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# Prevalence and Correlates of Elevated Body Mass Index among HIV-Positive and HIV-Negative Women in the Women's Interagency HIV Study

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## Abstract

Since the introduction of highly active antiretroviral therapy (HAART) and the subsequent increased life expectancy in HIV-infected persons, non-HIV–related diseases have become an important cause of morbidity and mortality. This cross-sectional study reports the prevalence of overweight and obesity, and sociodemographic, psychological, and substance use-related risk factors for elevated body mass index (BMI) among 2157 HIV-seropositive (HIV+) in comparison to 730 HIV-seronegative (HIV−) participants in the Women's Interagency HIV Study (WIHS). Separate univariable and multivariate linear regression analyses were completed for HIV+ and HIV− women. Our study revealed a similar proportion of obesity (body mass index [BMI] ≥30) among HIV+ (33%) and HIV− women (29%) ($p = 0.12$), as well as comparable median BMI (HIV+: 26.1 versus HIV−: 26.7, $p = 0.16$). HIV+ compared to HIV− women, respectively, were significantly ($p < 0.01$) older (median = 35.6 versus. 32.5), but similar ($p = 0.97$) by race/ethnicity (57% African American, 28% Hispanic, and 15% white for both). In multivariate models for both HIV+ and HIV− women, African American race/ethnicity was significantly ($p < 0.05$) associated with higher BMI, while higher quality of life score and illicit hard drug use were associated with lower BMI. Additionally, smoking, alcohol use, markers of advanced HIV infection (AIDS diagnosis, elevated HIV viral load, low CD4 count), and a history of antiretroviral therapy use (ART) were also associated with lower BMI among HIV+ women. In conclusion, risk factors for elevated BMI were similar for HIV+ and HIV− women in the WIHS. For HIV+ women, all markers of advanced HIV infection and ART use were additionally associated with lower BMI.

## Introduction

The prevalence of overweight (body mass index [BMI] ≥25) or obesity (BMI ≥30) is elevated among U.S. non-Hispanic blacks (82%) and Mexican Americans (75%) compared to non-Hispanic whites (58%).[1] A similar pattern has been shown among those with HIV infection, where African American and Latina men and women are more likely to be overweight or obese compared to those who are white.[2–5] Drug use and other cofactors have been shown to mitigate this increased risk. Previous studies showed that women with HIV who used drugs such as cocaine had a significantly lower BMI[4] and were at higher risk of developing AIDS-related wasting[6–8] compared to women who did not use drugs. Other studies showed similar findings among HIV+ individuals who reported either multiple or daily drug use[4,9] and among all smokers.[10–15] Sociodemographic cofactors including age,[1,16–18] education,[17,19] socioeconomic status,[19–21] behavioral

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factors such as alcohol use,[17,22,23] and psychosocial conditions including depression[24–27] have all been associated with obesity among the general population.

Before the availability of highly active antiretroviral therapy (HAART) in 1996, studies demonstrated a protective effect of obesity on progression to AIDS and mortality. Since the introduction of HAART and the subsequent increased life expectancy in HIV-infected persons, diseases unrelated to HIV have become an important cause of morbidity and mortality in this population.[28–30] Given the convergence of the HIV and obesity epidemics among ethnic minority women, we identified sociodemographic, psychological, and substance use-related factors associated with overweight and obesity among women with and without HIV infection in the Women's Interagency HIV Study (WIHS).

## Methods

WIHS is an ongoing, multicenter prospective cohort study established in 1994 to investigate HIV disease progression in women. A total of 3766 women (2791 HIV-seropositive [HIV+] and 975 HIV-seronegative [HIV−]) were enrolled in 1994–1995 ($n = 2623$) or 2001–2002 ($n = 1{,}143$) in six U.S. cities (New York [Bronx and Brooklyn], Chicago, Los Angeles, San Francisco and Washington, D.C.).[31] Every 6 months, participants complete a comprehensive physical examination, provide blood specimens for CD4 cell count, and HIV viral load determination, and complete an interviewer-administered questionnaire, which collects demographic, disease characteristics, and antiretroviral therapy (ART) use data. The WIHS study design and methods have been described previously.[31,32] The Institutional Review Board for each participating site approved the WIHS study protocol.

In this cross-sectional study, baseline visit BMI was calculated as body weight in kilograms/(height in meters)$^2$. Previously reported correlates of BMI in the U.S. population from the National Health and Nutrition Examination Surveys (NHANES) and other studies were evaluated, including race/ethnicity, education, marital status, and place of residence. The latter was evaluated as living in one's own home, someone else's home, or other places including halfway houses, shelters, correctional institution, or treatment facility. We also examined quality of life (QOL), which was assessed using a summary score from six domains (physical functioning, role functioning, energy/fatigue, social functioning, pain and emotional well-being) based on a published algorithm.[33] The domain scores were derived by averaging the raw scores for the corresponding items of each domain expressed on a 0–100 scale, with higher values for better functioning and well-being according to an established scoring recommendation. Depression was assessed using a score derived from the Center for Epidemiologic Study of Depression (CES-D) symptom checklist, where depressive symptoms and the likelihood of clinical depression were indicated by a score of 16 or higher[34] as validated by other studies in this population.[35] Current drug use pattern was examined as none, only marijuana use, or hard drug use, which included any injection drug use, heroin, cocaine, crack, or methadone amphetamine use. Other variables evaluated for all women included current alcohol use, which was examined as abstain-light (0–2 drinks per week) versus moderate-heavy drinking ($\geq$3 drinks per week), current and former smoking, current hormonal contraception use, current or prior cancer, and hepatitis C infection (HCV) status.

The following additional risk factors were evaluated for HIV+ women: AIDS status, CD4 count, $\log_{10}$ HIV viral load, and a history of ART (monotherapy, combination therapy, and HAART). ART classification definitions can be found at the WIHS study website (http://statepiaps.jhsph.edu/wihs/). AIDS is defined as reporting an AIDS-defining condition[36] and excluded the immunologic criterion of CD4 cell count less than 200 cells per microliter. Quantification of plasma HIV viral load was performed using the isothermal nucleic acid sequence-based amplification methods using several assays[32] and CD4 count (cells/mm$^3$) were determined by flow cytometry and performed by certified laboratories designated by AIDS Clinical Trials Group.

Women with race/ethnicity other than white, African American, or Hispanic (all; $n = 97$) or who had a current/unknown pregnancy status ($n = 88$) were excluded from the study. Additionally, only women with recorded BMI at the baseline and two consecutive visits were included in this cross-sectional study in order to compare our results to those of a planned longitudinal analysis, resulting in a final sample of 2887 (2157 HIV+, 730 HIV−).

To examine the impact of enrollment during the two study periods (1994–1995 and 2001–2002), we conducted stratified analyses by this variable and found that the direction and magnitude of regression coefficients for each enrollment period were similar ($p > 0.05$) to the pooled data estimates. Therefore, stratification by enrollment period was not indicated. However, WIHS enrollment period was included as a covariate to estimate any residual confounding associated with recruitment strategy-related differences and time. In 2001–2002, recruitment was targeted toward premenopausal AIDS-free women in order to augment the number of young women in the cohort. Although not statistically significant, women enrolling during 1994–1995 compared to 2001–2002 were slightly more likely to report having AIDS, a CD4 count less than 200, and a higher $\log_{10}$ HIV viral load (data not shown).

Univariate analyses were performed separately for HIV+ and HIV− women, with differences in characteristics between the two groups compared using the Pearson's $\chi^2$ test or the nonparametric median test. BMI was assessed as a $\log_{10}$ transformed continuous variable to improve the linearity assumptions of the model and stabilize the residual variance. Separate multivariate linear regression models were fitted for HIV− and HIV+ women. Both models adjusted for the same sociodemographic characteristics in order to compare the magnitude of each characteristic by HIV serostatus, with the additional inclusion of HIV clinical characteristics (AIDS status, CD4 count, $\log_{10}$ HIV viral load, and a history of ART use) in the model for HIV+ women. The adjusted $\beta$ estimates were multiplied by 100 to better visualize their relative magnitude in the multivariate models. All statistical analyses were performed using SAS for Windows Version 9.1 (SAS Institute, Cary, NC).

## Results

Table 1 summarizes the characteristics of the study population by HIV serostatus. Using guidelines established by the Centers for Disease Control and Prevention (CDC),[37] the

Table 1. Characteristics of Study Population by HIV Status

| | *HIV positive* (n = 2157) | | *HIV negative* (n = 730) | | |
|---|---|---|---|---|---|
| | *No.*[b] | %[b] | *No.*[b] | %[b] | p *value*[a] |
| Body mass index, kg/m$^2$ (*n* = **2887**) | | | | | |
| Underweight (<18) | 15 | 2 | 39 | 2 | |
| Normal (18–24.9) | 270 | 37 | 841 | 39 | 0.12 |
| Overweight (25.0–29.9) | 205 | 28 | 662 | 31 | |
| Obese (≥30) | 240 | 33 | 615 | 29 | |
| Median (interquartile range) | 26.1 (22.7–30.7) | | 26.7 (23.0–32.5) | | 0.16 |
| Enrollment period (*n* = **2887**) | | | | | |
| 1994–1995 | 1561 | 72 | 411 | 56 | <0.01 |
| 2001–2002 | 596 | 28 | 319 | 44 | |
| Age at baseline, years (*n* = **2887**) | | | | | |
| Median (interquartile range) | 35.6 (30.5–40.8) | | 32.5 (26.3–38.3) | | <0.01 |
| Race/ethnicity (*n* = **2887**) | | | | | |
| White | 326 | 15 | 109 | 15 | 0.97 |
| African American | 1233 | 57 | 415 | 57 | |
| Hispanic | 598 | 28 | 206 | 28 | |
| Marital status (*n* = **2840**) | | | | | |
| Married/partner | 802 | 38 | 254 | 36 | 0.24 |
| All others | 1326 | 62 | 458 | 64 | |
| Highest level of education (*n* = **2882**) | | | | | |
| <High school | 837 | 39 | 252 | 35 | 0.10 |
| Completed high school | 630 | 29 | 220 | 30 | |
| >Some college | 687 | 32 | 256 | 35 | |
| Place of residence (*n* = **2886**) | | | | | |
| Your own home | 1498 | 69 | 394 | 54 | <0.01 |
| Someone's home | 430 | 20 | 218 | 30 | |
| Outside a home (i.e., shelter, jail, street, etc.) | 228 | 11 | 118 | 16 | |
| Current smoking status (*n* = **2877**) | | | | | |
| Never smoker | 712 | 33 | 228 | 31 | 0.02 |
| Former smoker | 334 | 16 | 88 | 12 | |
| Current smoker | 1103 | 51 | 412 | 57 | |
| Current alcohol use (*n* = **2830**) | | | | | |
| Light, 0–2 drinks/week | 1642 | 78 | 512 | 71 | <0.01 |
| Moderate-heavy, ≥3 drinks/week | 471 | 22 | 205 | 29 | |
| Current drug use pattern (*n* = **2876**) | | | | | |
| No drugs | 1404 | 65 | 366 | 50 | <0.01 |
| Only marijuana | 258 | 12 | 130 | 18 | |
| Hard drugs[c] | 485 | 23 | 233 | 32 | |
| Quality of life scale (*n* = **2865**) | | | | | |
| Median (interquartile range) | 64.7 (48.8–81.1) | | 72.7 (57.2–84.7) | | <0.01 |
| Depression score (*n* = **2836**) | | | | | |
| Low (<16) | 979 | 46 | 361 | 50 | 0.06 |
| High (≥16) | 1139 | 54 | 357 | 50 | |
| Hormonal contraception (*n* = **2885**) | | | | | |
| No | 1909 | 89 | 614 | 84 | <0.01 |
| Yes | 246 | 11 | 116 | 16 | |
| Hepatitis C antibody status (*n* = **2820**) | | | | | |
| Negative | 1374 | 65 | 569 | 80 | <0.01 |
| Positive | 732 | 35 | 145 | 20 | |
| Self-reported diabetes (*n* = **2886**) | | | | | |
| No | 2064 | 96 | 695 | 95 | 0.55 |
| Yes | 92 | 4 | 35 | 5 | |
| Self-reported cancer (*n* = **2886**) | | | | | |
| No | 2036 | 94 | 704 | 96 | 0.03 |
| Yes | 120 | 6 | 26 | 4 | |
| AIDS status (*n* = **2157**)[d,e] | | | | | |
| No | 1688 | 78 | — | — | — |
| Yes | 469 | 22 | — | — | |
| Detect HIV viral load (*n* = **2157**)[d,f] | | | | | |
| No | 44 | 2 | — | — | — |
| Yes | 2113 | 98 | — | — | |

(*continued*)

Table 1. Continued

| | HIV positive (n = 2157) | | HIV negative (n = 730) | | |
|---|---|---|---|---|---|
| | No.[b] | %[b] | No.[b] | %[b] | p value[a] |
| CD4 count (***n*** = **2117**)[d] | | | | | |
| <100 | 190 | 9 | — | — | — |
| 100–199 | 248 | 12 | — | — | |
| 200–499 | 967 | 46 | — | — | |
| >500 | 712 | 34 | — | — | |
| Prior/current ART (***n*** = **2156**)[d,g] | | | | | |
| None | 853 | 40 | — | — | — |
| Mono | 522 | 24 | — | — | |
| Combo | 460 | 21 | — | — | |
| HAART | 321 | 15 | — | — | |

[a] $\chi^2$ (categorical) or nonparametric median test (continuous) *p* values.
[b] Number (no.) and percent (%) of HIV-positive and HIV-negative women.
[c] Current crack, cocaine, heroin, methadone amphetamine, or injection drug use.
[d] HIV-positive women only.
[e] Clinical AIDS definition used, which was assessed by asking a separate question for each of the 23 class C clinical conditions outlined in the 1993 case definition of AIDS and excluded the immunologic criterion of CD4 count <200 cells/$\mu$L.
[f] Several HIV RNA (viral load) quantification assays were used, with the lower detection limit of the individual test used to assess RNA detectability
[g] ART, antiretroviral therapy; Definitions of mono-, combo-, and highly active antiretroviral therapy (HAART) can be found at the Women's Interagency HIV Study website: http://statepiaps.jhsph.edu/wihs.

proportion of overweight (BMI ≥25 and ≤29.9) and obese (BMI ≥30) women were similar between HIV+ and HIV− women; as was the median BMI. There were no significant differences between HIV+ and HIV− women by race/ethnicity, marital status, education level, depression score and self-reported diabetes. Compared to HIV− women, HIV+ women were older, more likely to have enrolled in 1994–1995, report lower quality of life scores, be HCV seropositive (HCV+), and to report having a prior or current cancer. In contrast, fewer HIV+ compared to HIV− women reported living in a residence other than their own home, current smoking, current moderate-heavy alcohol consumption, current use of illicit hard drugs such as crack, cocaine, heroin or methadone amphetamines, and current use of hormonal contraception. Among HIV+ women, most had had a detectable HIV viral load, with less than one quarter having clinical AIDS or a CD4 count less than 200, and 60% had used some form of ART.

Table 2 displays the median BMI for HIV+ and HIV− women by race/ethnicity and age group. African American and Hispanic women had significantly higher median BMI than white women regardless of HIV serostatus. However, within each race/ethnicity category, the median BMI was similar for HIV+ and HIV− women. The median BMI was similar across all age groups among HIV+, but not HIV− women; HIV− women aged 35–43 years had the highest BMI.

Sociodemographic and psychosocial factors associated with BMI in multivariate analysis are reported in Table 3 for HIV− ($r^2 = 0.11$) and HIV+ ($r^2 = 0.10$) women. For both HIV+ and HIV− women, being African American was associated with higher BMI; a similar pattern was seen for Hispanic women, although only significant for HIV− women. For all women, current hard drug use and higher quality of life score were associated with lower BMI. Among HIV+ women, living in someone else's home and moderate-heavy alcohol consumption were associated with lower BMI; a

Table 2. Body Mass Index by Race, Age, and HIV Status

| | HIV positive (n = 2157) | | | HIV negative (n = 730) | | | |
|---|---|---|---|---|---|---|---|
| | n | Median (IQR) | P[b] | n | Median (IQR) | P[b] | p[a] |
| White | 326 | 24.7 (21.8, 28.0) | | 109 | 24.2 (21.4, 29.6) | | 0.94 |
| AA | 1,233 | 26.5 (22.7, 31.2) | <0.01 | 415 | 26.9 (23.0, 33.6) | <0.01 | 0.08 |
| Hispanic | 598 | 26.4 (23.4, 30.8) | | 206 | 27.6 (23.9, 31.9) | | 0.09 |
| ≤25 years | 202 | 24.8 (22.0, 30.1) | | 181 | 24.5 (21.5, 29.4) | | 0.48 |
| 26–34 years | 810 | 26.3 (23.0, 30.9) | | 250 | 27.2 (23.5, 32.2) | | 0.06 |
| 35–43 years | 833 | 26.2 (22.9, 30.6) | 0.22 | 226 | 28.1 (24.2, 35.0) | <0.01 | <0.01 |
| ≥ 44 years | 312 | 26.5 (22.4, 31.1) | | 73 | 25.8 (22.7, 34.2) | | 0.66 |

[a] Nonparametric median test *p* value reported separately for each racial/ethnic and age group by HIV status; *p* value <0.05 indicates a significant difference in median BMI by HIV status.
[b] Nonparametric median test *p* value reported separately for HIV+ and HIV− women; *p* value <0.05 indicates a significant difference in median body mass index (BMI) by race/ethnicity or age group.
IQR, Interquartile range; AA, African American.

Table 3. Multivariate Model: Risk Factors for $Log_{10}$ Body Mass Index by HIV Status

| | Parameter (95% confidence interval)[c] | |
|---|---|---|
| *Risk factors*[a,b] | *HIV positive* | *HIV negative* |
| Enrollment period: 2001–2002 vs. 1994–1995 | 0.8 (−0.6, 2.2) | 2.4 (0.8, 4.0) |
| Age at baseline (in decades)[d] | 0.7 (0.1, 1.2) | 2.0 (1.0, 3.0) |
| Race/ethnicity: African American vs. white | 2.5 (1.3, 3.7) | 3.5 (1.3, 5.8) |
| Race/ethnicity: Hispanic vs. white | 1.8 (0.5, 3.2) | 4.1 (1.7, 6.5) |
| Live in someone else's home vs. own home | −1.2 (−2.3, −0.1) | −1.0 (−2.8, 0.7) |
| Live in all other places vs. own home | −0.7 (−2.1, 0.8) | 2.2 (−0.0, 4.4) |
| Moderate-heavy vs. abstain-light alcohol use | −1.3 (−2.4, −0.2) | −0.5 (−2.3, 1.3) |
| Current smoker vs. nonsmoker | −1.1 (−2.1, −0.0) | 1.2 (−0.7, 3.1) |
| Former smoker vs. nonsmoker | 0.9 (−0.4, 2.2) | 1.3 (−1.3, 3.9) |
| Marijuana use vs. no drug use | −0.8 (−2.2, 0.5) | −1.5 (−3.7, 0.7) |
| Hard drug use[e] vs. no drug use | −4.0 (−5.2, −2.8) | −5.6 (−7.6, −3.6) |
| Quality of life score | −0.1 (−0.1, −0.0) | −0.1 (−0.1, −0.0) |
| AIDS diagnosis: yes vs. no | −1.3 (−2.4, −0.2) | NA |
| $Log_{10}$ HIV viral load | −0.9 (−1.4, −0.4) | NA |
| CD4 count: <100 vs. >500 | −2.4 (−4.2, −0.6) | NA |
| CD4 count: 100–199 vs. >500 | −1.6 (−3.1, −0.0) | NA |
| CD4 count: 200–499 vs. >500 | −1.4 (−2.4, −0.4) | NA |
| Mono therapy vs. no therapy[f] | −1.4 (−2.6, −0.2) | NA |
| Combo therapy vs. no therapy[f] | −2.3 (−3.6, −1.0) | NA |
| HAART vs. no therapy[f] | −2.6 (−4.2, −1.0) | NA |

[a]For all comparisons, referent category is listed last.
[b]Education level, marital status, depression score using the Centers for Epidemiologic Studies symptom checklist score, cancer status, and hepatitis C serostatus were evaluated, but were excluded because they did not improve the final models.
[c]Estimates and confidence intervals were multiplied by 100 to better visualize their relative magnitude.
[d]Age regression coefficient and confidence interval are reported for an increased of every 10 years (decade).
[e]Includes injection drug use, heroin, cocaine, crack, or methadone amphetamine use.
[f]Definition for mono-, combo-, and highly active antiretroviral therapy (HAART) therapy can be found at the WIHS study website: https://statepiaps.jhsph.edu/wihs/.

similar pattern was seen among HIV− women, albeit to a lesser degree. In HIV+ but not HIV− women, current smoking was associated with lower BMI. Among the HIV-related factors, all indicators of HIV disease progression (AIDS diagnosis, elevated HIV RNA, low CD4 count) and ART use were independently associated with lower BMI.

## Discussion

We found that being overweight or obese is prevalent among both HIV+ and HIV− women in WIHS. As in U.S. population studies,[1, 16] African American race/ethnicity was the strongest predictor of higher BMI in our study, independent of HIV serostatus and adjusting for other cofactors. The most robust predictor of lower BMI for all women was current hard drug use, which is supported by other investigations, independent of any correlation with smoking and alcohol.[7,38] One recent study among HIV+ individuals showed that drug users compared to non-drug users were more likely to have lower energy intake, an AIDS diagnosis, and lower adherence to ART use.[38] Other behavioral factors such as smoking and alcohol use were also associated with lower BMI among HIV+ women. Smoking is a well-established correlate of lower BMI.[10–12,15] It has been suggested that reductions in smoking prevalence may be associated with an increase in obesity in the U.S. population.[10,11,13] The association observed among HIV+, but not HIV− women, could be related to the longer duration of smoking reported among HIV+ current smokers (median = 18 years) compared to HIV− current smokers (median = 15 years). In addition, HIV+ current smokers compared to former smokers and non-smokers had higher proportions of other cofactors of lower BMI, including moderate-heavy alcohol consumption, use of hard drugs, reported AIDS status, and history of ART use.

Similar to current smoking, moderate-heavy alcohol consumption was associated with lower BMI, but only statistically significant for HIV+ women in multivariate analysis. Epidemiologic studies of alcohol consumption and BMI among women have shown inconsistent results,[17,23,39–41] partly resulting from different drinking pattern assessment methods. Our study evaluated quantity of drinks consumed rather than frequency of drinking times per week. Using both methods in the same study, opposite associations were found in a recent analysis.[22] Nonetheless, an association between increased alcohol use and lower BMI is plausible if alcohol is substituted for more caloric dietary intake.[41] Moreover, although not statistically significant, synergistic interactive effects between current alcohol, smoking and hard drug use were found in this study, which may partly explain the association between each of these factors with lower BMI.

Higher QOL score was associated with lower BMI among all women. Given the cross-sectional design of this study, it is not feasible to determine whether low quality of life is a cause or consequence of obesity. Nonetheless, the six domains measured in the QOL score might be a surrogate for previously supported risk factors for obesity including depression[42] and low socioeconomic status.[21] A closer examination showed lower median QOL scores among those with depression as well as those with lower education levels, regardless of HIV serostatus (data not shown).

The finding that living in someone else's home was associated with lower BMI among HIV+ and possibly HIV− is interesting. A possible explanation might be related to the age differences in these groups. We found that women living in someone else's home were slightly younger compared to those who lived in their own home and those who lived in other places (data not shown). Additionally, living in one's own home may be a reasonable surrogate marker for food security. In other words, food is more accessible when living in your own home. However, an association with lower BMI was not observed among those living in other places (i.e., halfway houses, hotels, shelters, jail, etc.; data not shown).

Among HIV+ women, markers of advanced HIV disease were independently associated with lower BMI as supported by previous studies.[38,43] The association between HAART and lower BMI is likely explained by selective initiation of ART use among persons with more advanced HIV disease and thus lower BMI due to cachexia. However, another possible explanation for the association of HAART with lower BMI is that after initial gains in fat due to a restoration to health, longer duration of specific antiretroviral drugs (particularly stavudine and zidovudine, which were commonly used during the timeframe of our study) has been associated with lipoatrophy.[2,44–48] Since HAART was primarily available during the second enrollment period, an examination of data from only this period showed that women on HAART had significantly ($p = 0.04$) lower median BMI (26.8) compared to those with no ART use (28.3). Among all study women, the median BMI among those on any ART (25.7) was also significantly lower ($p < 0.0001$) than those on no ART (26.9). Nonetheless, we were not able to determine the timing of ART initiation in relation to measurement of BMI.

Limitations of our study include its cross-sectional design, which prohibited a definitive determination of the temporal association between some independent variables and BMI. This study examined the role of current self-reported behaviors (i.e. smoking, alcohol consumption, illicit drug use, etc.) on BMI, which may not entirely reflect past behaviors that may have influenced the development of current BMI level. We were not able to assess the association of diet and physical activity with obesity in our cohort because these data were not collected at the baseline visit. Another limitation of our study was that we were unable to determine whether the increases or decreases in body mass were caused by alterations in fat mass or lean mass. Our study was designed to examine women at the enrollment visit only; collection of body composition data in our cohort did not begin until 5 years after enrollment of the initial cohort.

HIV+ and HIV− women in WIHS have a similar prevalence and racial/ethnic distribution of overweight and obesity to the general U.S. population, with African American and Hispanic women having significantly higher BMI than white women. Higher QOL score and current hard drug use were associated with lower BMI among all women, while current smoking, moderate-heavy alcohol use, advanced HIV infection, and ART use were associated with lower BMI only among HIV+ women. The potential interactions between receipt of HAART and obesity provide a compelling rationale for a planned analysis of the relationship between these factors over time. Finally, the consequences of lower BMI, which have been reported to include increased risk for osteoporosis, must be studied in the context of HIV infection.

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## Author Disclosure Statement

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## References

1. Ogden CL, Carroll MD, Curtin LR, McDowell MA, Tabak CJ, Flegal KM. Prevalence of overweight and obesity in the United States, 1999–2004. JAMA 2006;295:1549–1555.
2. Amorosa V, Synnestvedt M, Gross R, et al. A tale of 2 epidemics: The intersection between obesity and HIV infection in Philadelphia. J Acquir Immune Defic Syndr 2005;39:557–561.
3. Crum-Cianflone N, Tejidor R, Medina S, Barahona I, Ganesan A. Obesity among patients with HIV: The latest epidemic. AIDS Patient Care STDs 2008;22:925–930.
4. Jones CY, Hogan JW, Snyder B, et al. Overweight and human immunodeficiency virus (HIV) progression in women: Associations HIV disease progression and changes in body mass index in women in the HIV epidemiology research study cohort. Clin Infect Dis 2003;37(Suppl 2):S69–80.
5. Tedaldi EM, Brooks JT, Weidle PJ, et al. Increased body mass index does not alter response to initial highly active antiretroviral therapy in HIV-1-infected patients. J Acquir Immune Defic Syndr 2006;43:35–41.
6. Forrester JE, Tucker KL, Gorbach SL. Dietary intake and body mass index in HIV-positive and HIV-negative drug abusers of Hispanic ethnicity. Public Health Nutr 2004;7:863–870.
7. Forrester JE, Tucker KL, Gorbach SL. The effect of drug abuse on body mass index in Hispanics with and without HIV infection. Public Health Nutr 2005;8:61–68.
8. Forrester JE, Woods MN, Knox TA, Spiegelman D, Skinner SC, Gorbach SL. Body composition and dietary intake in

relation to drug abuse in a cohort of HIV-positive persons. J Acquir Immune Defic Syndr 2000;25(Suppl 1):S43–48.
9. Shor-Posner G, Campa A, Zhang G, et al. When obesity is desirable: A longitudinal study of the Miami HIV-1–infected drug abusers (MIDAS) cohort. J Acquir Immune Defic Syndr 2000;23:81–88.
10. Albanes D, Jones DY, Micozzi MS, Mattson ME. Associations between smoking and body weight in the US population: Analysis of NHANES II. Am J Public Health 1987; 77:439–444.
11. Flegal KM. The effects of changes in smoking prevalence on obesity prevalence in the United States. Am J Public Health 2007;97:1510–1514.
12. Flegal KM, Troiano RP, Pamuk ER, Kuczmarski RJ, Campbell SM. The influence of smoking cessation on the prevalence of overweight in the United States. N Engl J Med 1995;333:1165–1170.
13. John U, Meyer C, Rumpf HJ, Hapke U, Schumann A. Predictors of increased body mass index following cessation of smoking. Am J Addict 2006;15:192–197.
14. Kawada T. Difference of body mass index stratified by the period of smoking cessation from a cross-sectional study. Arch Med Res 2004;35:181–184.
15. Williamson DF, Madans J, Anda RF, Kleinman JC, Giovino GA, Byers T. Smoking cessation and severity of weight gain in a national cohort. N Engl J Med 1991;324:739–745.
16. Flegal KM, Carroll MD, Ogden CL, Johnson CL. Prevalence and trends in obesity among US adults, 1999–2000. JAMA 2002;288:1723–1727.
17. Kaplan MS, Huguet N, Newsom JT, McFarland BH, Lindsay J. Prevalence and correlates of overweight and obesity among older adults: Findings from the Canadian National Population Health Survey. J Gerontol A Biol Sci Med Sci 2003;58:1018–1030.
18. Ogden CL, Yanovski SZ, Carroll MD, Flegal KM. The epidemiology of obesity. Gastroenterology 2007;132:2087–2102.
19. Wang Y, Beydoun MA. The obesity epidemic in the United States—Gender, age, socioeconomic, racial/ethnic, and geographic characteristics: A systematic review and meta-regression analysis. Epidemiol Rev 2007;29:6–28.
20. Chang VW, Lauderdale DS. Income disparities in body mass index and obesity in the United States, 1971–2002. Arch Intern Med 2005;165:2122–2128.
21. Sobal J, Stunkard AJ. Socioeconomic status and obesity: A review of the literature. Psychol Bull 1989;105:260–275.
22. Breslow RA, Smothers BA. Drinking patterns and body mass index in never smokers: National Health Interview Survey, 1997–2001. Am J Epidemiol 2005;161:368–376.
23. Kleiner KD, Gold MS, Frost-Pineda K, Lenz-Brunsman B, Perri MG, Jacobs WS. Body mass index and alcohol use. J Addict Dis 2004;23:105–118.
24. Onyike CU, Crum RM, Lee HB, Lyketsos CG, Eaton WW. Is obesity associated with major depression? Results from the Third National Health and Nutrition Examination Survey. Am J Epidemiol 2003;158:1139–1147.
25. Pine DS, Cohen P, Brook J, Coplan JD. Psychiatric symptoms in adolescence as predictors of obesity in early adulthood: A longitudinal study. Am J Public Health 1997;87:1303–1310.
26. Stice E, Presnell K, Shaw H, Rohde P. Psychological and behavioral risk factors for obesity onset in adolescent girls: A prospective study. J Consult Clin Psychol 2005;73:195–202.
27. Vaidya V. Psychosocial aspects of obesity. Adv Psychosom Med 2006;27:73–85.
28. Lewden C, Salmon D, Morlat P, et al. Causes of death among human immunodeficiency virus (HIV)-infected adults in the era of potent antiretroviral therapy: Emerging role of hepatitis and cancers, persistent role of AIDS. Int J Epidemiol 2005;34:121–130.
29. Smit C, Geskus R, Walker S, et al. Effective therapy has altered the spectrum of cause-specific mortality following HIV seroconversion. AIDS 2006;20:741–749.
30. Cohen MH, French AL, Benning L, et al. Causes of death among women with human immunodeficiency virus infection in the era of combination antiretroviral therapy. Am J Med 2002;113:91–98.
31. Barkan SE, Melnick SL, Preston-Martin S, et al. The Women's Interagency HIV Study. WIHS Collaborative Study Group. Epidemiology 1998;9:117–125.
32. Bacon MC, von Wyl V, Alden C, et al. The Women's Interagency HIV Study: An observational cohort brings clinical sciences to the bench. Clin Diagn Lab Immunol 2005;12: 1013–1019.
33. Bozzette SA, Hays RD, Berry SH, Kanouse DE, Wu AW. Derivation and properties of a brief health status assessment instrument for use in HIV disease. J Acquir Immune Defic Syndr Hum Retrovirol 1995;8:253–265.
34. Radloff LS, Rae DS. Susceptibility and precipitating factors in depression: Sex differences and similarities. J Abnorm Psychol 1979;88:174–181.
35. Richardson J, Barkan S, Cohen M, et al. Experience and covariates of depressive symptoms among a cohort of HIV infected women. Soc Work Health Care 2001;32:93–111.
36. From the Centers for Disease Control and Prevention. 1993 revised classification system for HIV infection and expanded surveillance case definition for AIDS among adolescents and adults. JAMA 1993;269:729–730.
37. Centers of Disease Control and Prevention. Defining overweight and obesity. www.cdc.gov/obesity/defining.html (Last accessed October 8, 2009).
38. Quach LA, Wanke CA, Schmid CH, et al. Drug use and other risk factors related to lower body mass index among HIV-infected individuals. Drug Alcohol Depend 2008;95:30–36.
39. Gruchow HW, Sobocinski KA, Barboriak JJ, Scheller JG. Alcohol consumption, nutrient intake and relative body weight among US adults. Am J Clin Nutr 1985;42:289–295.
40. Liu S, Serdula MK, Williamson DF, Mokdad AH, Byers T. A prospective study of alcohol intake and change in body weight among US adults. Am J Epidemiol 1994;140:912–920.
41. Cordain L, Bryan ED, Melby CL, Smith MJ. Influence of moderate daily wine consumption on body weight regulation and metabolism in healthy free-living males. J Am Coll Nutr 1997;16:134–139.
42. Blaine B. Does Depression Cause Obesity? A meta-analysis of longitudinal studies of depression and weight control. J Health Psychol 2008;13:1190–1197.
43. Justman JE, Hoover DR, Shi Q, et al. Longitudinal anthropometric patterns among HIV-infected and HIV-uninfected women. J Acquir Immune Defic Syndr 2008;47:312–319.
44. Carr A, Samaras K, Burton S, et al. A syndrome of peripheral lipodystrophy, hyperlipidaemia and insulin resistance in patients receiving HIV protease inhibitors. AIDS 1998;12: F51–58.
45. Dube MP, Parker RA, Tebas P, et al. Glucose metabolism, lipid, and body fat changes in antiretroviral-naive subjects randomized to nelfinavir or efavirenz plus dual nucleosides. AIDS 2005;19:1807–1818.

46. Palella FJ, Jr., Cole SR, Chmiel JS, et al. Anthropometrics and examiner-reported body habitus abnormalities in the multicenter AIDS cohort study. Clin Infect Dis 2004;38:903–907.
47. Tien PC, Barron Y, Justman JE, et al. Antiretroviral therapies associated with lipoatrophy in HIV-infected women. AIDS Patient Care STDs 2007;21:297–305.
48. Tien PC, Cole SR, Williams CM, et al. Incidence of lipoatrophy and lipohypertrophy in the women's interagency HIV study. J Acquir Immune Defic Syndr 2003;34:461–466.

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