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Cardiovascular Risk Factor Knowledge, Risk Perception, and Actual Risk in HIV-Infected Patients

A Dissertation Presented

by

Patricia A. Cioe

Submitted to the Graduate School of Nursing
University of Massachusetts Worcester
in partial fulfillment of the requirements for the degree of

Doctor of Philosophy

Nursing

May 2012

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University of Massachusetts Worcester

Graduate School of Nursing

Cardiovascular Risk Factor Knowledge, Risk Perception and Actual Risk in HIV-infected Patients

A Dissertation Presented

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DEDICATION

This work is dedicated to my wonderful, supportive and loving family—

—to my husband, George, you provide me with endless support, and love me in all that I do. I promise not to go back to school again!

—to my parents, Joseph and Elizabeth, you give me unconditional love. Your support and belief in me have been the root of my self-confidence and, ultimately, my successes in life;

—to my two sons, Eric and Scott, you bring me joy! You are truly my greatest achievements. I love you both so much and am always so proud of you.

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—Dr. Carol Bova, my committee chair, whose mentorship, encouragement, and support kept me moving in the right direction,

—Dr. Sybil Crawford, my statistician, whose expertise and clear direction guided me and challenged me to think critically,

—Dr. Michael Stein, my good friend and colleague, whose keen mind, creative thinking, and enthusiastic support inspire me to do more every day.

I sincerely thank all of you from the bottom of my heart.

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ABSTRACT

Background: Cardiovascular disease (CVD) has emerged as a major cause of morbidity and mortality in HIV-infected adults. Research in noninfected populations suggests that knowledge of CVD risk factors significantly influences perception of risk. Understanding the level of risk factor knowledge and risk perception can inform the development of innovative interventions to reduce risk. The purpose of this study was to describe cardiovascular risk factor knowledge and risk perception in a cohort of HIV-infected adults.

Specific aims included (a) describing the estimated risk of CVD, the perceived risk of CVD, and the level of CVD risk factor knowledge; (b) describing the relationship between estimated and perceived risk, and (c) examining the influence of risk factor knowledge on perceived risk of CVD. The Health Belief Model was the theoretical framework that guided the study.

Methods: A prospective observational cohort; cross-sectional design. A convenience sample of 130 HIV-infected adults was recruited from two hospital-based HIV clinics. Each participant had one study visit in which all data were collected by direct interview.

Results: Mean age of enrollees was 48 years (SD 8.4); 62% were male; 41.5% White, 32% Black, 23% Hispanic; 56% current smokers; mean years since HIV diagnosis were 14.7; mean BMI 27 (SD 5.5); 48.5% had prehypertension. Higher scores on the Heart Disease Fact Questionnaire indicate a higher degree of knowledge. In this sample, the Mean was 19, (S.D. 3.5; range 6–25), indicating a fair degree of knowledge.

Estimated and perceived risk were significantly, though weakly, correlated r (126) = .24, p = .01. Controlling for age, risk factor knowledge was not predictive of perceived risk (F [1,117] = 0.13, p > .05)

Conclusions: HIV-infected adults are at increased risk for cardiovascular disease. Traditional CVD risk factors such as smoking, prehypertension, and being overweight are highly prevalent. Despite having a fair level of risk factor knowledge, knowledge did not influence perception of risk for CVD. Research to improve risk perception and to develop innovative interventions that reduce CVD risk is needed for this population.

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CHAPTER 1

State of the Science

Advances in the medical treatment of persons infected with HIV over the past 25 years have led to an increased lifespan for HIV-infected individuals. In fact, recent studies show that mortality rates for HIV-infected persons now mirror that of the general population (Bhaskaran et al., 2008; Lewden et al., 2007). Cardiovascular disease (CVD) is the leading cause of death in the United States (American Heart Association, n.d.) and over the past 7 years, CVD has emerged as a major cause of morbidity and mortality in adults with HIV-infection (Gill et al., 2010; Grinspoon, 2005; Lipshultz, Fisher, Lai, & Miller, 2003; Sackoff, Hanna, Pfeiffer, & Torian, 2006). A growing body of evidence suggests that there is an increased rate of acute myocardial infarction (AMI) in HIV-infected persons (Currier et al., 2003; Friis-Moller, et al., 2007; Triant, Lee, Hadigan, & Grinspoon, 2007). One-third of HIV-infected adults have a greater than 10% risk of having a cardiac event within 10 years (Hadigan, et al., 2003). A cross-sectional study of over 700 participants found that compared to HIV-negative age- and gender- matched controls, HIV-infected participants were twice as likely to have a high predicted 10-year risk (>20%) of AMI (Bergersen, Sandvik, Bruun, & Tonstad, 2004). AMI rates per 1,000 person-years were significantly higher in HIV-infected participants compared to matched controls (11.13 vs. 6.98), and the relative risk was 75% higher for those infected with HIV (Triant, et al., 2007). In addition, the rate of AMI by age group was significantly higher at each age for those infected with HIV when compared to HIV-negative controls. Lastly, in a retrospective cohort analysis in New York City, CVD accounted for 23.8% of all non-HIV-related deaths in HIV-infected persons (Sackoff, et al., 2006).

The increased incidence of AMI and increased prevalence of CVD in HIV-infected persons is multifactorial. Recent reports suggest that persons with HIV infection have a higher prevalence and degree of premature coronary atherosclerosis compared to non-HIV-infected counterparts (Grunfeld et al., 2009; Guaraldi et al., 2009; Hsue et al., 2004; Kingsley et al., 2008; Lo et al., 2010; Van Vonderen et al., 2009). Traditional risk factors for CVD, such as smoking, are more prevalent in this population (Cockerham et al., 2010; Friis-Moller et al., 2003; Glass et al., 2006; Kaplan et al., 2007) and may contribute to the elevated rate of AMI. Comorbidities, such as hypertension, diabetes, dyslipidemia, and obesity, also increase CVD risk. In one cross-sectional study of HIV-infected adults, more than 40% of men and 60% of women met criteria for being overweight or obese with a body mass index of greater than 25 (Kaplan et al., 2007). Importantly, however, the traditional risk factors did not completely account for the increased rate of coronary events (Lipshultz et al., 2003; Sterne et al., 2007). Chronic inflammation related to HIV viral replication and metabolic changes associated with antiretroviral therapy may confer additional risk (Hadigan et al., 2003; Kuller et al., 2008).

Research in non-HIV-infected populations suggests that knowledge of cardiovascular risk factors significantly influences perception of risk (Choi, Rankin, Stewart, & Oka, 2008; Christian, Mochari, & Mosca, 2005). However, studies in non-HIV-infected women demonstrate that women are often unaware of CVD risk factors (Hart, 2005; Lange et al., 2009; Oliver-McNeil & Artinian, 2002; Pregler et al., 2009; Thanavaro, Thanavaro, & Delicath, 2010; Wu, 2007) and subsequently underestimate their risk (Choi et al., 2008; Christian et al., 2005; Hart, 2005). Few studies have explored

CV risk factor knowledge or risk perception among HIV-infected adults. Most studies have focused on the prevalence of CVD, the actual risk of AMI, and the proposed mechanisms of CVD; however, to date the assessment of risk perception and knowledge of CVD risk factors has not been studied in the HIV-infected population. The purpose of this study was to describe cardiovascular risk factor knowledge and CVD risk perception in a cohort of HIV-infected adults. The primary aims of this study were the following:

1. To describe (a) the estimated risk of cardiovascular disease, using the Framingham Risk Assessment Tool, (b) the perceived risk of cardiovascular disease, and (c) the level of cardiovascular risk factor knowledge in HIV-infected adults;
2. To describe the relationship between estimated and perceived risk of CVD in HIV-infected adults;
3. To examine the influence of cardiovascular risk factor knowledge on perceived risk of cardiovascular disease in a sample of HIV-infected adults.

Background and Significance

HIV and cardiovascular disease. In the mid-1990s, with the approval of protease inhibitors and non-nucleoside reverse transcriptase inhibitors (NNRTIs), triple drug therapy became the standard of care for the treatment of HIV/AIDS. This development lead to maximal virologic suppression, and following 1996, mortality due to HIV infection decreased dramatically (Bozzette et al., 2008; Sackoff, et al., 2006) However, case reports of myocardial infarction in young HIV-infected patients caused alarm and spurred investigation (Currier et al., 2008).

Beginning in 2003, several published reports noted that there was an increased rate of myocardial infarction in HIV-infected individuals, possibly related to antiretroviral therapy. Fris-Moller et al. (2003) published initial data from the Data Collection on Adverse Events of Anti-HIV Drugs (D:A:D) Study in the *New England Journal of Medicine* (Friis-Moller et al., 2003). This prospective, longitudinal observational cohort (N = 23,468) conducted across three continents was designed to determine if exposure to antiretroviral therapy (ART) was associated with an increased risk of AMI. It was found that ART was associated with a 26% increase in the rate of AMI per year of exposure ($p<0.001$).

Other studies had similar findings. Crum et al. (2006), in a retrospective review of over 4,000 patients, found that non-HIV-related deaths increased from 8% to 32% from the pre-HAART (highly active antiretroviral therapy) to the late-HAART era. In patients with a CD4 rate greater than 200, cardiac disease accounted for 13% of deaths (Crum, et al., 2006). In a population-based retrospective analysis of death certificates (N = 68,669) of HIV/AIDS patients between 1999 and 2004 in New York City, the rate of HIV-related deaths decreased overall by 54% ($p<0.001$); however non-HIV-related deaths increased by 32% ($p = 0.015$) during this same period, and cardiovascular disease accounted for over 23% of the non-HIV-related deaths (Sackoff, et al., 2006).

A retrospective analysis comparing HIV-infected (n = 3851) and non-HIV-infected individuals (N = 1,044,589) found statistically significant differences in the rate of AMI in these two populations (Triant, et al., 2007). The rate of myocardial infarction (MI) was 11.13 per 1,000 person-years for HIV-infected individuals compared to 6.98 per 1,000 person-years for non-HIV-infected. This difference reflects a relative risk for

AMI related to HIV infection of 1.75 ($p < .001$). These reports further demonstrated the changing paradigm of HIV morbidity and mortality and the increased risk of myocardial events related to HIV infection and/or its treatment. Noting the increased incidence of AMI, investigators began to focus on the prediction of AMI risk using the Framingham Risk Score.

HIV infection and cardiovascular risk. Several cross-sectional studies demonstrated that HIV-infected individuals had significant rates of 10-year predicted risk for myocardial infarction (MI; Wilson, et al., 1998). Using the standard risk categories from the Framingham study, moderate risk was defined as 10–20% risk, and high risk was defined as greater than a 20% risk over a 10-year period. Across studies, it was found that 14–33% of HIV-infected individuals met criteria for moderate risk of MI (Glass, et al., 2006; Hadigan, et al., 2003; Sayler, Lyon, Settle, Elswick, & Rackley, 2006).

Three studies compared the rate of MI risk in HIV-infected and non-HIV-infected individuals. Saves et al. (2003) found that the risk for MI was higher among HIV-infected men (RR 1.20) and HIV-infected women (RR 1.59) as compared to their non-HIV-infected counterparts ($p < .00001$ for both; Saves et al., 2003). Similarly, De Socio et al. (2007) found that, when comparing 403 HIV-infected persons with 200 age- and gender-matched non-HIV-infected controls, the mean 10-year predicted risk in HIV-infected persons was higher; however it was not statistically significant ($p = .32$; De Socio, et al., 2007). Bergersen et al. (2004) reported in a cross-sectional study of 721 subjects that compared to non-HIV-infected age- and gender-matched controls, HIV-infected participants on ART were twice as likely to have a high (>20%) predicted 10-year risk of MI (11.9% vs. 5.3%, $p = .004$; Bergersen et al., 2004).

Law et al. (2006) used the D:A:D study group data to examine the accuracy of the Framingham Risk Equation to predict MI risk in this population (Law et al., 2006) In patients on ART, the rate of observed MI was higher than that predicted by the Framingham equation at each year of follow-up (1–4 years). The Framingham Risk Equation, which is not validated in patients with HIV infection, may underestimate the risk of MI in individuals receiving ART. This finding supports that metabolic changes caused in part by ART increased the observed rate of MI, or that a mechanism related to the medications themselves contributed to the increased rate of MI. The predicted rate of MI in patients not receiving ART was similar to or higher than patients who had received ART for one year or less, further supporting that continued ART increased MI risk (Law et al., 2006).

Low CD4 cell count has been implicated as an independent risk factor for cardiovascular events (Lichtenstein et al., 2010). In the HIV Outpatient Study (HOPS) cohort, a longitudinal analysis of 2005 patients spanning 7 years of follow-up, patients who had a CD4 cell count less than 350 had a higher rate of CVD events when compared to patients with a CD4 greater than 500 (HR 1.58, CI 1.09–2.30), suggesting an attributable risk of approximately 20%. Additionally, traditional CVD risk factors (such as smoking, hypertension, and diabetes) and baseline CD4 count less than 500 were significantly associated with a higher rate of events ($p < .05$). Using arterial stiffness as a marker of subclinical atherosclerotic disease, Ho et al. (2010) found that a nadir CD4 of less than 350 in HIV-infected patients was a predictor of arterial stiffness and was associated with increased cardiovascular risk, independent of other risk factors (Ho et al., 2010).

Traditional risk factors associated with CVD, such as smoking, are more prevalent in HIV-infected persons. In the D:A:D cohort, more than 56% of participants were previous or current smokers (Friis-Moller, et al., 2003). In both the univariate and multivariate models, this conferred a relative risk of 2.08 and 2.17, respectively, for MI ($p = .007$). The Swiss Cohort Study, a 6-year longitudinal study of over 8,000 participants, found that current smoking was the most common cardiovascular risk factor in the cohort, with a reported prevalence of 57% (Glass, et al., 2006). Similarly, Kaplan reported a 35–40% prevalence of current smokers in a cross-sectional study of over 4,000 HIV-infected and non-HIV-infected participants (Kaplan et al., 2007). The difference in smoking rates between HIV-infected men (35.5%) and non-HIV-infected men (28%) was statistically significant ($p < .001$).

Metabolic changes including hyperlipidemia have been implicated as factors associated with elevated cardiovascular risk and increased frequency of cardiac events (Aboud et al., 2010; Ford et al., 2010; Friis-Moller et al., 2003; Kotler, 2008; Saves et al., 2003). Increases in total cholesterol and triglyceride levels were initially reported in 1995 with ritonavir use and continued to be associated with protease inhibitor therapy (Colagreco, 2004). In 2006, Crum et al. compared changes in cause of death from the pre-ART to the late-ART era and reported that, in a cohort of over 4,000 participants, higher cholesterol levels were noted in the late-ART era (Crum et al., 2006). Saves et al. (2003) compared HIV-infected to non-HIV-infected adults and found significantly higher levels of total cholesterol and triglycerides in those infected with HIV. This contributed to a moderately elevated level of risk for CVD (RR 1.20 for men and RR 1.59 for women; $p < .00001$) when using the Framingham Risk Equation variables in this

population (Saves et al., 2003). The Swiss Cohort Study reported higher lipid parameters with 35% of participants having elevated triglycerides at baseline screening using the National Cholesterol Education Program (NCEP) guidelines (Glass et al., 2006).

While lipid changes related to antiretroviral use have been well described, some investigators have found that lipid values were altered in HIV-infected patients even prior to being exposed to antiretroviral therapy (Baker et al., 2010; El-Sadr et al., 2005). One prospective study of 419 patients, which included 20% women, 60% African-American and 10% Latino, found that a higher CD4 lymphocyte count was associated with higher HDL cholesterol concentration, while higher HIV-RNA levels were associated with lower HDL concentrations ($p<0.005$). In addition, a history of an AIDS-defining event was associated with higher total cholesterol, higher VLDL cholesterol, and higher triglyceride levels (El-Sadr et al., 2005).

The D:A:D Study Group (2003) found that 45% of participants had dyslipidemia at baseline (Friis-Moller et al., 2003). Elevated total cholesterol and elevated triglycerides were associated with an increased relative risk of 1.16 and 1.39 ($p = .01$, $p = .003$, respectively) of MI. Both the Swiss Cohort Study (Glass et al., 2006) and the D:A:D study (D:A:D Study Group, 2008) reported that the percentage of patients on lipid lowering therapy increased steadily over time—tripling in each of the 6-year periods reported. However, both studies found that the 10-year risk for CVD remained steady (as in the Swiss Cohort Study) or increased (in the D:A:D Study) despite the increased use of these medications. In fact, the number of participants in the D:A:D Study with high CVD risk increased from 35% to 41% over the same period ($p < .001$).

In a prospective study of 121 HIV-infected patients compared with 273 age- and gender-matched non-HIV-infected controls, Hadigan et al. (2003) reported that the risk of MI was significantly elevated (7.4% vs. 5.3%; p = .002) in those participants with HIV-associated lipodystrophy when compared to controls (Hadigan et al., 2003). Those without lipodystrophy did not have the same finding. Additionally, individuals with primary lipoatrophy had the highest overall 10-year risk estimate (9.2%; p = .04) when compared to those with lipohypertrophy and mixed lipodystrophy. Lipoatrophy generally is defined as a loss of fat in the face or extremities, while lipohypertrophy includes increased abdominal girth or increased chest/breast fat. In this study, self-reported fat redistribution by the study participants was used as the inclusion criteria. Individuals who reported change in more than one area were invited to participate, and body habitus changes were confirmed by inspection during the initial study visit.

A cross-sectional study of 347 patients found that moderate lipoatrophy or moderate lipohypertrophy (OR 4.3; p = .006; OR 2.3; p = .03) was associated with an increased odds ratio of having hypertension (Crane, Grunfeld, Harrington, & Kitahata, 2009). A cross-sectional study of 372 HIV-infected patients on ART found that lipoatrophy (OR 3.82), lipohypertrophy (OR 7.65), and mixed lipodystrophy (OR 4.36) were strongly associated with coronary artery calcium, a marker for subclinical atherosclerosis (Guaraldi et al., 2010). In the general population, visceral adiposity, higher BMI, and increased waist circumference are often associated with increased risk for cardiovascular disease (Ricciardi, Metter, Cavanaugh, Ghambaryan, & Talbot, 2009).

The Strategies for Management of Antiretroviral Therapy (SMART) trial randomized 5,472 participants to receive either continuous ART therapy or episodic ART

guided by the CD4 cell count (El-Sadr et al., 2006). The primary endpoint for this study was development of an opportunistic infection or death. A secondary endpoint was any major cardiovascular, renal, or hepatic event. The trial indicated that episodic ART was associated with an increased risk of AMI (HR 1.6, 95% CI, 1.0 to 2.5, p = .05). The investigators concluded that future research was needed to investigate the effect of treatment interruption and intermittent viremia on inflammation and biomarkers and their relationship to cardiac events.

Two large trials have demonstrated that individual antiretroviral agents are associated with an increased risk of MI—the SMART study and the D:A:D trial (Lundgren et al., 2008; Sabin et al., 2008). The D:A:D study found that recent use (within the preceding 6 months) of abacavir or didanosine was associated with a 90% increase (p = .0001) and 49% increase (p = .003), respectively, of rate of MI among the 33,347 patients enrolled in the study (Sabin et al., 2008). The increased rate was noted while patients were taking the drugs and shortly after discontinuing their use; however, the risk seemed to diminish within a few months after their discontinuation. No cumulative effect was noted. The SMART study findings were consistent with D:A:D with respect to abacavir use, finding that the risk of CVD was doubled in patients receiving abacavir when compared to other nucleoside backbones (Lundgren et al., 2008). Patients who were on abacavir at study entry were also noted to have elevated levels of biomarkers, including hsCRP and IL-6, and the investigators suggested that abacavir may have pro-inflammatory properties. A smaller study of 35 patients also found a significant increase in hs-CRP (p = .03) following a switch to an abacavir-containing regimen when

compared with patients who received a non-abacavir-containing regimen who served as controls (Kristoffersen et al., 2009).

Subsequent to the SMART trial findings, investigators began to focus on endothelial dysfunction (Baker et al., 2009; Dube et al., 2010; Francisci et al., 2009; Ross et al., 2008) and specific biomarkers, including C-reactive protein (CRP), interleukin-6 (IL-6), D-dimer, s-VCAM, CCL2, adiponectin, and IL-10 (Baker et al., 2010; Calmy et al., 2009; Kuller et al., 2008; Ross et al., 2009; Tien et al., 2010; Triant, Meigs, & Grinspoon, 2009) to explain the increased CVD risk in this population. Collectively, these studies suggest that persistent viremia, immune activation, and inflammation related to HIV viral infection and replication may account for some of the increased risk of cardiac events seen in the SMART trial.

In a nested case control study of the SMART trial participants, Kuller et al. (2008) reported that IL-6 and D-dimer levels were higher at study entry and were significantly related to all-cause mortality (OR 8.3, 95% CI, 3.3–20.8, $p < .0001$ and OR 12.4, 95% CI, 4.2–37.0, $p < .0001$; Kuller et al., 2008). Comparing SMART study participants with non-HIV-infected controls, Neuhaus et al. (2010) found that hs-CRP and IL-6 levels were 55% higher in those with HIV infection ($p < .001$) and remained so even after initiation of ART and achievement of virologic suppression (Neuhaus et al., 2010). Triant et al. (2009) found that CRP and HIV status were independently associated with AMI even after controlling for age, sex, race, hypertension, diabetes, and dyslipidemia (OR 2.13, 95% CI, 1.92–2.37, $p < .0001$; and OR 1.93, 95% CI, 1.21–2.93, $p = .004$; Triant et al., 2009). These investigators concluded that compared with non-HIV-infected patients with normal CRP, those with HIV infection and elevated CRP had a fourfold increase in the

risk of AMI. Calmy et al. (2009) concluded that HIV replication was associated with increased levels of VCAM and CCL2, and decreased levels of adiponectin and IL-10. The associations persisted even after adjusting for known cardiovascular risk factors, such as age, total cholesterol, and LDL cholesterol levels (Calmy et al., 2009). This cohort consisted of 62% women; however, overall predicted CVD risk was low with 49% of the cohort having a Framingham Risk Score of less than 10%.

Preclinical atherosclerosis, measured by carotid intima-medial thickness (cIMT), has been the focus of several investigations (Calza et al., 2009; De Socio et al., 2010; Grunfeld et al., 2009; Hsue et al., 2004, 2006; Maggi et al., 2009; Ross et al., 2009; Van Vonderen et al., 2009). A cIMT of greater than 0.9 mm is considered a pathologic finding, and the American Heart Association has endorsed the use of cIMT in cardiovascular risk assessment (Greenland et al., 2010). Comparing HIV-infected to noninfected persons, investigators found that HIV infection was associated with greater cIMT ($p < .0001$), even after adjusting for traditional CVD risk factors and demographic variables, and was similar to changes usually seen in smokers (Grunfeld et al., 2009). A longitudinal study of 37 ART-naïve patients examined cIMT for 24 months after the initiation of ART and found that cIMT increased progressively over time and did not differ by regimen prescribed (Van Vonderen et al., 2009). Changes in cIMT in this study were statistically significant ($p < .001$ and $p = .01$). Calza et al. (2009) found that the mean values of cIMT were more elevated in participants who were ART-experienced compared to those who were ART-naïve. In addition, the prevalence of carotid plaques was higher in those who were ART-experienced, had a longer duration of HIV infection, had hyperlipidemia, or had lipodystrophy (Calza et al., 2009). Maggi et al. (2009) found

that the presence of subclinical carotid lesions, measured by cIMT, was highly prevalent (20.9%) among antiretroviral-naïve subjects. A previous study had reported an increased cIMT prevalence of 6.7% in an age-matched HIV-negative control group (Maggi et al., 2000). This was significantly associated (OR 5.9, 2.1–16.3, $p < .006$) with participants with elevated Framingham Risk Scores (Maggi et al., 2009). De Socio et al. (2010) also found that subclinical atherosclerosis in ART-naïve patients was significantly associated with elevated Framingham Risk Scores ($p < .001$). A cross-sectional study examining young adults, between the ages of 17 and 23, found that HIV infection itself and duration of antiretroviral therapy were both associated with higher cIMT ($p < .001$; Vigano et al., 2010).

A previous study showed conflicting results—cIMT annual progression rates did not differ among HIV-infected participants taking protease inhibitor therapy, those not on protease inhibitor therapy, and uninfected controls. This study however included both participants on ART and on a treatment interruption; also, use of lipid-lowering therapies was not controlled for—these two factors may account for some of the difference (Currier et al., 2007).

Endothelial function, measured by brachial artery flow-mediated dilation (FMD), has been examined in several studies in an attempt to determine the etiology of increased cardiovascular risk in HIV infection. These studies included a retrospective analysis (Francisci et al., 2009), one prospective analysis (Ross et al., 2008), and one cross-sectional study (Baker et al., 2009). Across these studies, the data suggest that HIV infection itself, and not HIV treatment, induces endothelial activation and endothelial dysfunction. These changes may explain some of the association between chronic

inflammation seen with HIV viral replication and the increased incidence of MI in HIV disease.

Cardiovascular disease and perceived risk. In the non-HIV-related literature, studies demonstrated that women were often unaware of CVD risk factors (Hart, 2005; Lange et al., 2009; Oliver-McNeil & Artinian, 2002; Pregler et al., 2009; Thanavaro et al., 2010; Wu, 2007) and had low levels of risk factor knowledge (Lange et al., 2009; Oliver-McNeil & Artinian, 2002; Thanavaro et al., 2010). Thanavaro and colleagues (2006) found in a sample of 120 women that low education level was associated with poor CVD risk factor knowledge (Thanavaro, Moore, Anthony, Narsavage, & Delicath, 2006). Poor risk factor knowledge contributed to underestimation of perceived CVD risk in some studies (Choi et al., 2008; Christian et al., 2005; Hart, 2005). In a sample of 143 adults, Choi et al. (2008) found that higher levels of CVD knowledge were associated with higher perceptions of CVD risk (Choi et al., 2008). Another study suggested that knowledge of cardiovascular risk factors significantly influenced perception of risk (Christian et al., 2005). A population-based survey of 816 adults found that socioeconomic status was associated with CVD risk perception (Alwan, William, Viswanathan, Paccaud, & Bovet, 2009). No studies have been conducted in the HIV-infected population to evaluate CVD risk perception or risk factor knowledge.

Summary

The extensive review of the empirical literature examining the association of HIV infection and cardiovascular disease clearly indicates that there is an increased risk of cardiovascular disease and an increased rate of acute myocardial infarction associated with HIV infection and/or its treatment. The prevalence of acute events in HIV-infected

individuals appears to be elevated when compared to those of similar ages in the general population. Previous research has focused on physiologic measures of cardiovascular disease in this population. Investigators remain uncertain about whether the elevated risk and rate of disease is due to host factors, viral factors, treatment factors, lifestyle behaviors, or a combination of these.

To date, there is no published report of the level of knowledge that HIV-infected individuals have about cardiovascular risk factors. Additionally, it is not known whether HIV-infected persons perceive themselves to be at risk for cardiovascular disease. This study provides information about HIV-infected patients' perception of risk and their level of risk factor knowledge. It examines whether relationships exist between individuals' level of risk factor knowledge, perceived risk, and estimated risk of cardiovascular disease.

CHAPTER 2

Theoretical Framework

The purpose of this prospective study was to describe cardiovascular risk factor knowledge and CVD risk perception in a cohort of HIV-infected adults. Specifically, this study examined the influence of cardiovascular risk factor knowledge on perceived risk of cardiovascular disease in a sample of HIV-infected adults. Additionally, it described the estimated risk of cardiovascular disease (using the Framingham Risk Assessment Tool), the perceived risk of cardiovascular disease, the level of cardiovascular risk factor knowledge, and the relationship between estimated and perceived risk of CVD in HIV-infected adults. The Health Belief Model (HBM) was used as a guide to assess and describe the concepts of interest in the study.

Theory Overview

The HBM is a psychological model originally developed in 1966 by Irwin Rosenstock to identify determinants of health-related behavior. The purpose of the model is to explain, predict, and ultimately influence or modify behaviors (Rosenstock, 1974). Revisions to the model by Rosenstock and colleagues incorporated evidence from research about the role that health motivation, health knowledge, and self-efficacy played in behavior change (Rosenstock, Strecher, & Becker, 1988). This predictive model was initially designed to identify variables that underlie health-related decisions (related to immunization uptake) and assess the likelihood that an individual would adopt certain behaviors. Later, researchers used it to examine behavior related to health promotion, such as performance of breast self-exam (Janz & Becker, 1984), smoking cessation (Conrad, Campbell, Edington, Faust, & Vilnius, 1996), and decisions related to chronic

illness management—for example, management of asthma (Becker et al., 1978) and hypertension (Jones, Jones, & Katz, 1987; Wen-Wen, Wallhagen, & Froelicher, 2010). The theoretical model has been used as a guiding framework for numerous prospective and retrospective studies over the past 30 years.

Assumptions and Antecedents of the Theory

The Health Belief Model is predicated on two assumptions about human behavior: that an individual's behavior is influenced by (a) the value he/she places on achieving a particular goal, and (b) his/her estimate of the likelihood that certain behaviors or actions will lead to the achievement of a particular goal (Janz & Becker, 1984).

Antecedents in the HBM are those factors that precede and may determine the likelihood of a health action occurring. Individuals must possess sufficient concern about the heath issue, such that it is viewed as relevant to them. Individuals must believe that they are susceptible to the health issue or condition, so that there is the existence of a perceived threat. Individuals must also believe that certain health behaviors will be beneficial in reducing or eliminating the threat of the illness or disease (Rosenstock et al., 1988).

Concepts of the Model

The Health Belief Model incorporates individual perceptions (of susceptibility and severity), with modifying factors (such as sociodemographic variables, structural variables, and cues to action) to predict the likelihood of taking certain behaviors. There are four key concepts described in the original model that are considered independent predictors of health-related behavior. They include individual perceptions of

susceptibility (risk) and *severity (*which together comprise perceived threat), perceived *benefits,* and perceived *barriers*. *Cues to action* and *sociodemographic variables* are modifying factors that may alter perceptions and influence the likelihood of taking action. *Structural variables*, such as knowledge, are additional modifying factors. *Self-efficacy*, a separate independent variable, and *health motivation* are additional concepts that were added in the 1988 revision of the original model. Rosenstock et al. (1988) explained that the original model neglected to include these later concepts because of its focus on "circumscribed preventive actions" such as immunization uptake that did not require self-efficacy, per se (Rosenstock et al., 1988). With a change in focus to include acute and chronic illness behaviors, they felt the inclusion of self-efficacy was appropriate. This addition broadened the scope of the *potential barriers* construct and led to the model's ability to account for more variance in clinical studies.

Concept Definitions

Perceived susceptibility is defined as the individual's assessment of his/her risk of getting an illness or condition. *Perceived severity* is the individual's assessment of the seriousness of the illness or condition and the medical, clinical, and social consequences related to this condition. These consequences may include death, disability, pain, effect on family or work life, or social relationships. *Perceived susceptibility* and *perceived severity* are additive and comprise perceived threat. *Perceived barriers* is defined as the individual's assessment of the obstacles that may interfere with the adoption of the new behavior, such as cost, side effects, accessibility, and personality characteristics. *Self-efficacy*, or the lack thereof, is considered a potential barrier in the revised model. Rosenstock et al. use Bandura's original definition of *self-efficacy*—the belief that one

can successfully execute the behavior required to produce a desired outcome (Rosenstock et al., 1988). *Health motivation* is defined as the incentive to behave in a particular way, with incentive (or reinforcement) defined as the value of a particular object or outcome. *Perceived benefits* is defined as the individual's assessment of the positive outcomes associated with the adoption of the new behaviors. It may also refer to the individual's assessment of the effectiveness that certain behaviors will have in reducing the threat of illness or disease. *Sociodemographic and structural variables,* such as age, gender, race, education level, income level, and knowledge, are modifying factors that may affect individual perceptions and indirectly influence health-related behaviors. *Cues to action* are events that may be related to the body (such as physical symptoms) or the environment (such as media advertising, postcard reminders, information provided, or personal experiences) that propel an individual toward behavior change.

This study examined individual perceptions (including perceived susceptibility/risk), and modifying factors (including sociodemographic and structural variables and cues to action). Perceived barriers, represented by insurance status, was also measured. Perceived severity, perceived benefits, and the likelihood of taking action were not explored in this study. See **F**igure 1.

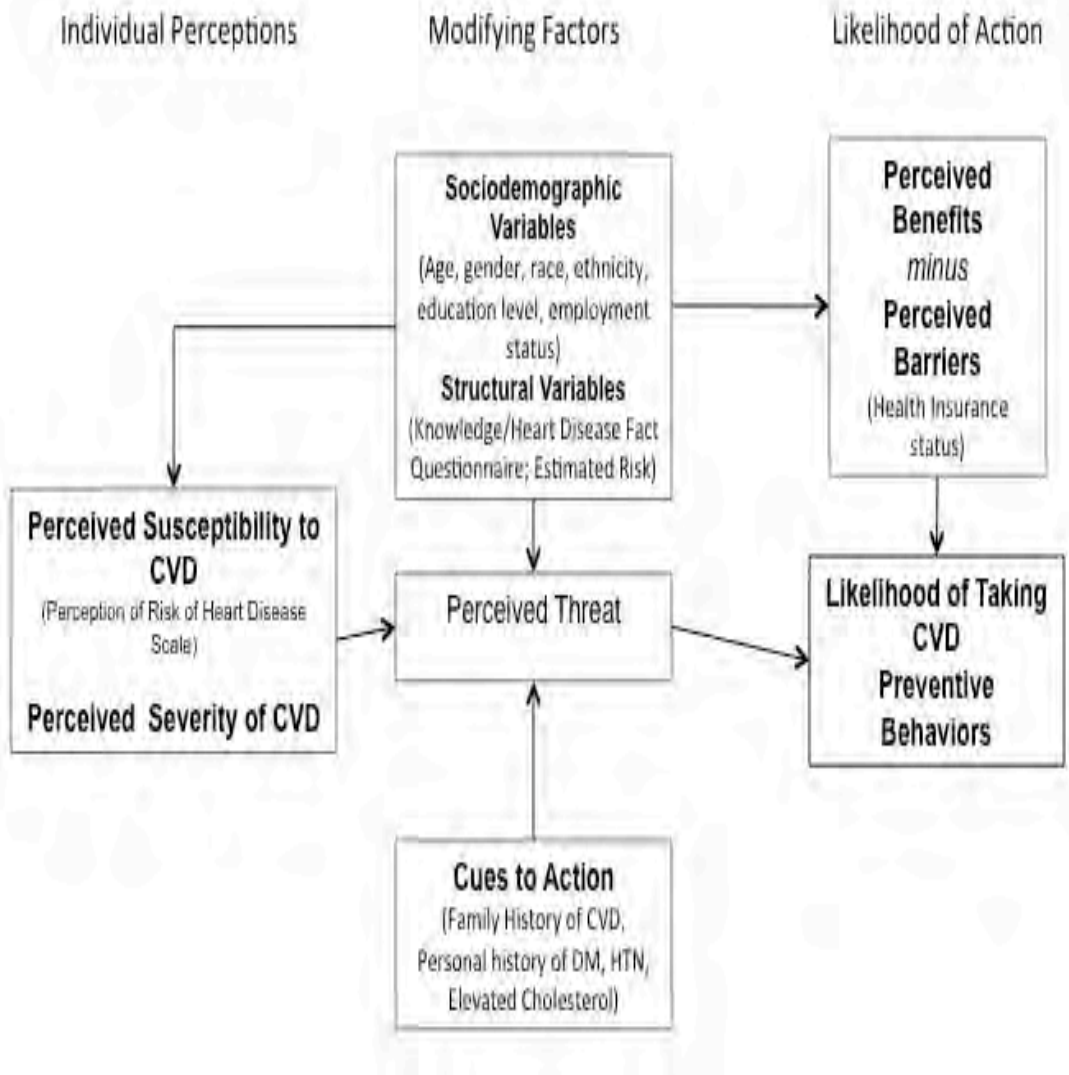

Figure 1. Health Belief Model and CVD Risk (Adapted from Stretcher, V., & Rosenstock, I. M., 1997)

Utility of the Health Belief Model in Research

The Health Belief Model is a useful model for nursing research. Elder and colleagues (1999) have suggested that the model can be used as a guide or tool to develop effective and efficient interventions to facilitate behavior and lifestyle changes in patients. Information gained from the assessment of an individual's level of perceived susceptibility and severity of the outcome can be used to frame specific health messages. Perceived barriers to behavior change can be assessed so that effective interventions to

reduce or eliminate some of these obstacles to change can be developed. Perceived benefits of behavior change can be assessed, and these can be incorporated into the interventions to reinforce the positive outcomes of behavior change and potentially increase motivation to change. Cues to action can be developed as reminders of the positive benefits of behavior change or negative consequences related to the lack thereof (Elder, Ayala, & Harris, 1999).

Review of Research Utilizing the HBM

Numerous prospective and retrospective studies have examined the utility of the HBM to explain health-promoting behavior (Abood, Black, & Feral, 2003; Chiou et al., 2009; Conrad et al., 1996; Janz & Becker, 1984; Jones et al., 1987; Jones, Weaver, & Friedmann, 2007; Wen-Wen et al., 2010; Wilson, Sisk, and Baldwin, 1997). In their integrative review of 46 studies that used the HBM as the guiding framework, Janz & Becker (1984) found that *perceived barriers* was most strongly associated with the adoption of new behaviors. *Perceived susceptibility* was also a strong predictor of behavior. *Perceived severity* was the least significant variable in the model when examining preventive behaviors but was slightly more predictive in sick role behaviors (in chronic illness management). Across studies examining cardiovascular health-promoting behaviors, perceived barriers and perceived susceptibility were most significantly correlated with adoption of new behaviors (Janz, 1988; Janz & Becker, 1984).

More recently, the Health Belief Model has been used as the guiding theoretical framework in three studies related to cardiovascular risk factor behaviors in the general population (Ali, 2002; Chiou et al., 2009; Jones et al., 2007). Self-efficacy was found to

be the strongest predictor of CVD-risk-modifying behaviors, such as taking medications regularly and eating a heart-healthy diet, in a cross-sectional study of 156 adults—explaining 24% of the variance in the study (Chiou et al., 2009). In a second study (which did not include self-efficacy as a variable), susceptibility to CVD explained 50% of the variance, while knowledge of CVD risk factors explained 19.5% (Ali, 2002). One longitudinal intervention study examined the effectiveness of a 5-week heart disease prevention program (Jones et al., 2007). This small study (N = 48) measured knowledge about heart disease and perceived susceptibility to heart disease before and following an educational program. Using the participants as their own controls, they found that perceived susceptibility increased significantly (r = -.293; p = .04) following the educational intervention.

Individual cardiovascular risk factors, such as smoking, elevated cholesterol, and hypertension, have been the focus of several studies using the HBM as the theoretical framework (Abood et al., 2003; Conrad et al., 1996; Jones et al., 1987; Wen-Wen et al., 2010; Wilson et al., 1997). Perceived barriers ($F[1,118] = 22.9$, $p < .001$) and self-efficacy ($F[1,118] = 28.1$, $p < .001$) were predictors of behavior in a cross-sectional study (N = 151) comparing participants and nonparticipants in a blood pressure and cholesterol screening program (Wilson et al., 1997). Two longitudinal studies, examining hypertension management behaviors and smoking reduction behaviors, found that perceived barriers was significantly associated with the behavior change ($p < .05$). In the study of smoking behaviors, perceived barriers and cues to action explained 74% of the variance in smoking reduction. Neither of these studies measured self-efficacy.

A frequent criticism of the model is that the variables often correlate weakly with behavior and that, in most studies, the effect size was small (all *r*'s < .21; Armitage & Conner, 2000). However, in studies that utilize the HBM, variables are often inconsistently measured. Use of nonvalidated instruments, variability in the operational definitions of the concepts, and inconsistent measurement of the variables have contributed to the inconsistency of the findings (Fleury, 1992; Janz & Becker, 1984). Rosenstock attributed the lack of predictability, small effect size, and low levels of variance to the exclusion of the self-efficacy variable in earlier studies and explained that this finding was the impetus to include self-efficacy in later versions of the model. Armitage & Conner (2000) explained that in a review of social cognitive models of health behavior studies, self-efficacy is often the dominant predictor of individuals' behavior. Perhaps its inclusion in the model will yield more significant findings.

In the HIV literature, studies using the Health Belief Model often examine medication adherence or condom use practices (Coleman & Ball, 2009; Cox, 2009; Volk & Koopman, 2001). In a cross-sectional study of 223 men and women in Kenya, Volk and Koopman (2001) found that only perceived barriers was significantly associated with the frequency of condom use in men and women ($t = -1.98$, $p < .05$; $t = -4.01$, $p < .001$, respectively). A measure of self-efficacy was not included in this model. Knowledge about HIV/AIDS, identified as a separate independent variable in this study, was significantly associated with condom use in men only ($t = 1.99$, $p < .05$).

Cox (2009) examined predictors of medication adherence in a 5-year prospective observational substudy of participants in a large phase III research study comparing daily versus thrice-weekly use of trimethoprim-sulfa for the prevention of pneumocystis carinii

pneumonia. Self-reported medication adherence was measured by participant and clinician assessments at 6 and 12 months. Older age (OR 1.5, 1.1–2.0, p = .005) and perceived seriousness of HIV (OR 2.8, 0.3–1.0, p = .04) were significantly associated with visit adherence in the study. Of the variables studied, only older age was significantly associated with medication adherence (OR 1.7, (1.2–2.5, p = .003). The use of creative definitions of the HBM concepts used in this study, the measurement of only select variables, and the use of self-reported adherence make the interpretation of this study's finding problematic.

The Health Belief Model has been used as a theoretical framework for numerous studies over the past 30 years. It was used to guide this study of perceived risk, estimated risk, and cardiovascular risk factor knowledge in the HIV-infected population.

Purpose and Specific Aims

The purpose of this study was to describe cardiovascular risk factor knowledge and CVD risk perception in a cohort of HIV-infected adults. The primary aims of this study were as follows:

1. To describe (a) the estimated risk of cardiovascular disease, using the Framingham Risk Assessment Tool, (b) the perceived risk of cardiovascular disease, and (c) the level of cardiovascular risk factor knowledge in HIV-infected adults;
2. To describe the relationship between estimated and perceived risk of CVD in HIV-infected adults;
3. To examine the influence of cardiovascular risk factor knowledge on perceived risk of cardiovascular disease in a sample of HIV-infected adults.

Operational Definitions

In this study, concepts were operationalized in the following manner:

1. *Perceived Susceptibility to CVD* was defined as the individual's perception that he/she was at risk for heart disease as measured by the Perception of Risk of Heart Disease Scale;
2. *Perceived Barriers* was defined as the obstacles that may influence or interfere with the individual's likelihood to take CVD preventive action, as measured by the individual's health insurance status;
3. *Cues to Action* was defined as modifying factors related to individual symptoms or their environment that affected their likelihood of taking CVD preventive behaviors, as measured by a family history of CVD, or a personal history of diabetes, hypertension, or elevated cholesterol;
4. *Sociodemographic and Structural Variables* was defined as modifying factors that may have affected individual perceptions and indirectly influence the adoption of CVD preventive behaviors. Several variables were measured, including age, gender, race/ethnicity, education level, employment status, and heart disease knowledge (measured by the Heart Disease Fact Questionnaire). Additionally, the 10-year estimated CVD risk of an individual was measured as a structural variable and was calculated using the Framingham Risk Score.

Perceived Severity, Perceived Benefit,s and the *Likelihood of Taking Preventive Action* were not measured in this study.

CHAPTER 3

Methods

This descriptive study employed a prospective, cross-sectional design to describe the level of cardiovascular risk factor knowledge, estimated risk of cardiovascular disease and CVD risk perception in a cohort of HIV-infected adults. Each participant had one study visit in which all demographic and clinical data were collected and all research instruments were completed. The principal investigator (PI) collected all data during the study visit by direct interview, and each visit lasted 30 to 40 minutes.

Sample

The convenience sample for this study consisted of 130 adult participants recruited from two hospital-based HIV clinics, located at the Miriam Hospital and the Rhode Island (RI) Hospital. A power analysis was conducted for the analyses planned to address specific aims #2 and #3 (Tabachnick & Fidell, 1996). Aim #1 is descriptive only. A linear regression analysis was conducted to address specific Aim #3 (one predictor—CVD risk factor knowledge—and one dependent variable—perceived risk of CVD) with three covariates, which required a minimum sample size of 84 subjects assuming an effect size of .15, alpha = .05 and 80% power. This is consistent with the power analysis for specific Aim #2 (2-tailed correlation between actual and perceived risk of CVD), which suggested that a sample size of 82 subjects would provide 80.3% power to yield a correlation of .30 or greater between these variables.

A sample of 130 HIV-infected adults were recruited for this study—it was assumed that there would be less than 5% missing data due to the interview format used for data collection from the two hospital-based HIV clinics. These clinics care for over

1,200 HIV-infected adults with an average of 6,000 patient visits annually. The accessible population included 30% women, 70% men, 40% Hispanic, 30% Caucasian and 30% Black. All patients were 18 years of age or older. The female population comprised approximately 30% of the clinic population, and recruitment of 37.7% women in the study sample was achieved.

Inclusion and exclusion criteria. Participants were eligible for participation in this study if they met the following inclusion criteria: (a) males and females age 18 or older, (b) HIV-infected per the medical record, (c) able to read and speak English, and (d) had the ability to give written informed consent. Participants were excluded if they (a) were unable to read and understand English, (b) had an established diagnosis of CVD (AMI or CVA) in the medical record, or (c) had a past cardiovascular event (myocardial infarction or stroke) or intervention (coronary artery bypass surgery, cardiac stent placement, or vascular surgery). The principal investigator (PI) documented the number of participants excluded, based on the screening history and the inclusion/exclusion criteria. The PI tracked the number of participants who declined participation and recorded the reasons for decline.

Setting

The setting for this study was Rhode Island Hospital and the Miriam Hospital. These hospitals are located in Providence, RI, approximately four miles apart and are unified under Lifespan Corporation, a private nonprofit health care organization. They are the major teaching hospitals for Brown University Warren Alpert School of Medicine. The two HIV clinics provide care to approximately 1,200 HIV-infected adults with diverse cultural and economic backgrounds. The clinics are staffed by eight

infectious disease physicians, one nurse practitioner, four nurses, and two clinical social workers.

Procedures

Institutional Review Board (IRB) approval was obtained from the University of Massachusetts Medical School IRB and the RI Hospital IRB. Participants were recruited from the RI Hospital and Miriam Hospital HIV clinics simultaneously beginning in March 2011. The PI recruited, enrolled and interviewed participants at the Miriam clinic on Mondays and Wednesdays. Participants were recruited and interviewed at the RI Hospital site on Wednesdays and Thursdays. Approximately five to ten subjects were recruited and interviewed weekly. Enrollment and data collection were completed by August 31, 2011. As patients presented for their regularly scheduled appointments, the clinic staff (including secretary, nursing assistants, and nurses) informed them of the ongoing study. If a patient expressed interest in participating, he/she was then referred to the PI for screening. The PI explained to potential participants that the study was examining the risk of heart disease in persons infected with HIV and measuring the level of knowledge of risk factors for heart disease. The PI discussed the study with prospective participants in person in a private exam room within the clinic. At the initial appointment, subjects who agreed to participate were read the IRB-approved informed consent form. The PI allowed time to answer all questions, and the participant's level of involvement was reviewed with each potential participant. Each participant was asked to sign the consent form, and a signed copy was given to each participant. Data collection began once the consent process was completed. The face-to-face study visits took place in a private room in the clinic and lasted 30 to 40 minutes. Patients who preferred to

participate on a day other than their appointment day were scheduled to return on another day to complete the screening and enrollment process, sign the informed consent, and complete the study visit. Less than 5% of participants preferred to do this.

The literature suggested that persons with HIV infection may have low literacy (Drainoni et al., 2008; Kalichman et al., 2000), and previous experience with this population demonstrated that approximately 10–20% of participants may have had difficulty completing self-report instruments. To address this potential barrier to participation, we decided to obtain study data by direct interview. To evaluate the interview procedure, the study instruments were piloted with nine HIV-infected adults prior to general study recruitment to examine the time it would take to complete the consent process and complete the study instruments.

Measures

Perception of Risk of Heart Disease Scale (PRHDS). This 20-item instrument was developed to measure an individual's perception of the probability of developing heart disease (Ammouri & Neuberger, 2008). It is a self-report scale that takes approximately 20 minutes to complete. Each item on the scale has a 4-point Likert scale response option ranging from 1 (strongly agree) to 4 (strongly disagree). Item scores may be summed for subscales and a total scale score. Higher scores on the overall scale indicate increased perception of risk. Subscales include dread risk, risk, and unknown risk. Initial testing with a primary care sample of 295 persons greater than 15 years of age without heart disease demonstrated internal consistency of .68–.80. Total scale alpha was .80. Test-retest reliability was .61–.76. Construct validity was demonstrated by achieving

a significant correlation between the PRHDS and the Health Promotion Lifestyle Profile II (r = .20–.39, $p < .01$).

CHD risk factor knowledge. The Heart Disease Fact Questionnaire (HDFQ) is a 25-item instrument that measures knowledge of major risk factors for the development of CVD (Wagner, Lacey, Abbott, de Groot, & Chyun, 2006; Wagner, Lacey, Chyun, & Abbott, 2005). Each item on this scale has responses of true, false, or I don't know. Total scale scores are calculated by summing the total number of correct answers and can range from 0–25. Higher scores indicate a higher level of knowledge. Good internal consistency (Kuder-Richardson r = .77) was demonstrated in a group of 524 ethnically diverse adults with diabetes. Test-retest reliability was .89.

Cardiovascular risk factors. Fasting lipid parameters and blood glucose levels obtained within the previous 12 months were extracted from the medical record. The standard of care in the clinics for obtaining this labwork is at least once annually. In the case where this data was missing, a reminder was given to the provider so that it could be obtained following the clinic visit. Height and weight were measured using the same scale within each clinic, and the body mass index was calculated using the National Heart Lung and Blood Institute formula (National Heart Lung & Blood Institute, n.d.). The PI assessed family history of premature CVD (at age less than 65 years in a mother, or less than 55 years in a father) and personal history of diabetes mellitus via self-report during the study visit.

HIV clinical variables. The most recent CD4 (T cell count), nadir CD4, and HIV viral load (HIV-RNA by PCR) were collected from the medical record. All current antiretroviral medications being taken were obtained from the medical record.

Framingham risk score. The Framingham Risk Score was calculated using the National Heart, Lung and Blood Institute worksheet, which includes the measures of total cholesterol, smoking status, high-density lipoprotein, and systolic blood pressure ("Third report of the National Cholesterol Education Program (NCEP) expert panel on detection, evlauation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III) final report," 2002). Blood pressure was recorded as the average of three seated measurements taken five minutes apart by the PI on a manual mercury sphygmomanometer during the study visit (Pickering, 2005). The literature suggests that averaging at least two measurements of blood pressure resulted in marked increases in the predictability of cardiovascular risk assessments (Bell, Hayen, McGeechan, Neal, & Irwig, 2011).

The Framingham Risk Score, defined as the percent risk of having a coronary event in the next 10-year period, was used to determine each participant's level of estimated risk. This score stratifies individuals into three risk categories: low (less than 10% risk of having an event in the next 10 years), intermediate (10–20% risk), and high (more than a 20% risk). Because diabetes mellitus is considered a cardiovascular risk equivalent ("Third report of the NCEP expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III) final report," 2002), any participant with a diagnosis of diabetes was categorized as having high risk (more than 20%).

Sociodemographic variables. Demographic variables, including age (in years), gender, educational level (in years), self-reported race and ethnicity, insurance status, employment status, and time since HIV diagnosis (in years) were obtained by the PI at

the initiation of the study interview session. In order to categorize those on ART, those on treatment interruption, those never started on ART, and those taking antihypertensive, diabetic, or cholesterol-lowering medications, a complete medication list was obtained from the medical record.

Data Management

Each participant was assigned a unique research ID number. A master sheet with participant names and matched study ID numbers was kept in a locked cabinet in the PI's office. All study data sheets were kept in the locked cabinet, and only the PI had access to the data. Only de-identified data were kept on a password protected research designated drive at the University of Massachusetts Medical School, which was backed up nightly. The PI entered the de-identified data into the database. A research assistant was trained and entered all de-identified data into a second identical database. Double entry was performed to ensure the accuracy of the data to be analyzed. The two data sets were merged, and correlations were run on each variable to identify errors in data entry. Any variable that did not have a correlation of one (1) was identified. The data sets were then reviewed line by line, and errors in entry were corrected. Correlations were rerun between the two data sets to ensure that all errors had been corrected. A clean data set file was established, and this data set was used for the remainder of the data analyses for the study.

Data Analysis

Descriptive statistics (frequencies, means, standard deviations, and percentages) were calculated on the final corrected data set for all study variables. For continuous variables, mean, median, skewness, standard error of the mean, standard deviation, and

histograms were calculated. Frequencies were run on all categorical variables. All continuous variables were checked for normal distribution by calculating Fisher's measure of skewness. A log transformation was performed on the Framingham Risk Score variable to achieve a normal distribution.

Eleven items in the Perception of Risk of Heart Disease (PRHD) scale were reverse coded as they were negatively worded items in the scale. Total scale scores were then calculated for each case. The reliability of this scale was calculated by running the Cronbach's alpha coefficient. All responses in the Heart Disease Fact Questionnaire were recoded to indicate whether the item had been answered correctly or incorrectly by the participant. The response for each item in the scale was dichotomized into a new variable indicating correct or incorrect response for that item. A total scale score was calculated by summing the number of correct responses. Internal consistency was calculated for the Heart Disease Fact Questionnaire using the Kuder-Richardson formula.

To address Aim #1, the actual (estimated) risk of cardiovascular disease was calculated using the Framingham Risk Assessment Tool and was entered as a continuous variable. The perceived risk of CVD (measured by the PRHD scale) was calculated and entered as a continuous variable. The level of cardiovascular risk factor knowledge, measured by the HDFQ, was also summed and entered as a continuous variable. Descriptive statistics were run on these variables to describe the sample.

To describe the relationship between estimated and perceived risk of CVD (Aim #2), Pearson correlation statistic was used. The effect of age on risk perception was controlled for in the data analysis by running a partial correlation between estimated and perceived risk while using age as a covariate in the statistical model.

To examine the influence of cardiovascular risk factor knowledge on perceived risk of cardiovascular disease (Aim #3), linear regression was used. Pearson *r* was used to examine the relationship between age, education level, and time since HIV diagnosis with risk perception and risk factor knowledge. T-test statistic was used to examine differences by gender and ART status. Chi square statistic was used to examine differences by race and ethnicity. These tests were performed to determine which variables would be included as covariates in the regression equation.

Statistical significance was accepted at the 95% confidence interval ($p < .05$ level). All statistical analyses were performed using the Statistical Package for the Social Sciences Version 17.0 (SPSS) program.

Protection of Human Subjects

This cross-sectional study recruited 130 participants from the Miriam and Rhode Island Hospital HIV clinics. All visits took place in a private exam room within the HIV clinics at each hospital. The study protocol was reviewed and approved by the Rhode Island Hospital Human Subjects Committee (IRB) and the University of Massachusetts Medical School IRB. All efforts were made to fully explain the study procedures with written informed consent obtained prior to enrollment. Every effort was made to protect each participant's confidentiality and minimize risk.

Human subjects involvement and characteristics. Participants were both male and female adults with HIV infection recruited from the two hospital-based HIV clinics. Participants were recruited as they presented to their clinic appointments. All subjects were at least 18 years of age and reflected the diverse sociodemographic composition of the clinic population. The patient demographics of the clinics was 70% male, 30%

female, 40% Hispanic, 30% Caucasian, and 30% Black. Ethnicity and gender were not used as criteria in determining eligibility.

Eligible subjects met the following criteria:

1. Males and females over age 18

2. HIV-infected per the medical record

3. Able to read and speak English

4. Ability to give written informed consent

Participants were excluded for these criteria:

1. Inability to read and understand English

2. Established diagnosis of CVD (acute myocardial infarction or stroke)

3. Past cardiovascular intervention (coronary artery bypass graft surgery, stent placement, or vascular surgery)

Sources of Research Materials

The following data were collected from human subjects for research purposes:

1. A 20-item instrument, Perception of Risk of Heart Disease Scale, was completed during the study visit to measure the participant's perception of his/her probability of developing heart disease.

2. A 25-item instrument, the Heart Disease Fact Questionnaire, was completed during the study visit to measure the participant's cardiovascular risk factor knowledge.

3. Blood pressure measurements were taken three times by the PI during the study visit at 5-minute intervals.

4. Height and weight were measured for each participant once during the study visit.

5. Demographic data, including age, gender, race/ethnicity, number of years diagnosed with HIV, number of years of education, smoking status, presence of diabetes, and family history of cardiovascular disease (myocardial infarction history in mother or father of participant) were collected by self-report. Current medications and most recent lab values (including CD4 count, nadir CD4, glucose and lipid values) were obtained from the medical record. Data extraction for demographic and laboratory information complied with Health Insurance Portability and Accountability Act of 1996 (HIPPA) regulations.

Potential Risks

There were no anticipated physical risks to participants. No participants became upset when talking about cardiovascular risk factors, however, some participants asked about their own level of risk, and these questions were referred back to the participants' medical provider. To guard against the potential for a loss of confidentiality during study instruments completion, a unique research ID number was the only identifier entered on the data collection instruments. The data were stored in a secure locked cabinet, which was accessible only to the PI.

Adequacy of Protection Against Risks

Participants were recruited from the Miriam Hospital and Rhode Island Hospital. Prior to recruitment, an informational meeting at each outpatient clinic was held to explain the study in detail to the clinicians and receptionists. The clinician or staff

directed all interested patients to the PI, who was present in the clinic on specified days for recruitment and enrollment. If a patient was interested in the study, the PI was available to proceed through the enrollment process while the patient was in the clinic. Any patient who expressed interest in the study on an alternate day was given a time to return on a day when the PI was present in the clinic.

The PI discussed the study with potential participants in person in a private office that was separate from other clinic patients and identified herself as the principal investigator of the study. The nature of the study was explained as well as the voluntary nature of participation. Each potential participant was reassured that participation, or lack thereof, would not influence their medical care. Subjects agreeing to participate were asked by the PI to sign an IRB-approved written informed consent. The consent was obtained from each participant only after she/he acknowledged understanding of the study and expressed willingness to participate. This was done prior to any study participation. The informed consent form addressed the purpose of the study, the study procedure, risks, confidentiality of materials, right to withdraw from the study at any time, and names of study contacts (i.e., the PI, and Human Subjects Committee) in case of any concerns or questions. Given the low literacy rates in this population, the PI read and explained the form to every potential subject, unless the subject insisted on reading it him- or herself.

Protection Against Risk

All study participants were monitored for risks related to study participation. Before participating in research assessments, participants were assured of confidentiality, and they were told that they had the right to terminate the interview at any time or could

refuse to answer specific questions. No participant became distressed, upset, or uncomfortable during assessments, and no interviews needed to be terminated.

Every precaution was taken to insure that all data were kept strictly confidential. Only the PI had access to identifiable data (a list that links the study ID with the participants' names), which was stored in a locked file cabinet in the PI's office. Personal identifiers were stripped from the data and kept separately in a locked file available only to the PI. All information provided by a participant was referenced to a unique participant ID number and was kept in a locked file cabinet. The participant's ID number could be connected to the participant's name only through a single master file, accessible only to the PI. Passwords and protection codes were used to protect data files against inadvertent change or unauthorized access. All data files were backed up nightly.

Documentation of data management procedures was carefully maintained. No results were reported in a personally identifiable manner; only grouped data were presented in publications or presentations, and the usual standards for medical confidentiality were observed.

Potential Benefits of the Proposed Research to the Subjects and Others

There were no direct benefits to individual subjects in this study. However, all participants were compensated with a $20 retail gift card as compensation for their time and participation.

Importance of Knowledge Gained

Knowledge gained from this study has the potential to greatly improve treatment services for the population of HIV-infected adults. Cardiovascular disease is a major cause of morbidity and mortality for this population and this study highlighted knowledge

deficits that exist regarding cardiovascular risk and risk factor knowledge. Innovative education programs could be developed to address this need. Finding ways to effectively reduce cardiovascular risk in the HIV-infected population is of critical importance to the promotion of health in this group, especially given the increased life span of HIV-infected individuals in the U.S. Finding ways to prevent disease and promote health is consistent with the goals of the Institute of Medicine and National Institute of Nursing Research.

Inclusion of Women

Women were actively recruited into this study. The female population comprised approximately 30% of the clinic population, and recruitment of 37.7% women was achieved. No one was excluded from participation in this project on the basis of gender. Differences were analyzed between males' and females' CVD knowledge and perception of CVD risk to determine any trends in the data.

Inclusion of Minorities

Recruitment of an ethnically diverse group of participants was a high priority, and the PI actively recruited minorities into this study. The minority composition of the study population was anticipated to be approximately 50% of the total study sample, which is reflective of the general clinic population. The state of RI's population is 12.3% African American and 12.5% Hispanic; however, the proportion of African Americans and Hispanics in the clinic population is higher. Thus it was expected that minorities would be well represented in the study sample. This study may have limited power to differentiate CVD knowledge level and perception based on ethnicity or race. However, an exploratory analysis of these variables was planned.

Inclusion of Children

This study included individuals age 18 and older. Children under age 18 were not recruited because cardiovascular disease related to atherosclerosis and metabolic changes is rare in the pediatric population.

Summary

Describing HIV-infected patients' level of cardiovascular risk factor knowledge, perceived risk, and actual (estimated) risk is an important initial step in planning interventions to reduce risk. Ultimately, examining these concepts and describing the relationships among them will lead to insight regarding the needs of this population.

CHAPTER 4

Results

This chapter contains the findings from the pilot study and all descriptive data and analyses related to the main study. Following the discussion of the pilot, the reminder of the chapter will be organized and presented by specific aims. Descriptive statistics will be presented to fully describe the sample studied. Correlations will be presented to examine relationships between the variables. Finally, the results of the linear regression model are presented to describe the predictive relationship between risk factor knowledge and perceived risk.

Pilot Study

In March 2011, the first nine participants were recruited and the study interview and instruments were piloted to evaluate the (a) study procedures, (b) informed consent process, (c) instruments to be used, and (d) time needed to complete the process with each participant.

The study interview lasted 30–40 minutes, which included the time to complete the informed consent process, collect the demographic data, and complete the study instruments with each participant. Following the interview, an additional 20 minutes were required of the PI to extract the laboratory values and the medication lists from the medical record.

The consent form was read to all participants and no revisions were needed. Several participants did not want to take home a copy of the consent form and this was recorded. The demographic and clinical data collection form was well organized for data collection and was without problems. The Heart Disease Fact Questionnaire was easy to

understand for participants, and no issues were identified related to the completion of this instrument.

Participants identified that the Perception of Risk of Heart Disease Scale lacked an “I don’t know” response option. It was also noted that one item in this scale contained the word “unattainable,” which was not understood by seven of the nine (78%) pilot participants in the study.

Following discussion with the committee chair, the PI decided to pilot an alternate risk scale, and it was administered to four participants. The Index of Perceived Risk, developed by Becker & Levine (1987), was chosen. The index was comprised of four items that used a Likert scale from one (1) to five (5); 1 indicated *no concern at all*, or *very low probability estimates of having an event*, and 5 indicated *very high level of concern* and *extremely high estimate for having an event*. The items in this scale addressed a person’s (a) frequency of concern over having a coronary heart disease event, (b) estimate of having an event in the next five years, (c) likelihood of having such an event in his/her lifetime, and (c) estimated coronary heart disease risk compared to people of the same age and gender in the general population. The fourth item offered responses of *much less, less, about the same, more,* and *much more.* Items on this scale were summed, and the potential range of perceived risk index was four (4) to twenty (20) points. A higher score on this scale indicated a higher level of risk. Internal consistency of this scale had a published alpha of .80.

The Index of Perceived Risk was administered to three male and one female participant. Response was mixed to this new scale: One participant reported that the original scale was “more thorough” and felt the second scale “was just guessing

numbers." A second male participant said "both (scales) were easy," and the female participant said "it doesn't matter, either one was okay." The range of education of these four participants was from 8 to 12 years. After discussion with the dissertation committee members and chair, it was decided that the original Perception of Risk of Heart Disease scale would be used and the PI would leave missing any item that participants did not know how to respond to and would explain that "unattainable" was similar to the word "unreachable." This explanation was consistently used when any participant expressed difficulty understanding this word.

Aim #1—Descriptive Data

Sociodemographic variables. During the 6-month recruitment period, 170 HIV-infected adults were screened and 130 were enrolled. Demographic and clinical characteristics of the enrolled participants are presented in Table 1 and Table 2. Mean age of the sample was 48.0 **(**SD±8.4) years; 62% were male, and 69% identified as currently unemployed. Race and ethnicity was self-reported with 41.5% as White, 32% Black, 23% Hispanic, 1% Asian, and 2% Native American. Mean years of education completed were 11.8 (SD± 2.7) years.

Table 1

Sample Demographics of Continuous Variables (N = 130)

	Mean (± SD)	Median	Range
Age (years)	47.9 (8.4)	49.0	22–67
Years since HIV diagnosis	14.6 (7.9)	15.0	1–30
Body Mass Index (BMI)	26.7 (5.5)	26.1	14.9–47.8
Years of Education	11.8 (2.7)	12.0	4–19
Framingham Risk Score	7.87 (6.0)	6.0	1–25
Heart Disease Fact Questionnaire	19.0 (3.5)	20.0	6–25
Perceived Risk of HD scale	53.1 (5.8)	53.0	27–68

Table 2

Sample Demographics of Categorical Variables (N = 130)

	n	**(%)**
Male gender	81	62.3%
Employment status (unemployed)	90	69.2%
Race/Ethnicity		
White	54	41.5%
Black	42	32.3%
Hispanic	30	23.1%
Other	4	3.1%
Hypertension Stage		
Normal	43	33.1%
Pre-Hypertension	63	48.5%
Stage 1 Hypertension	22	16.9%
Stage 2 Hypertension	2	1.5%
Type of ART Regimen		
NNRTI-based	47	36.2%
PI-based	54	41.5%
Nuc-sparing	5	3.8%
Other type of regimen	8	6.2%
Not on ART	16	12.3%
Hepatitis C antibody positive	63	48.5%
Current smoker	73	56.2%
Involved in smoking cessation	10	7.7%
Never discussed CVD risk with HCP	99	76.2%

Of those screened but not enrolled (N = 40), the mean age was 51.0 (SD± 9.3**)** years, not significantly different from the study sample (p = .05). With respect to gender and ethnicity, 67.5% who did not participate were male and 52.5% were Hispanic.

Of the 40 individuals who were screened but did not participate, 32 (80%) were ineligible to participate and eight (20%) declined to participate due to a lack of time or interest. Of those who were ineligible (N = 32), sixteen (50%) did not speak and understand English, and sixteen (50%) had an existing diagnosis of CVD or had a history of a prior event, including a myocardial infarction, stroke or an intervention such as a coronary artery bypass graft.

Clinical variables. Mean years since diagnosis with HIV infection was 14.7 **(**SD± 7.9) years. Eighty-seven percent (87%) of the sample was currently taking antiretroviral (ART) medications, and 8.5% reported being naïve to treatment. Of those taking ART, 36.2% were taking a non-nucleoside reverse transcriptase inhibitor-based regimen and 41.5% were taking a protease inhibitor-based regimen. Mean CD4 count was 546 **(**SD± 292), and mean nadir CD4 was 195 (SD± 142). An undetectable HIV viral load was noted in 71.5% of participants. Clinical care was covered by publicly funded insurance for 68% of participants. A positive Hepatitis C antibody was noted in 49% of the study sample. Slightly more than 12% of participants were receiving methadone or suboxone maintenance therapy for opioid dependence.

Clinical and demographic variables, including age, smoking status, hypertension, and discussion of CVD with a health care provider, were examined for differences based on gender. No significant differences were found, as noted in Table 3.

Table 3

Gender Differences in Key Variables (N = 130)

Variable	Males n = 81 **(Mean± SD)**	Females n = 49 **(Mean± SD)**	*p*
Age	48.2 (8.8)	47.6 (7.6)	.69
Heart Disease Fact Questionnaire	19.1 (3.5)	18.7 (3.5)	.57
Perceived Risk of Heart Disease	53.3 (5.9)	52.9 (5.7	.67
	N (%)	**N (%)**	**$\chi 2$ (*p*)**
Current Smoker	45 (55.6%)	28 (57.1%)	.03 (.86)
Pre-Hypertension	44 (54.3%)	19 (38.8%)	1.7 (.43)
Discussed CVD with HCP	21 (25.9%)	9 (18.4%)	5.1 (.16)

Cardiovascular risk factor variables. Mean total cholesterol of the sample was 170 (SD± 36) and mean LDL was 97 (SD± 33). Mean HDL was 44 (SD± 17). Mean fasting glucose was 96 (SD± 25). Fifty-six percent (56%) of participants self-identified as current smokers, with a mean number of cigarettes per day of 10 (SD± 6.8), for a mean 27 (SD± 12.3) years of smoking. Less than 8% of participants reported being involved in smoking cessation efforts at the time of the interview. Only 7% of participants reported that they were taking a daily aspirin, and 10% were diagnosed with diabetes mellitus. Eleven (8.5%) participants were currently taking a prescription statin medication. When asked if they had ever discussed cardiovascular disease with their health care provider, 76.2% responded negatively. A family history of heart disease in a father (defined as myocardial infarction before age 55) was reported by 11.5% and heart disease in a mother (defined as myocardial infarction prior to age 65) was reported by 16.2%. Mean BMI was 27 (SD± 5.5) with 58.5% of participants in the overweight category (BMI > 25). When categorizing blood pressure measurements according to the JNC7 classification categories, 48.5% of participants had a blood pressure reading within the prehypertension range, defined as a systolic blood pressure between 120 and 139 or a diastolic blood pressure between 80 and -89 (National Heart Lung And Blood Institute, 2010).

Framingham Risk Score. The mean Framingham Risk Score (estimated risk) was 7.9 (SD ± 6.0) with 67%, 21%, and 12% of participants scoring in the low-, moderate-, and high-risk categories, respectively.

Perception of Risk of Heart Disease Scale (PRHDS). After imputing means for 9 missing items, the mean total score on the PRHDS was 53.1 (SD ± 5.9). Possible range

of this scale score was 20–80. A Cronbach's alpha of .78 demonstrated good internal consistency of the scale when used with this population.

CHD risk factor knowledge. The mean score on the Heart Disease Fact Questionnaire (HDFQ) was 19 (SD± 3.5). The possible range of scores on this scale was 0–25. Using the Kuder-Richardson formula to examine internal consistency, the scale demonstrated good reliability (.74). It was noted that 126 (97%) of participants knew that smoking was a risk factor for heart disease; however, only 86 (66%) of participants knew that older age was associated with an increased risk of heart disease and only 33 (25%) understood the gender differences in CVD risk. Table 4 presents the responses that were answered correctly most and least often on the HDFQ.

Table 4

Five Questions on Heart Disease Fact Questionnaire Answered Correctly Most and Least Frequently (N =130)

	Number (%) who correctly answered question
Five questions correctly answered most often:	
Being overweight increases a person's risk for heart disease	127 (97.7%)
Smoking is a risk factor for heart disease	126 (96.9%)
Eating fatty foods does not affect blood cholesterol levels	120 (92.3%)
High cholesterol is a risk factor for developing heart disease	119 (91.5%)
Regular physical activity will lower a person's chance of getting heart disease	118 (90.8%)
Five questions correctly answered least often:	
The older a person is, the greater their risk of having heart disease	86 (66.2%)
People with diabetes rarely have high cholesterol	77 (59.2%)
If your good cholesterol (HDL) is high you are at risk for heart disease	48 (36.9%)
People with diabetes tend to have a low HDL (good) cholesterol	77 (59.2%)
Men with diabetes have a higher risk of heart disease than women with diabetes	33 (25.4%)

Aim #2—Relationship Between Estimated and Perceived Risk of CVD

Descriptive statistics were computed for the Framingham Risk Score and these data were found to be significantly skewed (Fisher's measure of skewness = 4.40). Therefore these data were transformed using a log transformation to achieve a normal distribution. The Perception of Risk of Heart Disease scale scores were normally distributed. A significant, though weak, positive correlation was found (r [126] = .24, p = .01) between *estimated* and *perceived risk* of heart disease. A partial correlation was run between *estimated* and *perceived risk* while controlling for age and a similar positive correlation was noted, r (126) = .22, $p < .05$.

The study sample was divided into two subgroups by age to examine whether participants greater than 40 years of age had a stronger correlation between estimated and perceived risk than those less than or equal to 40 years of age. Interestingly, for the over-40 age group, the correlation was weaker, (r [100[= .19, p = .06), but the correlation for participants 40 years or less became stronger (r [26] = .41, p = .03).

A significant, strong positive correlation was noted between *age* and *estimated risk*, r (126) = .57, $p < .01$. No significant relationship was found between *age* and *perceived risk*, r (130) = .112, p = .20.

Aim #3—Influence of CVD Risk Factor Knowledge on Perceived Risk of CVD

To examine the influence of CVD risk factor knowledge (predictor variable) on perceived risk of CVD (outcome variable) while adjusting for covariates, linear regression was used. The model assumptions for regression were satisfied. The scatterplot for perceived risk and risk factor knowledge was linear in distribution. The assumptions of normally distributed residuals were met based on the bell-shaped

histogram. The K-S statistic was not statistically significant (p = .79) consistent with normally distributed residuals. Also, there did not appear to be any influential observations (Cook's distance < 1). Controlling for age, risk factor knowledge was not predictive of perceived risk (F [1,117] = .130, p > .05).

To determine which covariates would be entered into the linear regression model, correlations were run to examine for potential relationships between the demographic variables and *perceived risk of CVD* and *CVD risk factor knowledge*. When these were examined, there were no significant relationships found between *age*, *years since HIV diagnosis*, or *years of education*, and *perceived risk of CVD* and *CVD risk factor knowledge.* Correlation statistics and corresponding p values are listed in Table 5.

Table 5

Correlation Matrix Between Perceived Risk of CVD, Risk Factor Knowledge, and Demographic Variables (N = 130)

Variable	Age	Years of education	Years since HIV DX
		Pearson Correlation	
Perceived risk of CVD	.110	.040	.002
P value	.20	.64	.98
		Spearman's Rho Correlation	
Risk factor knowledge	.09	.14	.11
P value	.27	.09	.21

An independent samples t-test was conducted comparing the mean score of the Heart Disease Fact Questionnaire of participants by gender. No significant difference was found between male participants (M = 19.13, SD = 3.56) and female participants (M = 18.77, SD = 3.56), (t [128] = -.559, p = .57). Also, when comparing risk factor knowledge by HIV medication status, there was no significant difference in HDFQ scores

between those currently taking ART (M =18.97, SD = 3.6) and those who took ART in the past (M = 19.1, SD = 1.1), (t [117] = -.13, *p* = .89) or between those currently taking ART and those who were naïve to ART (M = 19.1, SD = 4.0), (t [122] = .18, *p* = .85).

A Chi-square test of independence was calculated to compare scores on the Heart Disease Fact Questionnaire by race and ethnicity. No significant relationship was found ($\chi 2(64) = 78.95$, $p > 0.05$).

Similarly, an independent samples t-test was conducted comparing the mean scores on the Perception of Risk of Heart Disease scale by gender. No significant difference was found (t [130] = -.42, *p* = .67). The mean score of female participants (N = 49) was (M = 52.8, SD = 5.71), and this was not significantly different from the mean score of male participants (N = 81), (M = 53.3, SD = 5.97). Also, when comparing perceived risk by HIV medication status, there was no significant difference in PRHDS scores between those currently taking ART (M = 53.0, SD = 6.0) and those who took ART in the past (M = 51.1, SD = 1.8), (t [119] = .76, *p* = .44) or between those currently taking ART and those who were naïve to ART (M = 54.7, SD = 4.8), (t [124] = .86, *p* = .39).

The scores on the Perception of Risk of Heart Disease Scale were compared by education category using a one-way ANOVA. Participants' education level was recategorized as being less than a high-school (HS) graduate (n = 54, 41.5%), HS graduate (n = 36, 27.7%), or having some college education (n = 40, 30.8%). No significant difference between groups was found (F [2,127] = 1.2, *p* = .29). The participants who had less than a HS graduate degree (M = 52.1, SD = 6.1) did not differ

significantly from those with a HS degree (M = 53.7, SD = 4.2) or those with some college level education (M = 53.8, SD = 6.6) on their PRHDS scores.

Additionally, the Chi-square test of independence was calculated to examine for differences on the Perception of Risk of Heart Disease scale by race and ethnicity, and no significance relationship was found ($\chi 2[132] = 97.7$, $p > .05$).

Since there was a significant level of current smoking in this cohort (56%), the Perception of Risk of Heart Disease and CHD Risk Factor Knowledge scale scores were examined for differences based on smoking status. Using the independent samples t-test statistic, no significant difference on the PRHDS was found (t [130] = -1.95, $p = .053$). The mean score of smokers (N = 73) was (M = 54.0, SD = 5.54), and this was not significantly different from the mean score of nonsmokers (N = 57), (M = 52.0, SD = 6.10). Additionally, when examining the HDFQ scale scores by smoking status, no significant differences were found (t [130] = .05, $p = .96$). The mean score of current smokers on the HDFQ was (M = 18.9, SD = 3.60), and this was not significantly different from the mean score of nonsmokers (M = 19.0, SD = 3.51).

Summary of Results

Results of this study demonstrated that HIV-infected patients have a high prevalence of cigarette smoking, a major traditional risk factor for cardiovascular disease. However, very few were involved in cessation efforts, and a majority reported that they had not discussed CVD risk with their health care providers. Almost half of the participants had a BMI consistent with being overweight. Most patients were taking antiretroviral therapy and had an undetectable viral load, indicating good ART adherence. Mean T cell count was above 500, demonstrating good immune reconstitution; and, this

clinical indicator is associated with a lower risk of morbidity and mortality due to HIV infection, thus highlighting the importance of addressing co-morbidities in this population.

Nearly half of the participants had a mean systolic or diastolic blood pressure reading consistent with a diagnosis of prehypertension. One-third of the sample had more than a 10% risk of having a cardiovascular event in the next 10 years, as demonstrated by the Framingham Risk Score. Most patients had a fair degree of CVD risk factor knowledge; however, key concepts were not understood, such as the effect of increasing age on increased cardiovascular risk. The perceived risk of CVD was weakly associated with estimated risk, even when controlling for the age of the participant. However, the level of cardiovascular risk factor knowledge was not predictive of perceived risk of cardiovascular disease. Implications and further discussion of these findings will be addressed in the following chapter.

CHAPTER 5

Discussion

The purpose of this study was to describe the estimated and perceived risk of CVD and the level of risk factor knowledge in HIV-infected adults. Additionally, it described the relationship between estimated and perceived risk, and examined the influence of risk factor knowledge on perceived risk of CVD. The Health Belief Model was used to guide this study. This chapter will discuss and analyze the study findings that were presented in the previous chapter and will comment on the usefulness of the model as a framework for this study. Also, study limitations, implications for nursing practice, and directions for future research will be discussed.

The Changing Paradigm of HIV Care

Numerous studies suggest that HIV-infected adults on ART have lower rates of morbidity and mortality related to HIV infection and that survival is comparable to that of the general population (Bhaskaran et al., 2008; Lewden et al., 2007). Moreover, it is estimated that by 2015 more than half of those infected with HIV will be greater than 50 years of age (Effros et al., 2008). The participants in this study had a mean age of 48 years and had been HIV-infected, on average, for more than 14 years, suggesting that many had been infected in the mid-1990s. During this period, the epidemic was at its peak; drug development research was intense, and combination therapy was introduced, quickly becoming the standard of care. The prevalence of persons living with HIV increased steadily and deaths due to AIDS decreased significantly. In this study, 87% of participants were taking ART successfully, had a mean CD4 count greater than 500, and had good adherence; nearly two-thirds (71.5%) of participants had an undetectable HIV

viral load. These data exemplify the concept of HIV as a chronic illness and reinforce the need to direct more attention to the management and prevention of comorbidities that are more prevalent in the general population, such as cardiovascular disease.

Prevalence of CVD Risk Factors

In this study, traditional risk factors that were strong contributors to the elevated risk of CVD were smoking, elevated BMI, and prehypertension. The contribution of such risk factors to an increased risk of CVD in the HIV-infected population is well documented (Friis-Moller et al., 2003; Glass et al., 2006; Kaplan et al., 2007).

Smoking. In this study, current smoking was reported by 56% of participants, with a mean number of cigarettes per day of ten. In the general population, smoking prevalence is approximately 21% (U.S. Centers for Disease Control and Prevention, 2010). In the literature, the rate of smoking noted in the HIV-infected population is often substantially higher than the general population (Benard et al., 2007; Burkhalter, Springer, Chhabra, Ostroff, & Rapkin, 2005; Capilli, Anastasi, & Ogedegbe, 2011; Cockerham et al., 2010; Crothers et al., 2009; Friis-Moller et al., 2003; Glass et al., 2006; Kaplan et al., 2007; Mamary, Bahrs, & Martinez, 2002; Sterne et al., 2007). In the D:A:D study, the rate of current or previous smokers was 56% (Friis-Moller et al., 2003), which was related to a relative risk for MI that was twice that of the nonsmokers. Similarly, the Swiss Cohort found that current smoking was the most common CVD risk factor with a reported prevalence of 57% (Glass et al., 2006). Crothers et al. (2009) found that in a group of HIV-infected veterans, smoking was associated with an increased risk for mortality when compared to HIV-infected never smokers (RR 2.31, 95% CI 1.53–3.49, p

< .05). These data highlight the importance of addressing smoking cessation efforts in this group of individuals in the clinical setting.

Smoking cessation programs and clinical protocols often exclude participants with chronic illnesses, such as HIV infection. In our study, less than 8% of smokers reported being currently involved in smoking cessation efforts. However, several studies have demonstrated that HIV-infected smokers express a willingness to attempt smoking cessation. Mamary et al. (2002) found that two-thirds of current smokers in an urban HIV clinic setting were currently thinking about quitting smoking. Burkhalter et al. (2005) found that 42% of HIV-infected smokers were in the precontemplation stage of readiness, 40% were currently contemplating smoking cessation, and 18% were preparing to quit smoking. Benard found that 40% of current smokers were motivated to quit smoking. Often, a psychiatric comorbidity, such as depression, or the use of illicit drugs or alcohol, occurs in tandem with smoking in the HIV-infected population (Benard et al., 2007; Burkhalter et al., 2005).

The development of comprehensive smoking cessation programs for HIV-infected adults is critical to reduce the prevalence of current smoking and subsequently reduce cardiovascular risk in this population. Interestingly, 97% of participants in this study were aware that smoking was a risk factor for CVD. Programs that address depression, drug and alcohol use, and smoking cessation may be the most effective. Self-efficacy has been found to be a factor related to the success in these efforts (Lloyd-Richardson et al., 2008; Vidrine, Arduino, Lazev, & Gritz, 2006). Practical programs that can be incorporated into the clinical visit and focus on the specific needs of HIV-infected smokers are greatly needed.

BMI. A body mass index of greater than 25 is consistent with being overweight. In this study, the mean BMI was 27, and 58.5% of participants had a BMI of greater than 25. In a study designed to evaluate cardiovascular risk factors and Framingham Risk Scores among HIV-infected men and women, Kaplan et al. (2007) reported that more than 40% of men and more than 60% of women with HIV had BMIs greater than 25. Also, he found that being overweight was associated with an increased estimated CVD risk (measured by FRS). Similarly, Capilli et al. (2011) and Janiszewski et al. (2011) found that having a BMI greater than 25 was found to be associated with a higher waist circumference; and this was associated with increased cardiovascular risk in HIV-infected adults (Janiszewski et al., 2011). Dube et al. (2010) did not find an association between BMI and endothelial dysfunction (often a marker for increased CVD risk); however, a higher BMI has been related to the development of metabolic syndrome and diabetes mellitus, which are both associated with increased cardiovascular risk. Mangilli et al. (2007) found that the presence of metabolic syndrome in HIV-infected adults was associated with increased common c-IMT scores and coronary artery calcium scores.

Studies examining the effect of weight loss and exercise on cardiovascular risk in HIV-infected adults are lacking. One prospective study in overweight adults in the general population supported that weight loss through diet and exercise over an 18-month period could effect change in weight and significantly reduced cardiovascular risk, measured by Framingham Risk Scores (RR 0.86, CI 0.81–0.91, $p < .001$; Maruthur, Wang, & Appel, 2009). Tailored exercise and weight loss intervention studies designed for HIV-infected adults to reduce BMI and waist circumference could potentially lower the risk for metabolic syndrome, diabetes, and cardiovascular disease in these patients.

Prehypertension. Prehypertension, defined as a blood pressure of 120–139 systolic or 80–89 diastolic, is highly prevalent in the United States, affecting approximately 70 million individuals (Chobanion et al., 2003). Prehypertension, as a diagnosis, was identified in 2003 by the JNC7 report and was introduced to designate individuals who may be at increased risk for the development of cardiovascular disease (Chobanion, 2006). In this cohort, 48.5% of participants had a mean blood pressure consistent with a diagnosis of prehypertension. In the general population, prehypertension prevalences of up to 40% have been reported (Chobanian, 2006; Chobanian et al., 2003; Hernandez & Anderson, 2011). Furthermore, individuals with prehypertension have been found to have increased Framingham Risk Scores (Qureshi, Suri, Kirmani, Divani, & Mohammad, 2005), and were more likely to develop CVD (Vasan et al., 2001). One cross-sectional study conducted in Brazil identified that age, male gender, and elevated BMI were associated with prehypertension in HIV-infected adults (Roque de Arruda Junior et al., 2010). An abstract presented at the Conference on Retroviruses and Opportunistic Infections (CROI) in Seattle in March 2012 presented evidence that in a Veterans Health Administration study, prehypertension in HIV-infected adults was associated with a relative risk of MI of 1.88 (CI 1.19–2.99) when compared to HIV-negative controls (Armah et al., 2012). Management of prehypertension focuses on lifestyle modification—namely reduction of weight, adoption of a DASH diet, reduction in dietary sodium, increase in physical activity, and reduction of alcohol consumption. Current evidence demonstrates that reduction of blood pressure levels to within the normal range through middle age substantially reduces lifetime cardiovascular risk (Chobanian, 2006; Chobanian et al., 2003). This is an important consideration for the

clinical care of HIV-infected adults, and it may be important for future clinical research to modify CVD risk.

Estimated Risk/Framingham Risk Score (FRS)

This cohort of HIV-infected adults without a previous history of cardiovascular disease had a mean FRS of 7.9, a score that is consistent with the upper limit of low risk (defined as a FRS less than 10%). Lo et al. (2010), De Socio et al. (2007), and Falcone et al. (2011) reported similar results (mean FRS 7.7 ± 5.1, mean age 46.5; mean FRS 7.0 ± 5.0, mean age 43.0; and mean FRS 7.0 ± 5.2, mean age 44, respectively) in HIV-infected men and women. When participants were stratified into FRS categories, it was noted that 32% of this sample had Framingham Risk Scores consistent with moderate and high risk. Several large cross-sectional studies reported that 14–33% of HIV-adults met criteria for moderate risk (Glass et al., 2006; Hadigan et al., 2003; Sayler et al., 2006) and 3–12% met criteria for high risk (Bergesen et al., 2004; Glass et al., 2006; Law et al., 2006). Studies suggest that the FRS, which was not developed for HIV-infected adults, may not be a reliable measure of CVD risk for this population and suggest that it underestimates the risk of MI in individuals on antiretroviral therapy (Law et al., 2006). This implies that a significant percentage of HIV-infected adults have a considerable 10-year risk of having a CVD event and these individuals may be at even higher risk than is actually predicted by the FRS. These data have substantial implications for the management of adults with HIV infection. Lifestyle modification and the development of preventive therapeutic interventions may be advantageous.

CVD Risk Factor Knowledge

The mean CVD knowledge score in this study, using the HDFQ, was 19 (76%). Generally, a score of less than 70% is considered a failure on a standardized test. Nearly one-third of participants (27.7%) had a score less than 70%. Knowledge scores did not differ by age, gender, years living with HIV infection, or years of education. CVD knowledge had not previously been measured in an HIV-infected cohort. In diabetics, the mean knowledge score using the same instrument was 20.4, and significant differences by ethnic group were reported (Wagner et al., 2006). Female gender, higher education, and having health insurance was predictive of higher HDFQ scores. This study had a much larger sample size (N = 678), older cohort (mean age = 53.7), and was 62% female; 92% had health insurance, and only 4% of participants had less than a high school education. In a study of over 3,000 participants with a history of ischemic heart disease, Dracup et al. (2008), using a different scale, reported a mean CVD knowledge score of 71%.

The mean knowledge score in this study is fairly high considering that the cohort was younger, had a higher percentage of non-high school graduates, and more men. This level of risk factor knowledge may be attributed to rigorous public health campaigns such as the American Heart Association's *Go Red for Women* campaign (American Heart Association, 2007) to recognize heart disease in women, and more recently, the Department of Health and Human Services Million Hearts and Heart Truth initiatives (DHHS, 2011). Despite the level of knowledge reported in this study though, more than three quarters of participants reported that they had never discussed heart disease with their health care providers.

When individual items on the knowledge scale were examined, it was notable that approximately 97% of participants knew that smoking and being overweight were risk factors for heart disease. Furthermore, more than 90% knew that a high cholesterol, high fat diet, and lack of exercise could contribute to an increased risk for heart disease. Diet and exercise were not addressed in this study; however, smoking cessation was, and few participants reported being involved in efforts to stop smoking, inferring that knowledge alone is not an adequate motivator for behavior change. In several studies examining cardiovascular health-promoting behaviors, perceived barriers, perceived susceptibility, and self-efficacy were most significantly correlated with adoption of new behaviors (Janz, 1988; Janz & Becker, 1984; Wilson et al., 1997). Knowledge was a significant predictor in one HIV prevention study examining condom use, but this is more skill-related than health knowledge (Volk & Koopman, 2001). Efforts to broaden CVD risk factor knowledge and more importantly to link risk factor knowledge to risk perception and health-related consequences are needed.

Correlation Between Estimated and Perceived Risk of CVD

A significant, though weak, positive correlation was found between estimated and perceived risk of cardiovascular disease (r = .24). Perceived risk of CVD had not been previously measured in the HIV-infected population. In the general population, one study found that 40% of diabetic patients were unable to give an estimate of CVD risk, and agreement between risk perception and clinical data was weak (Martell-Claros et al., 2011). Alwan et al. (2009) had similar findings with only half of participants able to provide an estimate of their perceived risk, and the ability to do so was associated with higher socioeconomic status (Alwan et al., 2009). Homko et al. (2010) found that there

was no relationship between actual and perceived risk in a group of 211 adults with Type 2 diabetes (Homko et al., 2010). Barnhart et al. (2009) reported that perceived risk and actual risk were significantly correlated with a similar strength correlation (r = .22), as we found in our study. Age was not found to be associated with ability to perceive risk in any of these studies, so it is not surprising that we did not find this association either.

When the study sample was divided by age, it was found that for participants over 40 years, the correlation was weaker (r = .14), but for participants less than 40 years, the correlation became much stronger (r = .46). This seems counterintuitive but can be explained by two factors—the variability in the over-40 age group was lower; hence a lower correlation. Also, there was an outlier in the PRHDS score in the over-40 age group, again contributing to the lower correlation.

Influence of Knowledge on Perceived Risk of CVD

The final aim of this study was to examine the influence of CVD risk factor knowledge on perceived risk of CVD. Results of this study demonstrated that in HIV-infected adults, risk factor knowledge was not predictive of perceived risk of CVD. Previous studies in the HIV population have not been done. Often though in other populations, such as adults with Type 2 diabetes, knowledge has been predictive of risk (Choi et al., 2008; Homko et al., 2010). Also, the relationship between knowledge and perceived risk is suggested in the Health Belief Model. However, several studies using the Health Belief Model failed to demonstrate this relationship (Becker & Levine, 1987; Hart, 2005).

This author suggests that the HIV-infected population is inherently different from other populations, such as diabetics or women, with respect to perception of

cardiovascular risk for a historical reason. During its first 20 years in the United States, HIV was closely associated with mortality due to the lack of effective treatments. Until the early 1990s, most individuals diagnosed with this disease developed AIDS and eventually died, often within a short period of time. Other chronic illnesses such as diabetes do not share this same history. So by nature of history, individuals with HIV are different in their perspective and perceptions. In this study, the mean age was 48 years and the mean years of HIV infection were 14.7, so this sample consists of individuals to whom the epidemic's history is well known.

Paterson (2001) has proposed the Shifting Perspectives Model of Chronic Illness, a midrange theory that describes what it is like for individuals who live with chronic illnesses and offers an explanation of behavior. The model suggests that individuals living with a chronic illness are "continually shifting between the perspectives of wellness in the foreground and illness in the foreground in order to make sense of" their world at any given time (Paterson, 2001, p. 988). Having the perspective of illness in the foreground is consuming, and the individual is focused on the condition itself, the symptoms, the burden of self-care, and the potential negative outcomes associated with the illness. In this perspective, the individual is unable to perceive of concepts associated with wellness, may lack motivation for health promotion and prevention activities, or be uninterested in preventing events that may occur in the future. HIV-infected adults may have an illness in the foreground perspective and, for this reason, a focus on perception of risk for CVD is inconceivable for them.

Utility of the Health Belief Model

The Health Belief Model is a predictive model of behavior change, and it was used in this descriptive study to describe concepts and potential relationships related to cardiovascular risk in HIV-infected adults. The study did not support the relationships suggested by the Health Belief Model. In the model, knowledge, age, gender, race/ethnicity, and education level are all sociodemographic and structural variables that are supposed to influence perceived risk. In this study, none of these relationships were found. Several reasons may explain this. Concepts within the model are not clearly defined and may be operationalized differently across studies leading to difficulty when attempting to observe clear relationships and associations between the concepts. The development of standardized scales to measure constructs in the model may facilitate measurement and interpretation of results, and allow comparison of findings across studies.

In this study, the correlation between estimated and perceived risk was weak. This is consistent with the literature—the model is often criticized because the variables proposed in the model correlate weakly with each other and effect sizes were often small, $r < .21$ (Armitage & Conner, 2000). Rosenstock, the developer of the model, attributed the lack of predictability, small effect size, and low level of variance to the exclusion of the self-efficacy variable, and therefore included self-efficacy in a later version of the model. Since it is a predictive model, the Health Belief Model is probably best utilized as the theoretical framework for an intervention study, measuring and examining all potential predictors of behavior change over time.

Study Limitations

This study has some limitations. The use of a convenience sample and the cross-sectional nature of the study may have been limiting factors. Patients who felt they were not at risk or, conversely, were at high risk may have self-selected to not participate. Also, the sample consisted of patients who were actively engaged in care and may be different from those not seeking care. Data were collected by interview and therefore may have been affected by recall and social desirability bias. Also, the findings may not be generalizable to all patients infected with HIV since this study excluded individuals with known CVD and those who did not speak and understand English.

The instruments that were used in this study to measure perceived risk (developed for primary care patients in Jordan) and risk factor knowledge (developed for diabetic patients) were not developed for HIV-infected patients. Although they demonstrated good reliability in this study, they may not have been sensitive or specific enough to detect strong associations between the variables under study. Also, the Framingham Risk Score was not developed for individuals with HIV infection, and its reliability with this population is not known.

Study Strengths

This is the first study to measure cardiovascular risk factor knowledge and perceived risk of cardiovascular disease in HIV-infected adults in a clinic setting. The sample was heterogeneous, with 58.5% from ethnic and racial minorities. Additionally, use of the interview method for data collection resulted in a very low rate of missing data (less than 5%).

Implications for Research and Practice

This study supports the need to develop innovative programs to reduce cardiovascular risk in HIV-infected adults. The high prevalence of smoking highlights the need to have tailored interventions to assist this population in its smoking cessation efforts. Often, smoking cessation programs and research protocols have strict inclusion and exclusion criteria and exclude those with chronic illnesses, such as HIV infection. The development of smoking cessation trials specifically designed to meet the needs of the HIV-infected patient are much needed.

This study provides evidence that there is a need to develop reliable and valid instruments that will accurately measure perceived risk of CVD, CVD risk factor knowledge, and estimated (actual) risk of CVD in HIV-infected adults. The PRHDS scale contained items that were poorly understood by some participants with lower literacy or education levels and may need to be rewritten to include simpler language. The HDFQ had several items that were focused on heart disease risk factors in diabetic adults that may have not been applicable to this population. An instrument to measure CVD risk factor knowledge in HIV-infected patients may be a more accurate measure of knowledge in this group of adults. The literature suggests that the FRS underestimates risk of disease in HIV-infected adults and, until an HIV-specific risk equation is developed and validated, prediction of risk in this population may be imprecise.

Finally, the study supports the need to develop education programs to improve cardiovascular risk factor knowledge in this population, especially focusing on the role that advancing age, hypertension, obesity, and hyperlipidemia play in increasing cardiovascular risk. General CVD risk factor knowledge in this cohort was fairly high,

but it was not found to influence perception of risk. Perhaps HIV-infected adults do not personalize the general knowledge that they have about heart disease. Perhaps specific messages and interventions tailored to the individual in the clinical setting are needed. Calculating Framingham Risk Scores during clinical visits for each patient may help to personalize the information about CVD risk and increase their perception of risk for heart disease.

Conclusion

This study examined perceived risk of cardiovascular disease and knowledge of cardiovascular risk factors in HIV-infected adults. Findings suggest that HIV-infected adults are at risk for developing cardiovascular disease due to the high prevalence of traditional cardiovascular risk factors, such as smoking and elevated blood pressure. However, the study demonstrated that this group of individuals does not have a high level of risk factor knowledge and, more importantly, their level of knowledge did not influence their perception of risk for cardiovascular disease. As HIV-infected adults continue to age due to the advances in HIV treatment, researchers and health care providers will face the challenge of finding effective methods to estimate cardiovascular risk, assess risk perception, increase patients' level of risk factor knowledge, and ultimately develop interventions to effect behavior change, reduce risk, and improve cardiovascular health in this population.

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Appendix A

Data Collection Instrument

Study ID #: _____ DATE: _____

Site: (1) RI Hospital (2) Miriam Hospital

Interviewer: (1) Principal Investigator (2) Research Assistant

Age: _____ years

Gender: (1) female (2) male

Employment Status: (1) employed (2) unemployed

Education level: _____ # of years completed

Years since HIV diagnosis: _____ years

Race/Ethnicity:

(1) White
(2) African American
(3) Hispanic
(4) Asian American
(5) Pacific American
(6) Native American
(7) Other
(8) Unknown

Health Insurance: (1) none (2) private (3) public

HCV Status: (0) don't know/not tested (1) positive (2) negative

Current smoker: (0) no (1) yes (3) unknown

If Yes: _____ # of cigs/day _____ # years smoked

Are you currently engaged in any activities or a program to stop smoking?

(0) no (1) yes (2) don't know

Are you currently taking a daily aspirin?

(0) no (1) yes (2) sometimes (3) don't know

Are you taking medications for diabetes?

(0) no (1) yes (2) don't know

Are you currently taking, or have you in the past taken, medications for HIV?

(0) never took HIV medications (naïve)
(1) currently taking HIV medications (on ART)
(2) took HIV medications in the past, but not taking currently (on treatment interruption)

Have you ever discussed CVD (heart disease) risk with you provider?

(0) no (1) yes (3) don't know/can't remember

Family History of CVD:

MI in father <age 55: (0) no (1) yes (2) unknown

MI in mother <age 65: (0) no (1) yes (2) unknown

Diabetes in participant: (0) no (1) yes

Weight: _____ lbs **Height**: _____ **Calculated BMI**: _____

Blood Pressure: (1) _____ (2) _____ (3) _____

Current Medications:

_____
_____
_____
_____
_____

CLINICAL CHART DATA EXTRACTION:

Total Cholesterol: _____ Triglycerides:_____

LDL: _____ HDL: _____

Fasting Glucose: _____

Current CD4 Count: _____

Nadir CD4 Count: _____

Current HIV- PVL: _____

Framingham Risk Score: _____ % risk

APPENDIX B

Perception of Risk of Heart Disease Scale (PRHDS)

	Strongly Disagree	Disagree	Agree	Strongly Agree
1. There is a possibility that I have heart disease.	1	2	3	4
2. There is a good chance that I will get heart disease during the next 10 years.	1	2	3	4
3. A person who gets heart disease has no chance of being cured.	1	2	3	4
4. I have a high chance of getting heart disease because of my past behaviors.	1	2	3	4
5. I feel sure that I will get heart disease.	1	2	3	4
6. Healthy lifestyle habits are unattainable.	1	2	3	4
7. It is likely that I will get heart disease.	1	2	3	4
8. I am at risk for getting heart disease.	1	2	3	4
9. It is possible that I will get heart disease.	1	2	3	4
10. I am not doing anything now that is unhealthy to my heart.	1	2	3	4
11. I am too young to have heart disease.	1	2	3	4
12. People like me do not get heart disease.	1	2	3	4

	Strongly Disagree	Disagree	Agree	Strongly Agree
13. I am very healthy so my body can fight off heart disease.	1	2	3	4
14. I am not worried that I might get heart disease.	1	2	3	4
15. People my age are too young to get heart disease.	1	2	3	4
16. People my age do not get heart disease.	1	2	3	4
17. My lifestyle habits do not put me at risk for heart disease.	1	2	3	4
18. No matter what I do, if I am going to get heart disease, I will get it.	1	2	3	4
19. People who don't get heart disease are just plain lucky.	1	2	3	4
20. The causes of heart disease are unknown.	1	2	3	4

APPENDIX C

Heart Disease Fact Questionnaire (HDFQ)

1. A person always knows when they have heart disease.	True	False	Don't know
2. If you have a family history of heart disease, you are at risk for developing heart disease.	True	False	Don't Know
3. The older a person is, the greater their risk of having heart disease.	True	False	Don't Know
4. Smoking is a risk factor for heart disease.	True	False	Don't Know
5. A person who stops smoking will lower their risk of heart disease.	True	False	Don't Know
6. High blood pressure is a risk factor for heart disease.	True	False	Don't Know
7. Keeping blood pressure under control will reduce a person's risk for developing heart disease.	True	False	Don't Know
8. High cholesterol is a risk factor for developing heart disease.	True	False	Don't Know
9. Eating fatty foods does not affect blood cholesterol levels.	True	False	Don't Know
10. If your good cholesterol (HDL) is high you are at risk for heart disease.	True	False	Don't Know
11. If your bad cholesterol (LDL) is high you are at risk for heart disease.	True	False	Don't Know
12. Being overweight increases a person's risk for heart disease.	True	False	Don't Know
13. Regular physical activity will lower a person's chance of getting heart disease.	True	False	Don't Know
14. Only exercising at a gym or in an exercise class will lower a person's chance of developing heart disease.	True	False	Don't Know

15. Walking and gardening are considered exercise that will help lower a person's chance of developing heart disease.	True	False	Don't Know
16. Diabetes is a risk factor for developing heart disease.	True	False	Don't Know
17. High blood sugar puts a strain on the heart.	True	False	Don't Know
18. If your blood sugar is high over several months it can cause your cholesterol level to go up and increase your risk of heart disease.	True	False	Don't Know
19. A person who has diabetes can reduce their risk of developing heart disease if they keep their blood sugar levels under control.	True	False	Don't Know
20. People with diabetes rarely have high cholesterol.	True	False	Don't Know
21. If a person has diabetes, keeping their cholesterol under control will help lower their chance of having heart disease.	True	False	Don't Know
22. People with diabetes tend to have low HDL (good) cholesterol.	True	False	Don't Know
23. A person who has diabetes can reduce their risk of developing heart disease if they keep their blood pressure under control.	True	False	Don't Know
24. A person who has diabetes can reduce their risk of developing heart disease if they keep their weight under control.	True	False	Don't Know
25. Men with diabetes have a higher risk of heart disease than women with diabetes.	True	False	Don't Know