, *26*, 159.

Morrison, M. F., Petitto, J. M., Have, T. T. Gettes, D. R., Chiappini, M. S., Weber, A. N., Brinker-Spence, P., Bauer, R. M., Douglas, S. D. & Evans, D. L. (2002). Depressive and anxiety disorders in women with HIV infection. *American Journal of Psychiatry, 159,* 789-796.

Morgan, E. E., Woods, S. P., Childers, M., Marquie-Beck, J., Ellis, R. J. & Grant, I. (2008). Predictive validity of demographically-adjusted normative standards for the HIV Dementia Scale. *Journal of Clinical and Experimental Neuropsychology, 30*, 83-90.

Moskowitz, J. T., & Wrubel, J. (2005). Coping with HIV as a chronic illness: A longitudinal analysis of illness appraisals. *Psychology & Health, 20,* 509-531.

Muller, M., Tang, M. & Schupf, N. (2007). Metabolic syndrome and dementia risk in a multiethnic elderly cohort. *Dementia Geriatriatic Cognitive Disorder*, *24*, 185-192.

Mugavero, M.J., Pence, B.W., Whetten, K., Leserman, J., Swartz, M., Stangl, D. & Thielman, N.M. (2007). Predictors of AIDS-related morbidity and mortality in a southern US cohort. *AIDS Patient Care STDs*, *21*, 681-90.

Mugavero, M., Ostermann, J., Whetten, K., Leserman, J., Swartz, M., Stangl, D., & Thielman, N. (2006). Barriers to antiretroviral adherence: The importance of depression, abuse, and other traumatic events. *AIDS Patient Care and STDs*, *20*(6), 418-428.

Mukhtar, M., Harley, S., Chen, P., BouHamdan, M., Patel, C., Acheampong, E. & Pomerantz, R.J. (2002). Primary isolated human brain microvascular endothelial cells express diverse HIV/SIV-associated chemokine coreceptors and DC-SIGN and L-SIGN. *Virology, 297*, 78-88.

National AIDS Research Institute: Annual Report 2002-2003. NARI, Pune.

Navia, B., Cho, E.S., Petito, C. & Price, R. (1986). The AIDS dementia complex: II neuropathology. *Ann Neurol*, *19*, 525-535.

Neff, J.A. & Crawford, S.L. (1998). The health belief model and HIV risk behaviors: A causal model analysis among Anglos, African Americans and Mexican-Americans. *Ethnicity & Health*, *3*(4).

Nomenclature and research case definitions for neurologic manifestations of human immunodeficiency virus-type 1 (HIV-1) infection. Report of a Working Group of the American Academy of Neurology (AAN) AIDS Task Force. (1991). *Neurology, 41*(6), 778-85.

Norman, P., & Brain, K. (2005). An application of an extended health belief model to the prediction of breast self-examination among women with a family history of breast cancer. *British Journal of Health Psychology, 10*, 1-11.

O'Brien, J.T., Erkinjuntti, T. & Reisberg, B. (2003). Vascular cognitive impairment. *Lancet Neurology, 2*, 89-98.

Olesen, P. J., Schendan, H. E., Amick, M. M. & Cronin-Golomb, A. (2007). HIV infection affects parietal-dependent spatial cognition: Evidence from mental rotation and hierarchical pattern perception. *Behavioral Neuroscience, 121*(6), 1163-1173.

Ortiz-Torres, B., Serrano-García, I., & Torres-Burgos, N. (2000). Subverting culture: Promoting HIV/AIDS prevention among Puerto Rican and Dominican women. *American Journal of Community Psychology*, *28*, 859-881.

Pat-Horenczyk, R., & Brom, D. (2007). The multiple faces of post-traumatic growth. *Applied Psychology: An International Review*, *56*(3), 379-385.

Patterson, T.L., Shaw, W.S., Semple, S.J., Cherner, M., Nannis, E., McCutchan, J.A., Atkinson, J.H. & Grant, I. (1996). Relationship of psychosocial factors to HIV disease progression. *Annals of Behavioral Medicine*, *18*, 30-39.

Peavy, G., Jacobs, D., Salmon, D. P., Butters, N., Delis, D. C. & Taylor, M. (1994). Verbal memory performance of patients with human immunodeficiency virus infection: Evidence of subcortical dysfunction. *Journal of Clinical and Experimental Neuropsychology*, *16*, 508-523.

Pence, B., Reif, S., Whetten, K., Leserman, J., Stangl, D., Swartz, M., Thielman, N. & Mugavero, M. (2007). Minorities, the poor, and survivors of abuse: HIV-infected patients in the US deep south. *South Medical Journal, 100*, 1114-22.

Perry, S. & Jacobsen, P. (1986). Neuropsychiatric manifestations of AIDS-spectrum disorders. *Hospital and Community Psychiatry*, *37*(2), 135-142.

Plassman, B.L., Langa, K.M., Fisher, G.G, Heeringa, S.G., Weir, D.R. & Ofstedal, M.B. (2007). The prevalence of dementia in the United States: The aging, demographics, and memory study. *Neuroepidemiology*, *29*, 125-132.

Portegies, P., Enting, R.H. & de Gans, J. (1993). Presentation and course of AIDS dementia complex: 10 years of follow-up in Amsterdam, The Netherlands. *AIDS, 7*, 669-675.

Postle, B. R. (2006). Working memory as an emergent property of the mind and brain. *Neuroscience, 139,* 23-38.

Poston, C. W., Craine, M., & Atkinson, D. R. (1991). Counselor dissimilarity confrontation, client cultural mistrust and willingness to self disclose. *Journal of Multicultural Counseling and Development, 19*, 65-73.

Potter, G.G., Plassman, B.L., Burke, J.R., Kabeto, M.U., Langa, K.M., Llewellyn, D. J., Rogers, M.A. & Steffens, D.C.(2009). Cognitive performance and informant reports in the diagnosis of cognitive impairment and dementia in African Americans and whites. *Alzheimer's and Dementia*, *5*(6), 445-453.

Powderly, W. G. (2002). Sorting through confusing messages: The Art of HAART. *Journal of Acquired Immune Deficiency Syndromes*, *31*(1), S3-S9.

Power, C., Selnes, O. A., Grim, J. A., & McArthur, J. (1995). HIV Dementia Scale: A rapid screening test. *Journal of Acquired Immune Deficiency Syndromes and Human Retrovirology*, *8*, 273-278.

Puisieux, F., Bulckaen, H., Fuachais, A.L., Granier, F. & Dewailly, P. (2000). Ambulatory blood pressure monitoring and post prandial hypotension in elderly persons with falls or syncope. *Journal of Gerontology and Biological Medical Science, 55*, 535-540.

Qiu, C., Strauss, E. & Fastbom, J. (2003). Low blood pressure and risk of dementia in the Kungsholmen project. *Arch Neurol*, *60*, 223-228.

Radkowski, M., Wilkinson, J. & Nowicki, M. (2002). Search for hepatitis C virus negative-strand RNA sequences and analysis of viral sequences in the central nervous system: Evidence of replication. *Journal Virology, 76*, 600-608.

Ramani, S., Durvasula, E. N., Miller, H., Myers, F., & Wyatt, G. E. (2001). Predictors of neuropsychological performance in HIV positive women. *Journal of Clinical and Experimental Neuropsychology, 23*(2), 149-163.

Reger, M., Welsh, R., Razani, J., Martin, D.J. & Boone, K.B. A meta-analysis of the neuropsychological sequelae of HIV infection. *Journal of International Neuropsychological Society, 8*, 410-424.

Rhodes, S.D. (2004). Hookups of health promotion? An exploratory study of a chat room-based HIV prevention intervention for men who have sex with men. *AIDS Education and Prevention*, *16*(4), 315-327.

Rippeth, J. D., Heaton, R. K., Carey, C. L., Marcotte, T. D., Moore, D. J. & Gonzalez, R. (2004). Methamphetamine dependence increases risk of neuropsychological impairment in HIV infected persons. *Journal of the International Neuropsychological Society,* 10, 1-14.

Robertson, K. R., Smurzynski, M., Parsons, T. D., Wu, K., Bosch, R. J. & Wu, J. (2007). The prevalence and incidence of neurocognitive impairment in the HAART Era. *AIDS, 21*, 1915-1921.

Robertson, K., Liner, J. & Heaton, R. (2009). Neuropsychological assessment of HIV-infected populations in international settings**.** *Neuropsychological Rev*iew, *19*(2)**,** 232-249.

Robinson-Papp, J., Byrd, D., Mindt, R. R., Oden, N.L., Simpson, D. M. & Morgello, S. (2009). Motor function and human immunodeficiency virus-associated cognitive impairment in a highly active antiretroviral therapy-era cohort. *Archives of Neurology*, *65*(8), 1096.

Rohit, M., Levine, A., Hinkin, C., Abramyan, S., Saxton, E., Valdes-Sueiras, M. & Singer, E. (2007). Education correction using years in school or reading grade-level equivalent? Comparing the accuracy of two methods in diagnosing HIV-associated neurocognitive impairment. *Journal of the International Neuropsychological Society*, *13*(3), 462-470.

Rosenstock, I.M. (1974). Historical origins of the health belief model. *Health Education Monographs, 2,* 328-334.

Rosenstock, I.M., Strecher, V.J. and Becker, M.H. (1988). Social learning and the health belief model. *Health Education Quarterly, 15*(2*)*, 175-183.

Rotheram-Borus, M. J., Murphy, D. A., Reid, H., & Coleman, C. (1996). Correlates of emotional distress among HIV/ youth: health status, stress, and personal resources. *Annals of Behavioral Medicine*, *18*, 16-23.

Rourke, S. B., Halman, M. H. & Bassel, C. (1999). Neurocognitive complaints in HIV-infection and their relationship to depressive symptoms and neuropsychological functioning. *Journal of Clinical and Experimental Neuropsychology*, *21*, 737-756.

Ryan, E.L., Baird, R., Mindt, M.R., Byrd, D., Monzones, J., & Morgello, S. (2005). Neuropsychological impairment in racial/ethnic minorities with HIV infection and low literacy levels: Effects of education and reading level in participant

characterization. *Journal of the International Neuropsychological Society*, *11*, 889-898.

Sacktor, N. C. (2005). The International HIV Dementia Scale: A new rapid screening test for HIV dementia, *AIDS*, *19*(13), 1367-1374.

Sacktor, N., Lyles, R.H., Skolasky, R., Kleeberger, C., Selnes, O.A. & Miller, E.N. (2001). HIV-associated neurologic disease incidence changes: Multicenter AIDS cohort study, 1990-1998. *Neurology*, *56*(2), 257-60.

Sacktor, N., Skolasky, R., Selnes, O. A., Watters, M., Poff, P. & Shiramizu, B. (2007). Neuropsychological test profile differences between young and old human immunodeficiency virus-positive individuals. *Journal of Neurovirology*, *13*, 203-209.

Sahai-Srivastava, S. (2008). Medico-legal issues in HIV/AIDS in the 21st century. *The spectrum of neuro-AIDS disorders: Pathophysiology, diagnosis and treatment*. Washington, DC: ASM Press.

Sayles, J.N., Wong, M.D., Kinsler, J.J., Martins, D. & Cunningham, W.E. (2009). The association of stigma with self-reported access to medical care and antiretroviral therapy adherence in persons living with HIV/AIDS. *Journal of General Internal Medicine, 24*, 1101-1108.

Schmidt, R., Schmidt, H. & Fazekas, F. (2000). Vascular risk factors in dementia. *Journal of Neurology*, *247*, 81-87.

Schnurr, P. P., Spiro, A., & Paris, A. H. (2000). Physician-diagnosed medical disorders in relation to PTSD symptoms in older male military veterans. *Health Psychology*, *19*, 91-97.

Serovich, J.M., Kimberly, J.A., Mosack, K.E., & Lewis, T.L. (2001). The role of family and friend support in reducing emotional distress among HIV-positive women. *AIDS Care*, *13*, 335-341.

Shadlen, M.F., Siscovick, D., Fitzpatrick, A.L., Dulberg, C., Kuller, L.H. & Jackson, S. (2006). Education, cognitive test scores, and black-white differences in dementia risk. *Journal of American Geriatriatric Society*, *54*, 898-905.

Shannon, C., Douse, K., McCusker, C., Feeney, L., Barrett, S. & Mulholland, C. (2009). The association between childhood trauma and memory functioning in schizophrenia. *Schizophrenia Bulletin, 10*.

Shaw, B.A. & Krause, N. (2001). Exploring race variations in aging and personal control. *Journals of Gerontology: Series B: Psychological Sciences and Social Sciences, 56,* 119-124.

Seigel, D.M., Klein, D.I., Roghmann, K.J. (1999). Sexual behavior, contraception, and risk among college students. *Journal of Adolescent Health*, *25*(5), 336-343.

Simoni, J. M., Pantalone, D. W., Plummer, M. D. & Huang, B. (2007). A randomized controlled trial of a peer support intervention targeting antiretroviral medication adherence and depressive symptomatology in HIV-positive men and women. *Health Psychology*, *26,* 488-495.

Simpson, D. M. & Tagliati, M. (1994). Neurologic manifestations of HIV infection. *Annals of Internal Medicine, 121*(10), 769-785.

Smith, M. Y., Egert, J.,Winkel, G., & Jacobson, J. (2002). The impact of PTSD on pain experience in persons with HIV/AIDS. *Pain*, *98*, 9-17.

Smith, C. A., van Gorp, W. G., Ryan, E. R., Ferrando, S. J., & Rabkin, J. (2003). Screening subtle HIV related cognitive dysfunction: The clinical utility of the HIV Dementia Scale. *Journal of AIDS, 33*, 116-118.

Snowden, L. & Pingitore, D.P. (2002). Frequency and scope of mental health services to African-Americans in primary care. *Mental Health Services Research, 4*(3), 123-130.

Snider, W. D., Simpson, D. M., Nielsen, S., Gold, J. W., Metroka, C. E., & Posner, J. B. (1983). Neurological complications of acquired immune deficiency syndrome: analysis of 50 patients. *Annals of Neurology, 14*, 403-418.

Sprouse, D.S., Ogletree, S.L., Kern, R.M., Granville, H.G. & Comsudes, M.M. (2005). An Adlerian model for alliance building. *Journal of Individual Psychology*, *61*(2).

Stanley, M. M., Dienfenbach, G. J., & Hopko, D. R. (2004). Cognitive behavioral treatment for older adults with generalized anxiety disorder: A therapist manual for primary care settings. *Behavior Modification*, *28*(1), 73-117.

Starkstein, S.E. & Jorge, R. (2005). Dementia after traumatic brain injury. *Int Psychogeriatr, 17*, S93-S107.

Stein, J.H., Klein, M.A. & Bellehumeur, J.L. (2001). Use of human immunodeficiency virus-1 protease inhibitors is associated with atherogenic lipoprotein changes and endothelial dysfunction. *Circulation, 104*, 257-262.

Stern, Y., Albert, S. & Tang, M.X. (1999). Rate of memory decline in AD is related to education and occupation: Cognitive reserve. *Neurology*, *53*, 1942-1947.

Stern, Y., McDermott, M.P., Albert, S., Palumbo, D., Selnes, O.A. & McArthur, J. (2001). Factors associated with incident human immunodeficiency virus-dementia. *Arch Neurol*ogy, *58*(3), 473-479.

Stiles, B. L., & Kaplan, H. B. (2004). Factors influencing change behavior: risk reduction for HIV infection. *Behavior and Personality, 32*(6)*,* 511-534.

Streeck-Fischer, A., & van der Kolk, B. (2000). Down will come baby, cradle and all: Diagnostic and therapeutic implications of chronic trauma on child development. *Australia and New Zealand Journal of Psychiatry*, *34*, 903-918.

Stoff, D. M. (2004). Mental health research in HIV/AIDS and aging: Problems and prospects. *Aids, 18*(1), 3-10.

Sullivan, K.A & Waugh, D.J, (2007). Toward the development of the cerebrovascular attitudes and beliefs scale (CABS): A measure of stroke-related health beliefs. *Topics in Stroke Rehabilitation,* 41-51.

Sullivan, K.A., White, K.M., Young, R.M., Scott, C.J. & Mulgrew, K. (2008). Developing a stroke intervention program: What do people at risk of stroke want? *Patient Education and Counseling,* 126-134.

Sundquist, J., Winkleby, M.A. & Pudaric , S. (2001). Cardiovascular disease risk factors among older Black, Mexican-American, and White Women and Men: An analysis of NHANES III, 1988-1994. Third national Health and Nutrition Examination Survey. *JAGS, 49*, 109-116.

Swendeman, D., Rotheram-Borus, M.J., Comulada, S., Weiss, R., Ramos, M.E. (2006) Predictors of internalized HIV stigma among young people living with HIV. *Health Psychology, 25*, 501-509.

Tang, M.X., Cross, P., Andrews, H., Jacobs, D.M., Small, S., Bell, K.,Merchant, C., Lantigua, R., Costa, R., Stern,Y., & Mayeux, R. (2001). Incidence of AD in African-Americans, Caribbean Hispanics, and Caucasians in northern Manhattan. *Neurology*, *56*, 49-56.

Taylor, S.E. (1999). *Health psychology* (4th ed., pp. 64-68, 438-452). California: McGraw-Hill.

Taylor, J.Y., Washington, O. G. M., Artinian, N.T. & Lichtenberg, P. (2008). Relationship between depression and specific health indicators among hypertensive African American parents and Grandparents. *Prog Cardiovasc Nurs*, *23*(2), 68-78.

Tedaldi, E.M., Baker, R.K., Moorman, A.C., Alzola, C.F., Furhrer, J. & McCabe, R.E. (2003). Influence of coinfection with hepatitis C virus on morbidity and mortality due to human immunodeficiency virus infection in the era of highly active antiretroviral therapy. *Clinical Infectious Diseases, 36*(3), 363-367.

Tello, M., Yeh, H.C., Keller, J.M., Beach, M.C., Anderson, J.R. & Moore, R.D. (2008). HIV Women's Health: A study of gynecologic healthcare service utilization in a U.S. urban HIV clinic population. *Journal of Womens Health*, *17*, 1609-1614.

Thompson, P. M., Dutton, R. A., Hayashi, K. M., Toga, A. W., Lopez, O. L. & Aizenstein, H. J. (2005). Thinning of the cerebral cortex visualized in HIV/AIDS reflects CD4+ T lymphocyte decline. *Proceedings of the National Academy of Sciences of the United States of America, 102*, 15647-15652.

Tien, P.C., Schneider, M.F., Cole, S.R., Levine, A.M., Cohen, M. & DeHovitz, J. (2007). Antiretroviral therapy exposure and incidence of diabetes mellitus in the Women's Interagency HIV study. *AIDS, 21*, 1739-1745.

Tozzi, V., Balestra, P., Galgani, S., Narciso, P., Ferri, F. & Sebastiani, G. (1999). Positive and sustained effects of highly active antiretroviral therapy on HIV-1-associated neurocognitive impairment. *AIDS*, *13*, 1889-1897.

Treisman, G. J., Angelino, A. F. & Hutton, H. E. (2001). Psychiatric issues in the management of patients with HIV infection. *Journal of the American Medical Association*, *286,* 2857-2864.

Triant, V. A., Lee, H., Hadigan, C. & Grinspoon, S. K. (2007). Increased acute myocardial infarction rates and cardiovascular risk factors among patients with human immunodeficiency virus disease. *Journal of Clinical Endocrinology and Metabolism*, *92*, 2506–2512.

Uchiyama, C. L., Mitrushina, M., Satz, P., & Schall, M. (1996). Direct and indirect effects of demographic, medical, and psychological variables on neuropsychological performance in normal geriatric persons: A structural equation model. *Journal of the International Neuropsychological Society*, *2*, 299-305.

Ullman, S. E., & Brecklin, L. R. (2002). Sexual assault history, PTSD, and mental health service seeking in a national sample of women. *Journal of Community Psychology*, *30*, 1-19.

Ullman, S.E. & Filipas, H.H. (2005). Ethnicity and child sexual abuse experiences of female college students. *Journal of Child Sexual Abuse, 14*, 67-89.

van Doorn, C., Gruber-Baldini, A. L., Zimmerman, S., Richard Hebel, J. J., Port, C. L., Baumgarten, M. & Magaziner, J. (2003). Dementia as a risk factor for falls and fall injuries among nursing home residents. *Journal of the American Geriatrics Society*, *51*(9), 1213-1218.

Valcour, V., Watters, W. R., Williams, A. E., Sacktor, N., McMurtray, A. & Shikuma, C. (2008). Aging exacerbates extrapyramidal motor signs in the era of highly active antiretroviral therapy. *Journal of Neurovirology*, *14*, 362-367.

Varas-Diaz, N., Garcia, I. & Toro-Alfonso, J. (2005). AIDS-related stigma and social interaction: Puerto Ricans living with HIV/AIDS. *Qualitative Health Research, 15*(2), 169-187.

Varas-Díaz, N., Serrano-García, I. & Toro-Alfonso, J. (2004). *Stigma and social difference: HIV/AIDS in Puerto Rico*. San Juan: Ediciones Huracán.

Vittecoq, D., Escaut, L. & Chironi, G. (2003). Coronary heart disease in HIV-infected patients in the highly active antiretroviral treatment era. *AIDS, 17*(1), 70-76.

Volpicelli, J.R., Pettinati, H.M., Monterosso, J. & Lipkin, C. (2003). Patient attitudes toward treatment predict attendance in clinical pharmacotherapy trials of alcohol and drug treatment. *American Journal of Addiction*, *12*, 324-335.

Vyavaharkar, M., Moneyham, L., Tavakoli, A., Phillips, K.D., Murdough, C. & Jackson, K., (2007). Social support, coping, and medication adherence among HIV-positive women with depression living in rural areas of the southeastern United States. *AIDS Patient Care and STDs*, *21*(9), 667-680.

Walsh, F. (2002). Bouncing forward: Resilience in the aftermath of September 11. *Family Processes*, *41*, 34-36.

Wdowik, M. J., Kendall, P. A., Harris, M. A. & Auld, G. (2001). Expanded health belief model predicts diabetes self-management in college students. *Journal of Nutrition Education*, *33*, 17-23.

Weiss, J.J., Osorio, G., Ryan, E., Marcus, S.M. & Fishbein, D.A. (2010). Prevalence and patient awareness of medical comorbidities in an urban AIDS Clinic. *AIDS Patient Care STDS*, *24*(1), 39-48.

Whetten, K., Leserman, J., Lowe, K., Stangl, D., Thielman, N., Swartz, M., Hanisch, L. & Van Scoyoc, L. (2006). Prevalence of childhood sexual abuse and physical trauma in a southern HIV positive sample from the deep South. *American Journal of Public Health, 96*, 970-973.

Whetten, K., Reif, S., Whetten, R. & Murphy-McMillan, L. K. (2008). Trauma, mental health, distrust, and stigma among HIV-positive persons: Implications for effective care. *Psychosomatic Medicine*, *70*, 531-538.

Whitmer, R., Gunderson, E. & Barratt-Connor. E. (2005). Obesity in middle age and future risk of dementia. a 27 year longitudinal population based study. *BMJ*, *330*, 1360-1364.

Woods, S.P., Moore, D.J., Weber, E. & Grant, I. (2009). Cognitive neuropsychology of HIV-associated neurocognitive disorders. *Neuropsychol Rev, 19*(2), 152-168.

Woods, S. P., Carey, C. L., Moran, L. M., Dawson, M. S., Letendre, S. L. & Grant, I. (2007). Frequency and predictors of self-reported prospective memory complaints in individuals infected with HIV. *Archives of Clinical Neuropsychology*, *22*, 187-195.

Woods, S. P., Carey, C. L., Tröster, A. I. & Grant, I. (2005). Action (verb) Generation in HIV-1 Infection. *Neuropsychologia, 43*, 1144-1151.

Woods, S. P., Conover, E., Rippeth, J. D., Carey, C. L., Gonzalez, R. & Marcotte, T. D. (2004). Qualitative aspects of verbal fluency in HIV-associated dementia: A deficit in rule-guided lexical-semantic search processes? *Neuropsychologia, 42*, 801-809.

Woods, S. P., Ludicello, J. E., Moran, L. M., Carey, C. L., Dawson, M. S. & Grant, I. (2008). HIV-associated prospective memory impairment increases risk of dependence in everyday functioning. *Neuropsychology*, *22*, 110-117.

Woods, S.P., Moore, D.J., Weber, E. & Grant, I. (2009). Cognitive neuropsychology of HIV-associated neurocognitive disorders. *Neuropsychology Review, 19*(2), 152-168.

Wu, E., El-Bassel, N., Witte, S.S., Gilbert, L., & Chang, M. (2003). Intimate partner violence and HIV risk among urban minority women in primary health care settings. *AIDS and Behavior*, *7*, 291-301.

Yaffe, K. 2007. Metabolic syndrome and cognitive disorders: Is the sum greater than its parts? *Alzheimers Disease Associative Disorders*, *21*, 167-171.

Yaffe, K., Vittinghoff, E., Lindquist, K., Barnes, D., Covinsky, K. E., Neylan, T., Kluse, M. & Marmar, C. (2010). Posttraumatic stress disorder and risk of dementia among US veterans. *Arch Gen Psychiatry*, *67*(6), 608-613.

Yale University (2001). African Americans have a higher rate of dementia due to strokes. *ScienceDaily*. Retrieved on 8/26/2010 from http://www.sciencedaily.com/releases/2001/06/010605074015.htm

Yehuda, R., Flory, J.D., Southwick, S., Charney, D.S. (2006). Developing an agenda for translational studies of resilience and vulnerability following trauma exposure. *Ann N Y Acad Sci*, *1071*, 379-396.

Yi, J.P., Smith, R.E. & Vitaliano, P.P. (2005). Stress-resilience, illness, and coping: a person-focused investigation of young women athletes. *Journal of Behavioral Medicine*, *28*, 257-265.

Zierler, S., Cunningham, W. E., Andersen, R., Shapiro, M.F., Nakazono, T., Morton, S., Crystal, S., Stein, M., Turner, B., St Clair, P. & Bozzette, S. A. (2000). Violence victimization after HIV infection in a US probability sample of adult patients in primary care. *American Journal of Public Health*, *90*, 208-15.

Zrinyi, M. & Horvath, T. (2003). Impact of satisfaction, nurse-patient interactions and perceived benefits on health behaviors following cardiac event. *European Journal of Cardiovascular Nursing*, *2,* 159-166.