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HIV and General Cardiovascular Risk

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Abstract

The incidence of cardiovascular disease (CVD) is increasing in HIV-infected people. Risk factors such as hyperlipidemia, impaired glucose tolerance, and insulin resistance have become common. CVD in HIV may also be related to non-traditional risk factors including accumulation of visceral fat, inflammation secondary to HIV, and effects of some antiretroviral drugs. This cross-sectional study described the CVD risk factors of 123 adults living with HIV and calculated the 10-year estimate for general cardiovascular risk score. Results showed that approximately 25% of the participants were considered to be at high risk for developing CVD in the next 10 years. Increased waist circumference and longer duration of smoking habit were associated with elevated general cardiovascular risk scores. Similar to the general population, most of the identified risks could be modified through lifestyle management.

Keywords

cardiovascular disease; HIV infection; metabolic syndrome; obesity

The availability of effective antiretroviral therapy (ART) has dramatically reduced the morbidity and the mortality associated with HIV infection (Grinspoon et al., 2008). With prolonged life spans, an increased incidence of cardiovascular disease (CVD) has been reported for people living with HIV infection (PLWH; Grinspoon et al., 2008); up to 10% of deaths in PLWH have been associated with CVD (Sabin et al., 2008). CVD in this population may be related to traditional risk factors (i.e., advancing age, dyslipidemia, hypertension [HTN], diabetes mellitus [DM]; Carr et al., 1998), and lifestyle behaviors such as smoking, dietary intake, and lack of physical activity (Grinspoon et al., 2008). CVD may also be due to non-traditional risk factors including accumulation of visceral fat, inflammation secondary to HIV infection, and effects of some antiretroviral drugs, such as protease inhibitors (PIs) and nucleoside reverse transcriptase inhibitors (NRTI; Green, 2002;

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Kotler, 2003; Sabin et al., 2008). Recent observational data have shown that specific PIs (lopinavir/ritonavir and fosamprenavir; U.S. Food and Drug Administration [FDA], 2009) and NRTIs (abacavir and didanosine) have been linked with an increased risk for myocardial infarction (MI; Stein & Currier, 2008). At this time, the exact mechanism associated with CVD and HIV is not fully understood. Despite this limitation, estimating CVD risk in this population has been recommended to provide appropriate treatment strategies and to reduce risk (De Socio et al., 2008; Friis-Moller & Worm, 2007).

Several tools are available to estimate CVD risk. The Framingham Heart Study (2010), a well renowned, prospective, longitudinal study initiated in 1948, produced numerous tools to estimate CVD risk and identified common risk factors for CVD. The original cohort of Framingham study participants continues to be followed, in addition to their offspring, their offspring's spouses, and their grandchildren. Eleven Framingham Risk Score (FRS) profiles were developed to estimate various aspects of CVD risk including atrial fibrillation, stroke, coronary heart disease, intermittent claudication, and general CVD. FRS profiles have been validated in the North American population.

The general CVD FRS profile, derived from the original Framingham cohort data, was used for this study to estimate CVD risk. The general CVD FRS profile is composed of two separate FRS profiles, the General Cardiovascular Risk Profile (GCRP) and the Office-Based Non-Laboratory Risk Profile (OBNLRP). The GCRP and the OBNLRP are sex-specific, validated tools designed to estimate the 10-year risk for developing any major atherosclerotic CVD (coronary death, MI, coronary insufficiency, angina, ischemic stroke, hemorrhagic stroke, transient ischemic attack, peripheral artery disease; D'Agostino et al., 2008). Data from the original Framingham Heart Study cohort and the offspring study groups were used to validate these tools (Wilson et al., 1998). Study participants ($n = 8,491$), ages 30-74 years, without CVD were included in the study sample and followed for 12 years. The GCRP and OBNLRP have been useful for primary care providers because they avoid having to use multiple risk tools (or multiple FRS profiles) to assess for specific cardiovascular end points such as stroke or MI. The components used to determine CVD risk using either the GCRP or OBNLRP, are readily available in clinical practice; these include assessments of blood pressure, cholesterol levels, diabetes history, and smoking status.

In this study, we report on the first phase of a two-part study. The first phase collected information associated with cardiovascular risk factors, including diet and physical activity levels. The second phase used group interviews to better understand the perceptions and knowledge of PLWH regarding CVD risk and prevention. Sample size for phase 1 was determined by data saturation from the group interviews. For this phase 1 study, we wanted to describe the 10-year risk of developing CVD using the GCRP and the OBNLRP and how associations appeared when applied to a small cohort of people with HIV, because a specific HIV CVD risk assessment tool is not available and PLWH have demographics that may differ from the general population.

Methods

Research Design and Study Participants

The study was a cross-sectional design; enrollment began in February 2008 and data collection ended in January 2009. Participants were recruited using flyers posted in HIV community-based organizations and health fairs specific to HIV care. Potential participants initiated inquiry for the study and were telephone screened by a member of the study team to determine preliminary eligibility. Participants were eligible if they were at least 18 years of age, provided documentation of HIV status (written verification of HIV status by primary

care provider and/or possession of an HIV medication with participant's name on the label of the prescription bottle), achieved a score of 24 or higher on the Mini Mental State examination, and were able to read, write, and speak in English. Potential participants were excluded if they had a history of CVD, defined as self-report of MI, angina pectoris, coronary artery bypass graft surgery, coronary angioplasty or stent placement, symptoms of congestive heart failure, insertion of pacemaker or known atrial fibrillation, stroke, peripheral arterial disease (intermittent claudication), or history of surgery for peripheral arterial disease (Barzilay et al., 2001). The study was conducted in an academic medical center in New York City and received approval from the institutional review board.

Measures

Members of the research team (nurse practitioner and registered nurse) were trained in the study protocol to assist participants in the completion of study questionnaires and perform the following measurements: blood pressure, anthropometric measures, lipid profile, and blood glucose assessment. Appropriate referrals for medical follow-up were provided when indicated.

Cardiovascular risk factor assessment—Blood pressure was measured using an oscillometric device (Omron, Omron Healthcare Inc, Bannockburn, Ill). Team members were instructed to measure blood pressure after participants rested for 5 minutes in a sitting position. Cuff size was adjusted according to arm circumference. The arm was unrestricted by tight fitting clothing, legs uncrossed, feet flat on the floor, and arm supported so that the cuff was level with the heart. Three blood pressure measurements, 1 minute apart, was taken as recommended by the American Heart Association (Perloff et al., 1993). The mean of the readings was used in the analysis. Height was measured to the nearest centimeter using a stadiometer. Participants removed their shoes and stood with their backs against the vertical mounted ruler with their heels and feet together. Weight was obtained using a digital scale after shoes and heavy outer garments were removed. Body mass index (BMI) was calculated using the standard formula of weight (kg)/height (m²). Waist circumference was recorded using an inelastic tape measure with light clothing and belts removed. Waist was measured midway between the lower rib margin and iliac crest with the tape positioned around the body in a horizontal position. The measuring tape was held loose enough to allow the research team member to place one finger between the tape and the participant's body. Participants stood with their feet close together (approximately 12-15 cm) and weight equally distributed on each leg. Participants were asked to breathe normally and the reading of the measurement was taken at the end of exhalation.

The lipid profile and blood glucose assessments were obtained using the *CardioChek PA*[®] (Orlando, FL) portable whole blood and lipid testing system. This system used finger-stick technology to analyze the lipid profile and blood glucose. *CardioChek PA*[®] is Clinical Laboratory Improvement Amendments (CLIA) waived and results from this system have been shown to be comparable to traditional laboratory lipid profile analysis (correlation coefficients between *CardioChek PA*[®] and clinical diagnostic methods: total cholesterol, $r = .83$; triglyceride, $r = .94$; HDL, $r = .73$; LDL, $r = .86$; $p < .0001$ for all values; Panz, Raal, Paiker, Immelman, & Miles, 2005). The lipid profile consisted of triglyceride, total cholesterol, low-density lipoprotein (LDL), and high-density lipoprotein (HDL). Participants were requested to fast for 12 hours prior to the lipid and glucose testing.

Study questionnaires—Each participant completed a questionnaire consisting of demographic data, medical history, smoking habit, current and former intravenous drug use (IVDU), and family (parent or sibling) history of CVD and risk factors (i.e., HTN, DM, dyslipidemia). Smoking habit was categorized as *never smoked*, *former smoker*, or *current*

smoker. Former smoker was defined as cigarette free for 30 days or more (Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults, 2001). *Current smokers* listed the number of cigarettes smoked per day and the duration of habit. The quantity of cigarettes smoked per day was categorized as: (a) no cigarettes, (b) 1-15 cigarettes, (c) 16-25 cigarettes, and (d) more than 25 cigarettes (Prokhorov et al., 2000).

Participants also completed the International Physical Activity Questionnaire (IPAQ) and a 3-day prospective food diary. The IPAQ (short-form; Craig et al., 2003) is a 7-item instrument that uses a reference period from the previous 7 days. The test-retest reliability of the IPAQ was good, Spearman's $p = 0.76$ (95% CI 0.73-.77) and concurrent validity was assessed by comparing physical activity results on the long- and short-form versions of the instrument (Spearman's $p = .67$ [95% CI 0.64-0.70]). IPAQ measures three types of physical activity: walking, moderate intensity activities, and vigorous intensity activities. Separate scores for each activity and a total score for all activities in terms of duration in minutes and frequency in days were computed. The volume of activity was computed by weighting each type of activity by the energy requirements defined in metabolic equivalent of task (MET), to yield a score in MET-minutes and MET-minutes/week (Craig et al., 2003).

3-day food diary—Food diaries are prospective tools, used to estimate dietary intake (Schroder et al., 2001). With the 3-day food diary, participants were instructed to record all meals, snacks, and beverages, including portion size, quantity, and method of preparation consumed from 2 alternating work days (weekdays) and 1 weekend day. The accuracy of nutritional assessments has been shown to increase by examining both weekday and weekend consumption (Stotts & Bergstrom, 2004). The food diaries were analyzed using the *Food Processor SQL (FP SQL®)*, Salem, OR) software to determine nutritional intake.

GCRP and OBNLRP calculations—GCRP and OBNLRP risk scores were calculated by following established procedures (D'Agostino et al., 2008). GCRP risk scores were computed using age- and sex-specific categories for HDL, total cholesterol, systolic blood pressure (SBP), cigarette smoking status, and DM diagnosis. OBNLRP risk scores were also computed using age- and sex-specific categories for BMI, SBP, smoking status, and DM diagnosis. Age, smoking status, and DM diagnosis were based on self-report. Total points and percentages for a 10-year risk for general CVD were calculated from each measure. Both the GCRP and the OBNLRP risk scores were categorized as low risk (< 7%), moderate risk (7%-20%), and high risk (> 20%) based on classifications proposed by D'Agostino and team (2008). These tools have also demonstrated good discrimination and calibration (C statistic, 0.763[men] and 0.793 [women]; D'Agostino et al., 2008).

Definitions

Hypertension (HTN) was defined as SBP 140 mm Hg or greater and/or diastolic blood pressure (DBP) 90 mm Hg or greater or reported use of an anti-hypertension medication. Dyslipidemia was defined using the National Cholesterol Education Program (NCEP) guideline (Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults, 2001) as total cholesterol greater than or equal to 200 mg/dl or triglycerides greater than or equal to 150 mg/dl or HDL less than or equal to 50 mg/dl (for women) or less than or equal to 40 mg/dl (for men), or on drug therapy for lipid management. Impaired fasting glucose (IFG) was defined as glucose greater than or equal to 100mg/dl (American Diabetes Association, 2009). Individuals using a glucose-lowering agent met the criteria for diabetes mellitus (DM). Metabolic syndrome (MS) was defined using the clinical criteria by the Adult Treatment Panel (ATP) III (Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults, 2001). According to the ATP III definition, MS requires the presence of 3 or more of the following: (a) abdominal obesity (waist

circumference > 102 cm in men or > 88 cm in women), (b) triglyceride level greater than or equal to 150 mg/dl, (c) low HDL level (< 40 mg/dl for men or < 50 mg/dl for women), (d) elevated blood pressure (SBP $\geq$ 130 mm Hg or DBP $\geq$ 85 mm Hg), and (e) IFG ($\geq$ 100 mg/dl).

Statistical Analysis

Data analysis was conducted using the Statistical Package for the Social Sciences (SPSS version 17; SPSS Inc., Chicago, IL) software. Descriptive statistics were calculated for all variables, and distributions of continuous measures were examined for departures from normality using Q-Q plots.

Two-way contingency tables were conducted to evaluate the relationship between gender (independent variable) and cardiovascular risk factors (dependent variables; HTN, DM, MS, abdominal obesity, former IVDU, cigarette smoking category, use of HIV medications [PI, NRTI]). The same procedures were performed to assess the relationship between age groups (30-39 years, 40-49 years, 50-59 years, 60-69 years; independent variable) and cardiovascular risk factors (dependent variable; HTN, DM, MS, abdominal obesity, former IVDU, cigarette smoking category, use of HIV medications (PI, NRTI). In separate analyses, duration living with HIV (1-10 years, 11-20 years, $\geq$ 21 years) and CD4+ T cell levels (< 200 cells/mm³, 200-499 cells/mm³, $\geq$ 500 cells/mm³) were used as control variables to further clarify the relationships between gender and cardiovascular risk factors and in the relationship between age groups and cardiovascular risk factors.

Pearson correlation coefficients were also calculated to examine the relationship between the GCRP and OBNLRP percentage risk scores (dependent variable), in separate analyses and variables not associated with the GCRP and OBNLRP calculations (independent variables; triglyceride [mg/dl], LDL [mg/dl], waist circumference [cm], physical activity level [MET-minute/week], CD4+ T cell/mm³, years living with HIV, duration of smoking habit in years, mean nutritional intake: cholesterol in milligrams, sodium in milligrams, carbohydrate in grams, fiber in grams, protein in grams, sugar in grams, fat in grams, saturated fat in grams). The bivariate correlation presented above was also re-analyzed, with the variable of age added, to clarify the relationship between age and years living with HIV. Partial correlation coefficients were then computed controlling for CD4+ T cell counts and years living with HIV. Results were considered significant if p was less than or equal to .003 (.05/15) and p was less than or equal to .004 (.05/13) using the Bonferroni method to control for type I error across the bivariate and partial correlations respectively.

In separate analyses, two-way contingency tables were also conducted to evaluate the relationship between GCRP and OBNLRP risk scores groups (dependent variable; low risk [< 7%], moderate risk [7%-20%], and high risk [> 20%]), and cardiovascular risk factors (independent variables; HTN, DM, MS, abdominal obesity, former IVDU, cigarette smoking category, use of HIV medications [PI, NRTI]) For significant results with more than two levels, follow-up tests were conducted using the Bonferroni method to control for type I error across multiple pair-wise comparisons.

Predictors of GCRP and OBNLRP risk scores groups were evaluated using separate multinomial logistic regressions and a backward stepwise procedure using the likelihood ratio, with a p to enter less than 0.05 and p to remove greater than 0.1. Predictor variables (independent variables) used in the models excluded variables used in the GCRP or OBNLRP calculations. Predictor variables were triglyceride, LDL, waist circumference, physical activity level, CD4+ T cell count, use of HIV medications (PI, NRTI), former IVDU, years living with HIV, duration of smoking habit in years, number of cigarettes smoked per day, family history of CVD and risk factors, and mean nutritional intake

(cholesterol, sodium, carbohydrate, fiber, protein, sugar, fat, saturated fat). All statistical analyses were two-tailed tests and statistical significance was assessed at the 0.05 level.

Results

The sample included 123 men and women living with HIV (see Table 1). The mean age of the participants was 48 years old ($SD \pm 7.3$). More men and Blacks were enrolled in this study, as is seen in the population of PLWH in New York State (Bureau of HIV/AIDS Epidemiology, New York State Department of Health, 2009). Of the 123 participants, 88.6% reported using ART, with a mean number of CD4+T cells equaling 478 cells/mm³ ($SD \pm 289$). Twenty-nine percent of the participants were obese, with a BMI greater than or equal to 30.0 kg/m², 51.2% had a waist circumference indicative of abdominal obesity (waist circumference > 102 cm for men, > 88 cm for women; Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults, 2001), and 33% met the clinical definition for MS. Twenty-five percent had hypertension and 11% had a diagnosis of DM. Approximately 5% ($n = 7$) of the sample reported that their physical activity intensity was “vigorous” (activity that causes one to breathe harder) for at least 10 minutes per session. A large number of the participants smoked ($n = 82$, 67%), while the majority of smokers consumed 1-15 cigarettes per day ($n = 61$ of 82, 74.4%). Of the 123 participants, 107 completed the 3-day food diary. The mean daily intake for sodium was 3,526 mg ($SD \pm 1876$), fiber 16.5g ($SD \pm 10$), cholesterol 438 mg ($SD \pm 283$), and total fat 83.3 g ($SD \pm 43$) or 35% of the total caloric intake per day (See Table 1).

Effects of Gender and Age on Cardiovascular Risk Factors and Duration Living with HIV

In examining the relationship between gender and cardiovascular risk factors, only abdominal obesity was statistically significant (Pearson $\chi^2 [1, n = 123] = 22.6, p = .0001$). When duration living with HIV was included in the analysis, interactions were observed. For individuals living with HIV from 1-10 years, women had a greater proportion of abdominal obesity compared to men (women = .65, men = .35, Pearson $\chi^2 [1, n = 123] = 5.0, p = .025$). Women also had a higher proportion of abdominal obesity among those living with HIV for 11-20 years (women = .61, men = .39, Pearson $\chi^2 [1, n = 123] = 11.2, p = .001$). No differences were found in terms of abdominal obesity between genders in participants living with HIV for more than 21 years. In the analysis with age groups and cardiovascular risk factors, statistical differences were not observed, including an analysis with the control variable, duration living with HIV.

Effects of Gender and Age on Cardiovascular Risk Factors and CD4+ T Cell Level

Similar to the results above, only gender and abdominal obesity were statistically significant and interactions were observed when the control variable, CD4+ T cell level was included in the analysis. Results showed that more women had a greater proportion of abdominal obesity compared to men when CD4+ T cell levels were between 200-499 cell/mm³ (women = .58, men = .41, Pearson $\chi^2 [1, n = 123] = 13.7, p = .0001$). Likewise, women with CD4+ T cell levels greater than 500 had a larger proportion of abdominal obesity compared to men in the same CD4+ T cell level (women = .62, men = .38, Pearson $\chi^2 [1, n = 123] = 6.1, p = .014$). No differences between gender and abdominal obesity were observed for participants with CD4+ T cell levels less than 200 cell/mm³. Significant differences were not detected between age groups and cardiovascular risk factors, including analysis with the control variable CD4+ T cell level.

GCRP and OBLNRP Risk Scores and Cardiovascular Risk Factors

For the correlation coefficients, only duration of smoking habit for both the GCRP and OBLNRP risk score percentages were statistically different from zero, for the bivariate

(GCRP: $r = .292, p = .001$, OBNLRP: $r = .330, p = .0001$) and the partial correlations, indicating a moderate positive relationship between duration of smoking habit and GCRP and OBNLRP risk score percentages. The results of the partial correlation with the inclusion of the control variables, CD4+ T cells and years living with HIV, suggest a slight interaction with the rise in magnitude of correlations observed (GCRP: $r = .297, p = .001$, OBNLRP: $r = .333, p = .0001$). In order to ascertain the relationship between the variables age and years living with HIV, and to review whether the correlation between the variables could potentially be testing age as a modifier, we ran the bivariate analysis. A correlation statistically different from zero was not observed (GCRP: $r = .081, p = .374$, OBNLRP: $r = .081, p = .374$, 95% CI [-0.097 to 0.259]). Based on our sample size, results, and conducting a two-tailed test, we had a better than 86% chance of making a Type II error. It is reasonable to interpret that our statistical test was underpowered.

In evaluating the relationship between the cardiovascular risk factors and the GCRP and OBNLRP risk score groups, several variables were statistically significant. Results were significant for gender, age groups, MS, DM, and HTN for both measures (See Table 2 and Figure 1). Based on the analyses, men and participants who were older and who had clinical histories of MS, DM, or HTN, were more likely to belong to the high-risk group for both measures. The data revealed differences between men and women in the high-risk group (GCRP: male = .34, female = .04, Pearson $\chi^2 [2, n = 123] = 22.5, p = .0001$; OBNLRP: men = .37, female = .04, Pearson $\chi^2 [2, n = 123] = 25.0, p = .0001$). In conducting the pair-wise comparisons for age groups, participants in the 60-69 year age group had a larger proportion of individuals in the high-risk group compared to younger groups. All comparisons were statistically significant, using the Bonferroni method, except for the 50-59 year age group (GCRP: Pearson $\chi^2 [2, n = 123] = 3.2, p = .200$; OBNLRP: Pearson $\chi^2 [2, n = 123] = 2.8, p = .251$).

Predictive Variables Associated with GCRP and OBNLRP Risk Scores

The impact of the independent variables were similar for both measures when the variables associated with the cardiovascular risk factors were entered into the logistic regression model to predict GCRP and OBNLRP risk scores. In using the backward stepwise procedure to predict the GCRP and OBNLRP risk scores, waist circumference (cm) and duration of smoking habit (years) met criteria for inclusion in the models. For participants who were in the low-risk group, a one centimeter reduction in waist circumference reduced the odds of being in the high-risk group by 15% ($p = .011$) and 19.5% ($p = .002$) respectively for the GCRP and OBNLRP (see Table 4), controlling for other variables in the model. A 1-year reduction in smoking duration for the low risk group decreased the odds of being in the high-risk group by 5.5% ($p = .005$) for GCRP and by 6.1% ($p = .004$) for OBNLRP, controlling for waist circumference. For the moderate-risk group, waist circumference and duration of smoking habit were not significant for either GCRP or OBNLRP risk score.

Discussion

In describing cardiovascular risk factors and assessing the GCRP and OBNLRP risk scores, almost 25% of the participants in our study had a greater than 20% 10-year risk for developing general CVD. Increased waist circumference and longer duration of smoking habit were predictors for increased GCRP and OBNLRP risk scores. A large subset of the participants had modifiable cardiovascular risk factors such as DM, HTN, unhealthy eating patterns, physical inactivity, cigarette smoking, and obesity. Our results for modifiable cardiovascular risk factors were similar to other HIV studies (Adeyemi, Rezai, Bahk, Badri, & Thomas-Gossain, 2008; Capili & Anastasi, 2008; Worm et al., 2009) and show that well-established CVD risk factors were likely to impact cardiac risk in this population (Mondy et al., 2007).

Based on recent findings from the Data Collection on Adverse Events of Anti-HIV Drugs Study (Sabin et al., 2008), the use of PIs, NRTIs, and specific agents such as lopinavir/ritonavir, fosamprenavir, and abacavir were associated with increased risk for MI. Law et al. (2006) observed similar findings in that individuals with HIV and naïve for ART had fewer MIs compared to those using ART (definition of ART for this study: combination therapy that included a PI and/or NRTI), and those taking ART for a longer duration had higher incidences of MI. We included these agents in our regression models to assess current evidence suggestive of risk factors associated with CVD in HIV.

Our results for HIV medications were similar to those found by Adeyemi and colleagues (2008), in that a statistically significant difference was not observed in association with the use of specific HIV agents (PIs, NRTI,) and higher GCRP and OBNLRP risk scores. It is important to underscore that our study may not have been adequately powered to detect differences between HIV medication use and GCRP and OBNLRP risk scores. In addition, we did not assess for historical use of other HIV regimens or duration of use, which could have influenced the results. Despite observing no association between HIV medication use and GCRP and OBNLRP risk scores in this study, it would be prudent to avoid, if possible, agents linked with the risk for MI, especially among individuals with recognized CVD risk factors.

Smoking was the most prevalent, modifiable risk factor noted among our study participants. Smoking prevalence in this study was much higher than national smoking rates. The Behavioral Risk Factor Surveillance System showed that 18.4 % of the adult population smoked (Centers for Disease Control and Prevention [CDC], 2008), and the 2008 smoking prevalence rates, found by the National Health Interview Survey (NHIS) in a study of 21,781 United States adults, were 22.0% for Whites, 21.3% for Blacks, 15.8% for Hispanics, 19.8%-27.5% for adults without a high school education, and 31.5% for adults living below the poverty level (CDC, 2009). The rate of smoking in our study resembled other HIV studies (Mondy et al., 2007; Palacios et al., 2006), which showed that cigarette smoking was more common among PLWH. It is important to note, that chronic smoking can decrease adiponectin levels and increase the risk for endothelial dysfunction (Iwashima et al., 2005; Matsuzawa, 2006) and susceptibility to becoming insulin resistant (van der Vaart, Postma, Timens, & Ten Hacken, 2004; Van Gaal, Mertens, & De Block, 2006). These are additional reasons to maintain vigilance in encouraging and supporting HIV-infected smokers to abstain from cigarettes.

Unexpectedly, we learned that the number of cigarettes smoked per day did not differ across the GCRP and OBNLRP risk score groups (low risk, moderate risk, high risk). For this study, a possible explanation for the lack of smoking difference among the GCRP and OBNLRP risk score groups was that participants had similar smoking patterns in terms of the number of cigarettes smoked per day ($M = 7$ cigarettes smoked/day for each risk group). Forty-three percent of the participants were considered “light smokers,” defined as smoking fewer than 10 cigarettes/day (Hukkinen, Kaprio, Broms, Koskenvuo, & Korhonen, 2009). The city where our study was conducted underwent a massive campaign for tobacco control in 2002. Higher taxes were imposed on cigarettes and the *Smoke Free Act* was launched in 2003 to protect the health of city workers from the effects of second-hand smoke by making all workplaces smoke free, including bars and restaurants (New York City Department of Mental Health and Hygiene, 2007). Smoking cessation programs were also administered throughout the city in various clinical settings and provided free or minimal-cost counseling, nicotine patches, and other resources to help individuals quit smoking.

Additional research is needed to better understand how to effectively help HIV-infected smokers quit smoking, due to the high prevalence of smoking in this population. Burkhalter,

Springer, Chhabra, Ostroff, and Rapkin (2005) reported that 75% of HIV-infected smokers were interested in quitting, yet smoking cessation protocols specific for HIV care were not available (Reynolds, 2009). It is important to underscore that regardless of the quantity smoked, smokers have increased CVD morbidity and mortality, even at low levels of cigarette smoking (≤ 5 cigarettes/day; Bjartveit & Tvedal, 2005). Smokers with compromised immune systems can also have greater health consequences and complications than those with intact immune systems (Burkhalter et al., 2005).

In addition to routinely assessing a patient's smoking status, waist circumference should be measured during initial clinic visits and used to monitor progress of weight loss efforts (Klein et al., 2007). Increased waist circumference is a recognized risk factor for CVD (Klein et al., 2007) and is a surrogate measure for visceral fat (Ryo et al., 2005). Our results suggesting that increased waist circumference was associated with elevated GCRP and OBNLRP risk scores was, therefore, not surprising and similar to other HIV studies and the available evidence on the risk of abdominal obesity (Adeyemi et al., 2008; Mondy et al., 2007). Abdominal obesity is linked with increased blood coagulability, endothelial dysfunction, inflammation, and insulin resistance (Ferroni, Basili, Falco, & Davi, 2004). It is an independent risk factor for diabetes, and can exacerbate existing CVD-related problems such as HTN and dyslipidemia (Van Gaal et al., 2006). In addition, it has been well established that the addition of each cardiovascular risk factor raises the risk for CVD and its complications (Wilson, Kannel, Silbershatz, & D'Agostino, 1999) because multiple risk factors for CVD are interrelated and reducing them can have a beneficial impact on the outcome.

Study Limitations

We acknowledge that our study had limitations. First, the cross-sectional design limited the ability to make causal inferences and the ability to re-evaluate blood pressure measurements, as routinely conducted in clinical practice settings, to verify diagnosis of hypertension. Second, study participants were self-selected, potentially overestimating the prevalence of CVD risk factors. Third, the Framingham cohort used to develop the GCRP and OBNLRP risk scores were not infected with HIV and, therefore, may not have provided an accurate measure of CVD risk in association with HIV infection. Lastly, several aspects of the study used self-report such as duration of smoking, family history of CVD, and associated risk factors. Therefore, under reporting or over reporting cannot be ruled out and may have led to inaccurate estimates. It is important to note that the cardiovascular risk factors we report are similar to other HIV studies, which collected such data (De Socio et al., 2008; Mondy et al., 2007; Palacios et al., 2006).

At this time, there is no CVD risk score equation specifically tailored for HIV. The usefulness of additional surrogate biomarkers to evaluate the risk for CVD such as apolipoprotein (apo) A-I, apoB, homocysteine, c-reactive protein, micro-albuminuria, or noninvasive markers for atherosclerosis such carotid intimal wall thickness or coronary calcium scores, is not clear in this population (Hsue et al., 2008). However, efforts are underway to develop methods to estimate CVD risk more precisely in this population (Friis-Moller & Worm, 2007).

Conclusion

Until better tools are available, using established CVD risk score assessments, such as measures developed from the Framingham cohort, is a viable option to alert clinicians and patients to possible CVD risks that can be modified with life-style changes. On a routine basis, simple measures such as body weight, blood pressure, waist circumference, and

smoking status can be conducted in any clinical setting and can help to reduce CVD risk in HIV.

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Figure 1.
GCRP and OBNLRP risk scores and clinical characteristics.

Table 1
Demographic and Clinical Data

Characteristics or Laboratory Markers <i>N</i> = 123	Results
Male gender, <i>n</i> (%)	74 (60.2%)
Age, years $\pm$ <i>SD</i>	48 $\pm$ 7.3
age range of sample = 34-68 years	
Ethnicity, <i>n</i> (%)	
Black	88 (71.0%)
White	3 (2.4%)
Hispanic	20 (16.1%)
Native American	2 (1.6%)
Other	10 (8.1%)
Marital Status, <i>n</i> (%)	
Single	82 (66.7%)
Married	11 (8.9%)
Domestic Partner	9 (7.3%)
Divorced	12 (9.8%)
Widowed	9 (7.3%)
Cigarette Smoker, <i>n</i> (%)	82 (66.6%)
Number Years Smoked, years $\pm$ <i>SD</i>	15.4 $\pm$ 14.3
Duration of HIV Diagnosis, years $\pm$ <i>SD</i>	15.3 $\pm$ 5.9
CD4+ T cells/mm ³ $\pm$ <i>SD</i>	478 $\pm$ 289
Cholesterol mg/dl $\pm$ <i>SD</i>	188 $\pm$ 54.2
High Density Lipoprotein mg/dl $\pm$ <i>SD</i>	41.7 $\pm$ 18.3
Low Density Lipoprotein mg/dl $\pm$ <i>SD</i>	125 $\pm$ 51
Triglyceride mg/dl $\pm$ <i>SD</i>	145 $\pm$ 109.5
Glucose mg/dl $\pm$ <i>SD</i>	84.3 $\pm$ 20
Systolic Blood Pressure mm Hg $\pm$ <i>SD</i>	125.8 $\pm$ 13.8
Diastolic Blood Pressure mm Hg $\pm$ <i>SD</i>	81.2 $\pm$ 14.1
Waist Circumference, cm $\pm$ <i>SD</i>	
Male	95.8 $\pm$ 12.2
Female	99.3 $\pm$ 13.2
Body Mass Index, kg/m ² $\pm$ <i>SD</i>	
Male	26.0 $\pm$ 4.4
Female	29.0 $\pm$ 5.9
Physical Activity, MET-minutes/wk $\pm$ <i>SD</i>	1475.9 $\pm$ 1726.7

Note. SD = Standard Deviation

Table 2
Frequency of GCRP and OBNLRP Risk Scores Based on Demographic and Clinical Characteristics

Clinical Characteristic	General Cardiovascular Risk Profile (GCRP)				Office-Based Non-Laboratory Risk Profile (OBNLRP)			
	Low Risk <i>n</i> (%)	Moderate Risk <i>n</i> (%)	High Risk <i>n</i> (%)	Between-group differences <i>p</i> - value	Low Risk <i>n</i> (%)	Moderate Risk <i>n</i> (%)	High Risk <i>n</i> (%)	Between-group differences <i>p</i> - value
Female (<i>n</i> = 49)	29 (59.2%)	18 (36.7%)	2 (4.1%)	χ^2 (2) = 22.5 <i>p</i> = .001	25 (51%)	22 (44.8%)	2 (4.2%)	χ^2 (2) = 25.0 <i>p</i> = .000
Male (<i>n</i> = 74)	17 (23.0%)	32 (43.2%)	25 (33.8%)		12 (16.2%)	35 (47.3%)	27 (36.5%)	
Age Group in Years:								
30-39	10 (8.1%)	0 (0%)	0 (0%)	χ^2 (6) = 35.5 <i>p</i> = .000	9 (7.3%)	1 (0.80%)	0 (0%)	χ^2 (6) = 33.8 <i>p</i> = .000
40-49	26 (21.1%)	34 (27.6%)	8 (6.5%)		20 (16.3%)	38 (30.8%)	10 (8.0%)	
50-59	10 (8.1%)	12 (9.8%)	14 (11.4%)		7 (5.7%)	16 (13%)	13 (10.5%)	
60-69	0 (0%)	4 (3.3%)	5 (4.1%)		1 (0.80%)	2 (1.5%)	6 (4.9%)	
Ethnicity:								
Black	35 (28.5%)	34 (27.6%)	19 (15.5%)	χ^2 (8) = 12.1 <i>p</i> = .148	27 (22%)	42 (34.2%)	19 (15.5%)	χ^2 (8) = 15.4 <i>p</i> = .052
White	2 (1.5%)	1 (0.80%)	0 (0%)		2 (1.5%)	1 (0.80%)	0 (0%)	
Hispanic	7 (5.7%)	6 (4.9%)	7 (5.7%)		5 (4%)	7 (5.7%)	8 (6.6%)	
Native American	0 (0%)	1 (0.80%)	1 (0.80%)		0 (0%)	0 (0%)	2 (1.5%)	
Other	2 (1.6%)	8 (6.6%)	0 (0%)		3 (2.5%)	7 (5.7%)	0 (0%)	
Body Mass Index Category								
Normal weight (18.5–24.9 kg/m ²)	19 (15.4%)	17 (13.8%)	10 (8.1%)	χ^2 (4) = 10.8 <i>p</i> = .029	16 (13%)	19 (15.4%)	11 (8.9%)	χ^2 (4) = 12.2 <i>p</i> = .016
Overweight (25.0-29.9 kg/m ²)	17 (13.8%)	11 (8.9%)	13 (10.6%)		16 (13%)	13 (10.6%)	12 (9.8%)	
Obese (≥ 30.0 kg/m ²)	10 (8.1%)	22 (17.9%)	4 (3.4%)		5 (4%)	25 (20.4%)	6 (4.9%)	
Abdominal Obesity								
≥ 102 (cm) men, 88 (cm) women	24 (19.5%)	27 (22%)	12 (9.8%)	χ^2 (2) = .668 <i>p</i> = .716	18 (14.3%)	32 (26%)	13 (10.3%)	χ^2 (2) = 1.13 <i>p</i> = .570
< 102 (cm) men, 88 (cm) women	22 (17.9%)	23 (18.7%)	15 (12.2%)		19 (15.4%)	25 (20.3%)	16 (13.0%)	
Cigarette Smoking Category								
0 cigarettes/day	19 (15.4%)	16 (13%)	6 (4.9%)	χ^2 (6) = 7.98 <i>p</i> = .240	19 (15.4%)	15 (12.2%)	7 (5.7%)	χ^2 (6) = 11.5 <i>p</i> = .074
1-15 cigarettes/day	20 (16.3%)	24 (19.5%)	17 (13.8%)		12 (9.7%)	31 (25.2%)	18 (14.6%)	
16-25 cigarettes/day	3 (2.4%)	8 (6.5%)	4 (3.3%)		3 (2.4%)	8 (6.5%)	4 (3.3%)	
> 25 cigarettes/day	4 (3.3%)	2 (1.6%)	0 (0%)		3 (2.4%)	3 (2.4%)	0 (0%)	
Former Intravenous Drug User								

Clinical Characteristic	General Cardiovascular Risk Profile (G-CRP)					Office-Based Non-Laboratory Risk Profile (OBNLRP)				
	Low Risk <i>n</i> (%)	Moderate Risk <i>n</i> (%)	High Risk <i>n</i> (%)	Between-group differences <i>p</i> - value		Low Risk <i>n</i> (%)	Moderate Risk <i>n</i> (%)	High Risk <i>n</i> (%)	Between-group differences <i>p</i> - value	
Yes	6 (4.8%)	13 (10.6%)	5 (4.0%)	χ^2 (6) = 2.58 <i>p</i> = .275		5 (4.0%)	14 (11.4%)	5 (4.0%)	χ^2 (6) = 1.87 <i>p</i> = .393	
No	40 (32.5%)	37 (30.1%)	22 (17.9%)			32 (26.0%)	43 (35.0%)	24 (19.5%)		

Table 3
Frequency of GCRP and OBENLRP Risk Scores based on HIV Medication Use

Clinical Characteristic	General Cardiovascular Risk Profile (GCRP)				Office-Based Non-Laboratory Risk Profile (OBENLRP)			
	Low Risk n (%)	Moderate Risk n (%)	High Risk n (%)	Between-group differences w/ p-value & *95% (CI)	Low Risk n (%)	Moderate Risk n (%)	High Risk n (%)	Between-group differences w/ p-value & *95% (CI)
Protease Inhibitor Use								
Yes	30 (24.4%)	32 (26.0%)	19 (15.4%)	$\chi^2 (2) = .330$ $p=.848$ (-0.17 to 0.28)	24 (19.5%)	38 (30.9%)	19 (15.4%)	$\chi^2 (2) = .034$ $p=.983$ (-0.23 to 0.24)
No	16 (13.0%)	18 (14.6%)	8 (6.5%)		13 (10.6%)	19 (15.4%)	10 (8.1%)	
Nucleoside Reverse Transcriptase Inhibitor Use								
Yes	29 (23.6%)	33 (26.8%)	16 (13.0%)	$\chi^2 (2) = .348$ $p=.840$ (-0.19 to 0.27)	24 (19.5%)	37 (30.0%)	17 (13.8%)	$\chi^2 (2) = .376$ $p=.829$ (-0.17 to 0.29)
No	17 (13.8%)	17 (13.8%)	11 (40.7%)		13 (10.6%)	20 (16.3%)	12 (9.8%)	
Lopinavir/ritonavir (Kaletra®)								
Yes	5 (4.1%)	10 (8.1%)	7 (5.7%)	$\chi^2 (2) = 2.88$ $p=.237$ (-0.17 to 0.47)	5 (4.1%)	11 (8.9%)	6 (4.9%)	$\chi^2 (2) = .714$ $p=.700$ (-.011 to .025)
No	41 (33.3%)	40 (32.5%)	20 (16.3%)		32 (26.0%)	46 (37.4%)	23 (18.7%)	
Fosamprenavir (Lexiva®)								
Yes	3 (2.4%)	4 (3.3%)	2 (1.6%)	$\chi^2 (2) = .078$ $p=.962$ (-0.11 to 0.13)	1 (0.80%)	7 (5.7%)	1 (0.80%)	$\chi^2 (2) = 3.87$ $p=.144$ (-.075 to .089)
No	43 (35.0%)	46 (37.4%)	25 (20.3%)		36 (29.3%)	50 (40.7%)	28 (22.8%)	
Ziagen (Abacavir®)								
Yes	6 (4.9%)	11 (8.9%)	5 (4.1%)	$\chi^2 (2) = 1.32$ $p=.517$ (-0.11 to 0.23)	7 (5.7%)	10 (8.1%)	5 (4.1%)	$\chi^2 (2) = .040$ $p=.980$ (-.016 to 0.36)
No	40 (32.5%)	39 (31.7%)	22 (17.9%)		30 (24.4%)	47 (38.2%)	24 (19.5%)	
Didosine (Videx®)+	NA	NA	NA		NA	NA	NA	

* 95% Confidence Interval (CI) based on proportional differences between Low Risk and High Risk Profile scores

+No participants reported use of didanosine

Table 4
Multinomial Logistic Regression Results: Significant Predictor Variables

	B (Logistic Regression Coefficient)	Standard Error	Wald	Degrees of Freedom	Significance	Exp(B)	95% Confidence Interval
General Cardiovascular Risk Profile^a							
Low Risk:							
Intercept	7.52	2.48	9.22	1	.002		
Waist (cm)	-.159	.063	6.41	1	.011	.853	.754 to .965
Duration Smoke	-.056	.020	7.76	1	.005	.945	.909 to .983
Goodness of Fit: Deviance: $\chi^2 = 213.4$, df (208), $p = .382$							
Office-Based Non-Laboratory Risk Profile^a							
Low Risk:							
Intercept	9.36	2.8	11.44	1	.001		
Waist (cm)	-.217	.072	9.21	1	.002	.805	.700 to .926
Duration Smoke	-.063	.022	8.3	1	.004	.939	.900 to .980
Goodness of Fit: Deviance: $\chi^2 = 205.2$, df (208), $p = .542$							

^aReference category: High Risk

Note. DF = Degrees of Freedom