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# Risk for Cardiovascular Disease in Blacks with HIV/AIDS in America: A Systematic Review and Meta-analysis

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## ABSTRACT

Cardiovascular disease (CVD) related to HIV infection is becoming a major public health concern in the United States. Epidemiologic studies show that prolonged use of Highly Active Antiretroviral Therapy, HIV/AIDS itself, and a combination of traditional vascular risk factors increase the risk for CVD among people with HIV/AIDS. However, little is known about any racial disparities in the risk for CVD in the HIV/AIDS population. We conducted a systematic review and meta-analysis of the literature on HIV/AIDS and CVD (June 1, 2010-July 31, 2014) through MEDLINE to examine whether and how HIV-positive African Americans are disproportionately affected by CVD compared to their white counterparts. The corrected pooled effect from the eligible studies was 1.26 (95% confidence interval 1.22-1.30). Blacks living with HIV/AIDS have a higher risk for CVD than non-Hispanic whites. The findings of this study provide an important basis for prevention efforts as well as recommendations for addressing the existing racial disparities in the risk for CVD among people living with HIV/AIDS.

**Keywords:** Cardiovascular disease, HIV/AIDS, Health Disparities, Race/Ethnicity

## INTRODUCTION

Societal concern about HIV/AIDS seems to be waning as public health professionals and researchers turn their focus on diseases such as heart disease and cancer that are considered to be the leading causes of death in the United States (CDC, 2016a). It is therefore not surprising that while there is a projected increase in National Institutes of Health (NIH) funding for research in other major diseases for the 2016 and 2017 fiscal years, there is no projected increase in funding for HIV/AIDS research (NIH, 2016). This shift in the public attitude can be mainly attributed to the successes of highly active antiretroviral therapy (HAART). The efficacy of HAART has resulted in prolonged survival among HIV-infected people, thus making AIDS a chronic disease instead of one of the leading causes of death that it was at the height of the AIDS epidemic in the 1980s and 1990s (CDC, 1991). However, these successes have led to new sets of HIV-associated

complications, resulting in AIDS-related life-threatening chronic illnesses such as cardiovascular disease (Deeks, 2013). Epidemiological literature has shown a strong link between HIV/AIDS and cardiovascular disease (CVD) - whether due to long-term exposure to HIV, long-term exposure to antiretroviral therapy, or to traditional CVD risk factors (Barbaro, 2013; Muhammad, 2013; Oramasionwu, et al., 2012; Guaraldi, et al., 2011; Heffernan, et al., 2013; Kaplan et al., 2008; Islam, et al., 2012; Bavinger, et al., 2013). Taken together, this epidemiological evidence suggests that people living with HIV/AIDS (PLWHA) are at increased risk for CVD compared to the uninfected population.

CVD is the leading cause of death among blacks in the United States (CDC, 2016b). Furthermore, even though African Americans represent only around 13% of the U.S. population, they account for about 44% of new HIV infections and a disproportionate share of PLWHA and AIDS-related deaths (CDC, 2015). While several studies have examined disparities in HIV/AIDS and CVD independently among African Americans, little is known about any racial differences in the risk for CVD among PLWHA. The few studies that have examined this question have produced contradictory results (Gebo et al., 2005; Triant et al., 2007; Gardner et al., 2003; Richter et al., 2005; Silverberg, et al., 2009; Oramasionwu et al., 2012). For instance, Gebo et al. (2005) investigated racial difference in CVD-related hospitalizations among people with HIV/AIDS in 12 states and concluded that blacks were at more increased risk for cerebrovascular-related hospitalizations compared to whites. However, the authors found that blacks were at a decreased risk for ischemic heart disease-related hospitalizations than their white counterparts. Triant et al. (2007) investigated the risk for myocardial infarction (AMI) among people with HIV/AIDS who had sought hospitalization. This study concluded that blacks had a higher rate of AMI compared to whites. Like the first two studies, Gardner et al. (2003) also focused on hospitalizations and assessed the risk for CVD-related hospitalization among people with HIV/AIDS. The authors concluded that there were no CVD-related black-white disparities among HIV/AIDS patients. The Richter et al. (2005) study examined HIV patients receiving outpatient care while the Silverberg et al. (2009) study evaluated racial differences in antiretroviral therapy adherence and CVD-related mortality. Both studies concluded that there were no differences between blacks and whites. The high prevalence of HIV/AIDS and CVD among blacks and the strong association between these two epidemics raises the question of whether the confluence of these two diseases creates a double-jeopardy for blacks and increases any existing racial health disparities. Through a systematic review and meta-analysis of the literature on HIV/AIDS and CVD, this study examines whether and how blacks who have HIV/AIDS experience increased risk for CVD relative to their white counterparts. Considering the well-established racial disparity in CVD morbidity and mortality, a failure to examine racial inequalities in the risk for CVD among PLWHA undermines efforts to truly understand, address, or stem the tide of HIV/AIDS and CVD.

The present study makes a significant contribution to the literature on the intersection between HIV/AIDS, CVD, and race in the United States by expanding an earlier review done by Oramasionwu et al. (2012). Based on our comprehensive and systematic review of the literature on this topic, the study by Oramasionwu et al. (2012) is the only existing systematic literature review on racial disparities in relation to the risk for CVD among people living with HIV/AIDS. In the review, Oramasionwu and her team searched the electronic MEDLINE database for studies done between January 1, 1950 and May 31, 2010 to identify relevant articles. Of the original 43 studies they identified, the researchers ended up with 5, after excluding 38 studies that did not meet their inclusion criteria. Two of the studies the authors included in the final

systematic review concluded that blacks with HIV/AIDS had a higher risk for CVD than whites, while the remaining three studies did not find any significant racial difference in the risk for CVD. Acknowledging that their finding was inconclusive, Oramasionwu et al. (2012) point out the limitations in the time periods of the studies they reviewed, emphasize the need for "further research in this area" and underscore "the need for up-to-date data" on this topic. In addition, the authors call on future investigations to "differentiate between the different forms of CVD." This is particularly critical in light of the possibility that blacks with HIV/AIDS are at increased risk for some forms of CVDs while, at the same time, at a decreased risk for others. Our study takes up these challenges and seeks to fill the research gap identified by Oramasionwu at al. (2012). In addition, our study also overcomes two other limitations in the earlier review.

First, some of the studies that Oramasionwu et al. (2012) included in their review examined racial differences in the risk for *hospitalization* for CVD, instead of the risk for incident CVD. In so doing, the review by Oramasionwu and her team included studies with different outcome variables – some having the risk for hospitalization as the outcome variable while others having the risk for contracting CVD as the outcome variable. Our review focuses on studies that examined the *risk for CVD* as the outcome variable. Second, by Oramasionwu's own admission, their study was inconclusive. This suggests that the question of whether there are black-white disparities in the risk for CVD among people with HIV/AIDS has not been answered definitively. This is what our study attempts to do.

## METHODS

### Search Strategy

We conducted a systematic search of the electronic MEDLINE database for studies published between June 1, 2010 and July 31, 2014. The search was limited to articles published in the English language that evaluated CVD among patients with HIV/AIDS. The primary outcome for our analysis was the incidence of CVD. As with the earlier review by Oramasionwu et al. (2012), the current study adapted the definition of CVD from the American Heart Association (AHA). Based on this definition, our search terms for CVD included "angina", "atherosclerosis", "atrial fibrillation", "carotid artery", "cerebrovascular disorders", "coronary artery disease", "coronary heart disease", "heart failure", "hypertension", "ischemic heart disease", "myocardial infarction", "peripheral artery disease", and "stroke". These search terms also correspond to what existing literature identifies as additional biomarkers for CVD. In addition to the CVD-related search terms, our search strategy also included terms associated with race/ethnicity such as "race", "ethnicity", "black", and "African-American", and search terms associated with HIV/AIDS such as "Human Immunodeficiency Virus", "Acquired Immune Deficiency Syndrome", "HIV", and "AIDS."

### Selection of studies

Two researchers in our team independently screened the abstracts of all articles that were identified as relevant from the search strategy described above. Any disagreement between the two reviewers was resolved by consulting with the third reviewer. Studies were included if at least two reviewers agreed on their relevance, based on our pre-determined inclusion criteria. In addition, we hand-screened references of selected articles for additional relevant studies, using the same method described above. Similar to the inclusion criteria used by Oramasionwu and her team, we included any published journal articles if the studies: i) involved patients infected with HIV/AIDS, ii) evaluated CVD, iii) described original research data, iv) included only U.S. populations, v) compared black and white patients, and vi) involved non-pediatric populations.

To facilitate the meta-analysis, we also required that the studies to be included report a summary measure such as the odds ratio. Studies that failed to incorporate all seven of the above criteria were excluded. The selection process is presented in Figure 1.

Data extraction

Data was extracted by two researchers using a standardized form. The following information was extracted from each study: author, year of study, study design, study setting, sample size, racial distribution of sample, target population, CVD definition, effect size measure, and how blacks and whites compared on the outcome measure. Table 1 presents details of the studies included in the systematic review.

Statistical Synthesis and Analysis

We used Review Manager (RevMan, version 5.3 for Windows) to generate the results that have been used in this analysis and Stata to perform meta-regression. The Odds Ratio (OR) with 95% confidence intervals (CIs) was used as the primary outcome measure. We analyzed the outcome variable with both fixed- and random-effects models to see if there was a difference. Results were similar for the two models. We present the pooled study effects only for the fixed model based on the conventional wisdom that if the two models yield similar pooled estimates then the fixed-effect model is preferred because the fixed-effect is usually more precise than the random- effects model (Ryan, 2013). We assessed heterogeneity of the effect across the studies by using the I-squared ($I^2$) statistic. The $I^2$ value provides an estimate of the amount of variance across studies resulting from heterogeneity rather than chance (Ageno et al., 2008). Forest plots were generated for each of the individual effect estimates of the studies examined.

We assessed publication bias using a funnel plot, while sensitivity analysis was done through the sequential exclusion of studies that appeared as outliers, followed by an assessment of statistical heterogeneity after each exclusion. A two-tailed p-value of 0.05 or less was considered statistically significant in assessing the overall effect of the outcome variable. We conducted secondary analysis through subgroup analysis and meta-regression. In general, this review confirms to the Preferred Reporting Items for Systematic Reviews and Meta-analysis (PRISMA) and with the Cochrane handbook on systematic reviews.

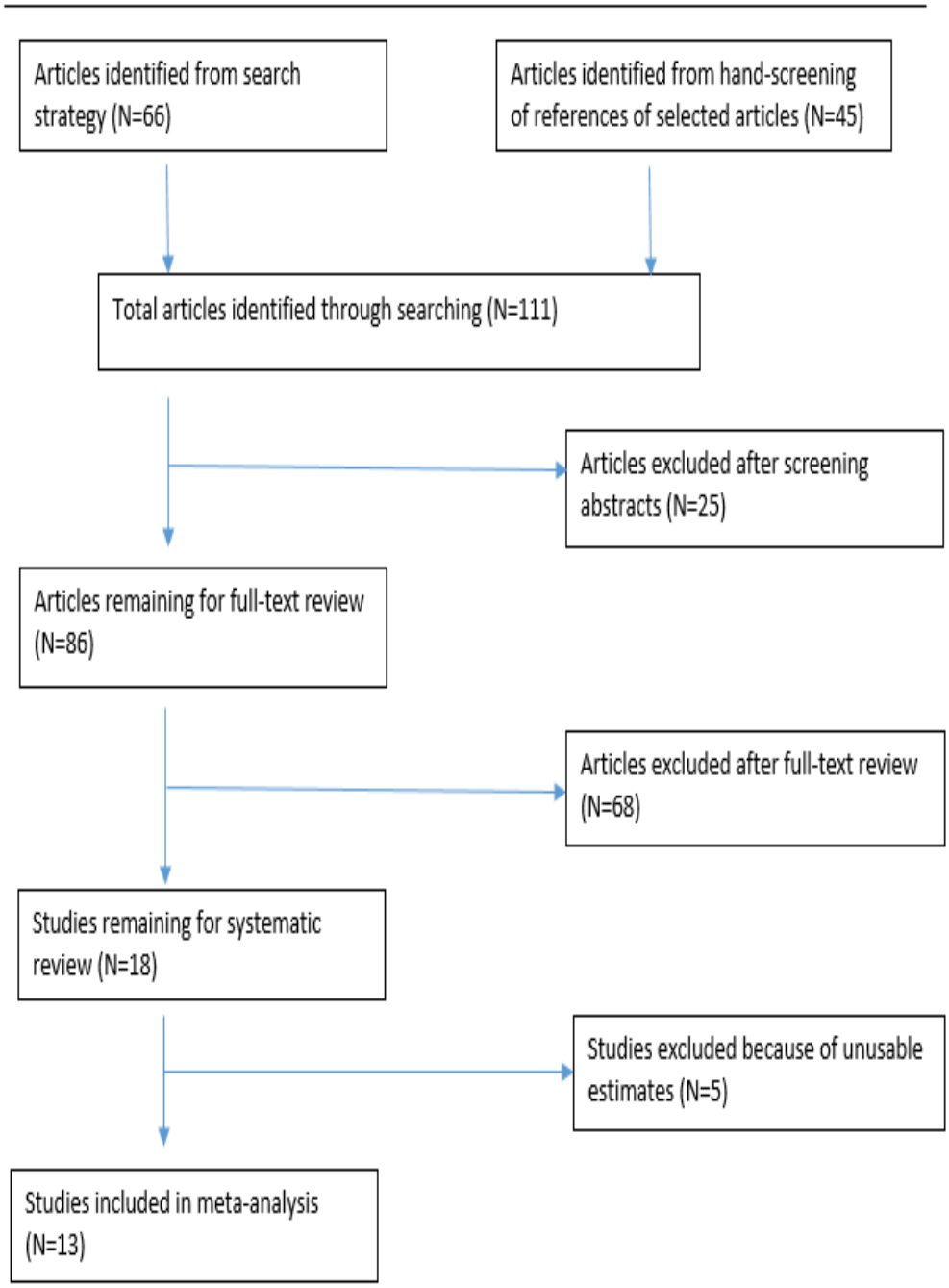


**Figure 1. Flow diagram of article selection process for the systematic review and meta-analysis**

Table 1. Characteristics of Initial Studies Included in the Systematic Review

| Study/Year | Study design | Study Setting | N | Target Population | CVD definition | Relative Risk of CVD in Blacks vs Whites |
|---|---|---|---|---|---|---|
| Armah et al., 2012 | Observational, prospective longitudinal | Veterans Aging Cohort Study (VACS) | N=2368<br>B (68.4%)<br>W (19.7%)<br>H (8.1%)<br>O (3.8%) | HIV infected and uninfected people | D-dimer | B>W |
| Borges et al., 2014 | Cross sectional | Participants in three randomized controlled trials: *SMART, †ESPRIT, and ‡SILCAAT | N=21,264 | HIV infected individuals under Active Anti-retroviral Therapy | D-dimer | B>W |
| Buchacz et al., 2012 | Prospective Longitudinal observation | HIV outpatient study data | N=3,166<br>B (31.5)<br>W (56.6)<br>H (11.9%) | HIV infected adults (age 18 and over) | Hypertension | B>W |
| Dawood, 2014 | Retrospective Cross sectional | Data from SMART trial | N= 1,444<br>B (37.6%)<br>W (46.5%)<br>O (16%) | HIV infected and SMART participants | Unspecified CVD | B>W |
| Factor, 2013 | Prospective Longitudinal | Participants in the Natural History of Menopause in HIV-infected Drug Users of the Cohort of HIV at risk Aging Men's Prospective Study | N=659<br>B (49%)<br>W (13%)<br>H (32%)<br>O (6%) | Peri-menopausal women and older man with or at risk of HIV infection | Hypertension | B>W |
| Friedberg et al., 2013 | Prospective longitudinal | The veterans Aging Cohort Study Virtual Cohort | N= 82459<br>B (48%)<br>W (37.8%)<br>H (14.2%) | HIV infected and uninfected persons | Acute Myocardial Infarction | B<W |

* Strategies for Management of Antiretroviral Therapy
† Evaluation of Subcutaneous Proleukin in a Randomized International Trial
‡ Subcutaneous Recombinant, Human Interleukin-2 in HIV-Infected Patients with Low CD4+ Counts under Active Antiretroviral Therapy

| | | | | | | |
|---|---|---|---|---|---|---|
| Hsu et al., 2013 | Prospective longitudinal | Veterans Affairs HIV Clinical case registry from 1996-2011 | N=30,533 B (54.1%) W (44.6%) O (1.3%) | HIV infected persons | Atrial Fibrillation | B<W |
| Krauskopf et al., 2013 | Prospective longitudinal | Longitudinal study of the Ocular Complications of AIDS | N=2,390 B (37%) W (45%) O (18%) | HIV infection and age from 13-60 | Hypertension | B>W |
| Lichtenstein et al., 2010 | Longitudinal | Ten HIV clinics that participated in the HIV Outpatient Study (HOPS) after 2002 | N=2005 W (52%) B (33%) O (12.3%) | HIV, patients, and CVD | Unspecified CVD | B=W |
| Medina-Torne et al., 2012 | Cross sectional | Medical care at the Naval Medical Center San Diego (NMCSD) or the National Naval Medical Center (NNMC) in Bethesda, MD. | N= 707 B (37.5%) W (48.9%) O (13.6%) | HIV & HAART | Hypertension | B=W |
| Monroe, 2012 | Cross sectional | Multicenter AIDS Cohort Study (MACS) | N=856 | HIV/AIDS infected men | Unspecified CVD | B=W |
| Parrinello et al., 2012 | Longitudinal | Women's Interagency HIV Study (WIHS) | N= 691 B (64%) W/O (9%) H (27%) | HIV infected women | Carotid Artery | B=W |
| Tripathi et al., 2014 | Prospective Longitudinal | South Carolina Medicaid system and HIV/AIDS reporting system | N= 13,632 B (70.7) W (20.9) O (8.4%) | HIV infected and diabetes | Diabetes Mellitus | B>W |

**Abbreviations: W=Whites, B=Blacks, H=Hispanics, A=Asians, O=Others**

## RESULTS

Our combined search strategy yielded 111 studies (Figure 1). Of these, 25 studies were excluded after scanning the abstracts for relevance. Another 73 were excluded after a full-text review because they did not meet our inclusion criteria. The remaining 13 studies included information that was relevant for the systematic review. We found it necessary to disaggregate

data from three studies for purposes of the meta-analysis. The first two studies were those by Buchacz et al. (2012) and Factor et al. (2013). These studies included male and female respondents and reported the findings separately by gender. Since, in some cases, the outcome variables in these studies differed by gender, aggregating the outcome variables would have masked these gender differences. Each of these studies were therefore entered in the analysis twice – the first time using the statistics generated for females and the second time using statistics for males. The third study was by Parrinello et al. (2012). This study was based on a female sample but the sample was divided into three groups that varied by HIV treatment and viremia status. The first group was of women who were treated and aviremic. The second group was of women who were treated and viremic. The third group was of women who were not treated. Since the results for these three groups were different, we decided to consider the groups separately in the meta-analysis. In all, the disaggregation of these three studies resulted in a total of 17 articles, as shown in Table 2.

Table 2. Characteristics of Final Studies Included in the Meta-Analysis

| **Study/Year** | **Study Design** | **CVD definition** | **Gender** | **Mean Age** | **Study size** | **Study location** | **Black vs White CVD Risk** |
|---|---|---|---|---|---|---|---|
| Armah et al., 2012 | Longitudinal | D-dimer | Combined M&F | 51.8 | N=2,368 | National | B>W |
| Borges et al., 2014 | Cross sectional | D-dimer | Combined M&F | 43 | N=21,264 | National | B>W |
| Buchacz et al., 2012 (F) | Longitudinal | Hypertension | F | 45 | N=673 | Regional (8 cities) | B=W |
| Buchacz et al., 2012 (M) | Longitudinal | Hypertension | M | 47 | N=2,493 | Regional (8 cities) | B>W |
| Dawood, 2014 | Cross sectional | Unspecified CVD | Combined M&F | 44.1 | N= 1,444 | National | B>W |
| Factor et al., 2013 (F) | Longitudinal | Hypertension | F | 43.4 | N= 171 | Bronx, NY | B>W |
| Factor et al., 2013 (M) | Longitudinal | Hypertension | M | 54.4 | N= 213 | Bronx, NY | B>W |
| Friedberg et al., 2013 | Longitudinal | Acute Myocardial Infarction | Combined M&F | 48.2 | N=27,350 | National | B<W |
| Hsu et al., 2013 | Longitudinal | Atrial Fibrillation | Combined M&F | 53.6 | N=30,533 | Nationwide | B<W |
| Krauskopf et al., 2013 | Longitudinal | Hypertension | Combined M&F | 43 | N=2,390 | National | B>W |
| Lichtenstein et al., 2010 | Longitudinal | Unspecified CVD | Combined M&F | 42 | N=2,005 | Regional (6 states) | B=W |
| Medina-Torne et al., 2012 | Cross sectional | Hypertension | Combined M&F | 41 | N= 707 | San Diego, CA & | B=W |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| | | | | | | Bethesda, MD | |
| Monroe, 2012 | Cross sectional | Coronary Artery Calcification | M | 48.9 | N=856 | National | B=W |
| Parrinello et al., 2012-1 | Longitudinal | Carotid Artery | F | 41.7 | N=226 | National | B>W |
| Parrinello et al., 2012-2 | Longitudinal | Carotid Artery | F | 42.8 | N=148 | National | B=W |
| Parrinello et al., 2012-3 | Longitudinal | Carotid Artery | F | 40.3 | N=227 | National | B=W |
| Tripathi et al., 2014 | Longitudinal | Diabetes Mellitus | Combined M&F | 39 | N= 13,632 | South Carolina | B>W |

In all, 53% of the studies concluded that blacks living with HIV/AIDS had higher risks for CVD than their white counterparts. The remaining studies either reported that there was no racial difference in the black-white CVD risk (35%) or that whites had higher risks for CVD than blacks (12%). Thus, a majority of the studies examined concluded that blacks with HIV/AIDS had higher risks for CVD than their white counterparts.

Figure 2 presents two forest plots for the primary and secondary analyses, respectively. In the primary analysis we estimated the pooled effect of all 17 articles that were included in the review. We calculated the Odds Ratio (OR) for the 17 articles to be 1.21 (95% CI 1.17-1.24, $p < 0.00001$), suggesting that blacks living with HIV/AIDS had a 21% higher risk of CVD compared to their white counterparts. However, we detected substantial heterogeneity ($I^2 = 89\%$; $p < 0.00001$), suggesting that not all of the studies included in the analysis evaluated the same effect and, thus, the calculation of the summary effect measure would be invalid. A visual inspection of the primary funnel plot (not included) showed that there were five outliers (Factor et al., 2013 (M); Friedberg et al., 2013; Hsu et al., 2013; Krauskopf et al., 2013; and Parrinello et al., 2012-2) suggesting that these five studies could be inconsistent with the rest of the studies in the analysis. We decided to perform a sensitivity analysis on the five studies.

We examined the effect of excluding the studies, excluding one study at a time and assessing heterogeneity after each exclusion. The result of the sensitivity analysis (Table 3) showed that excluding the five studies reduced the overall heterogeneity ($I^2$) from 89% ($p<0.0001$) to 34% ($p=0.12$). These results are also consistent with the forest plot generated after excluding the five studies as shown in the corrected model presented in the second half of Figure 2, thus justifying the exclusion of the five outliers. The low $I^2$ suggests that there is no statistically significant heterogeneity in the corrected model, implying greater consistency across the remaining studies. The corrected OR of 1.26 (95% CI 1.22, 1.30) suggests that the risk for CVD was 26% higher among blacks with HIV/AIDS, compared to their white counterparts. The symmetrical funnel plot (Figure 3) suggests the absence of any significant heterogeneity in the corrected model.

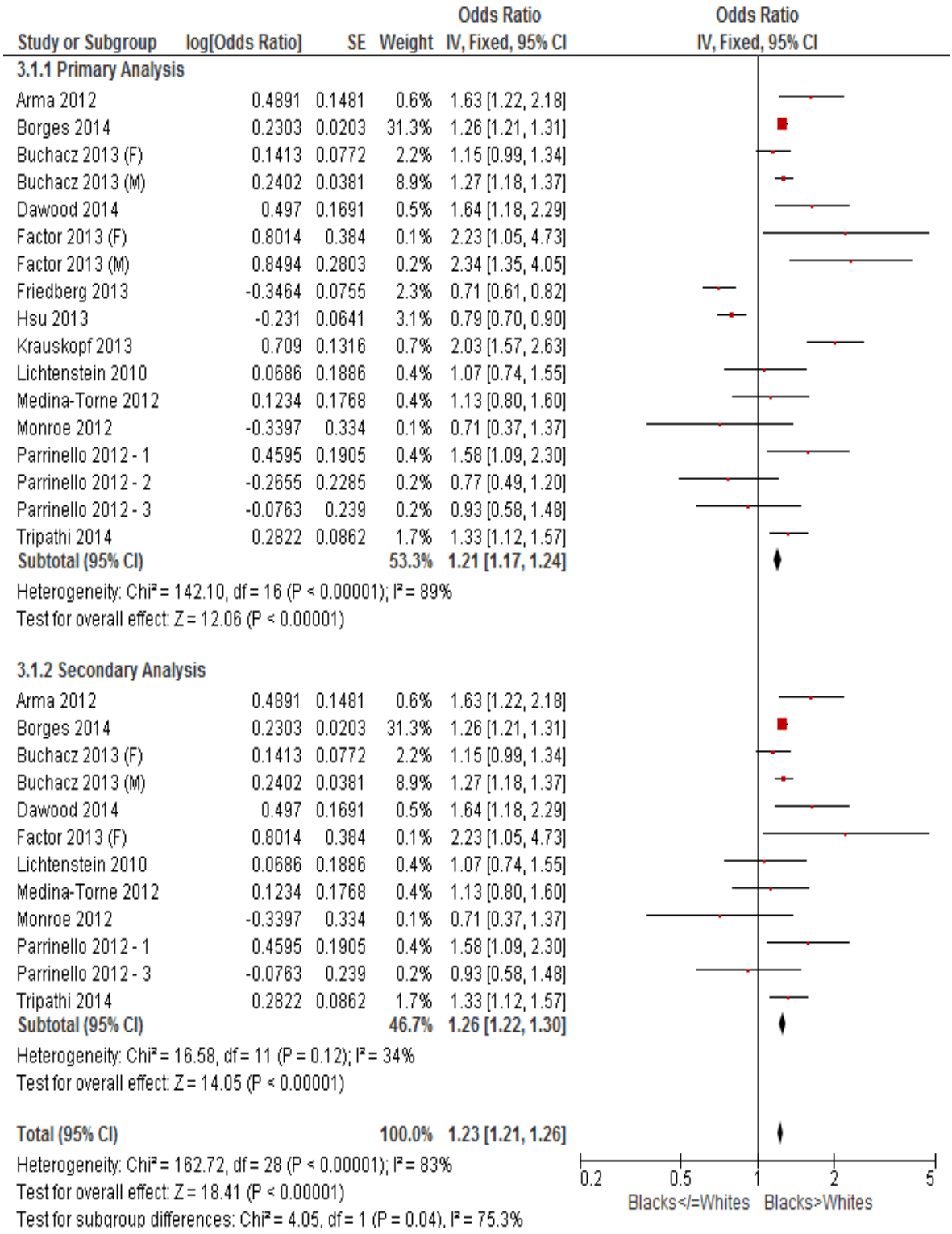


**Figure 2: Forest Plot Comparing the Risk for CVD between Blacks and Whites Living with HIV/AIDS for the Primary and Sensitivity Analyses**

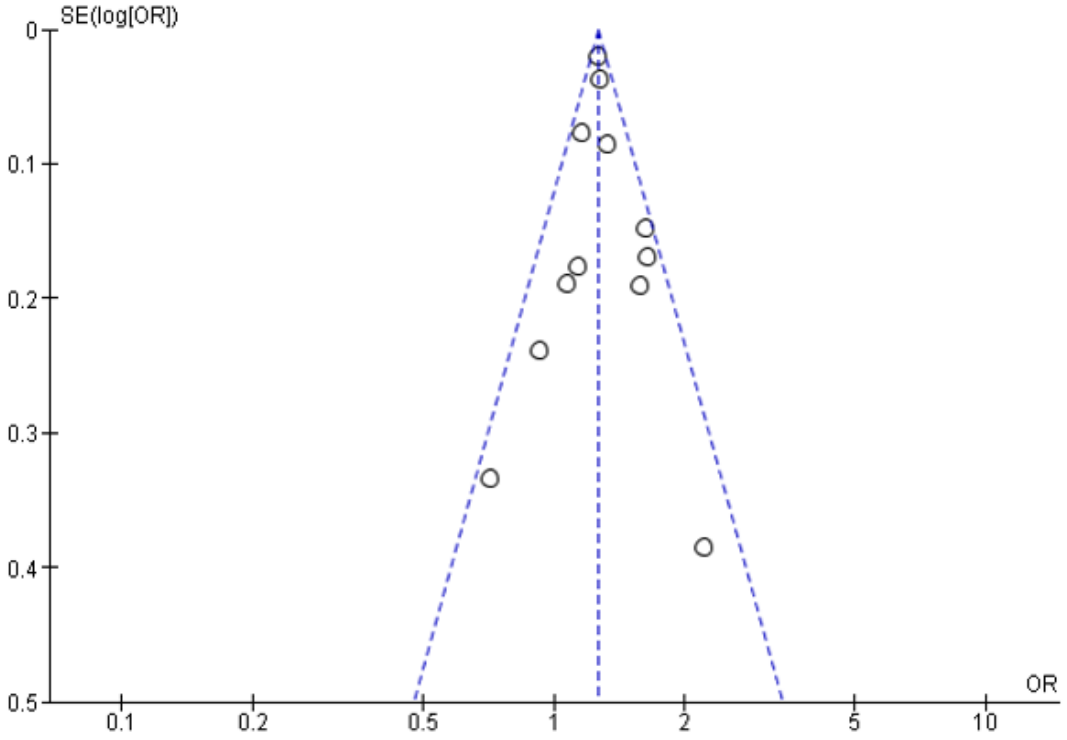


**Figure 3.** Funnel Plot of ORs and Standard Errors to assess publication Bias

**Table 3: Sensitivity Analysis for Studies included in the Secondary Analysis**

| Study | No. of Studies in the Meta-Analysis | OR (95% CI) | $I^2$ Parameter | P-Value |
|---|---|---|---|---|
| Hsu, 2013 | 16 | 1.24 (1.20-1.28) | 84 | 0.00001 |
| Friedberg, 2013 | 15 | 1.27 (1.23-1.31) | 64 | 0.0004 |
| Krauskopf, 2013 | 14 | 1.26 (1.22-1.30) | 50 | 0.02 |
| Factor, 2013 (M) | 13 | 1.26 (1.22-1.30) | 44 | 0.05 |
| Parrinello, 2012 | 12 | 1.26 (1.22-1.30) | 34 | 0.12 |

We further explored potential heterogeneity by examining effect sizes in subgroups based on differences in types of cardiovascular disease, study design, study location, gender, age, and study size. None of the six variables in the subgroup analysis presented in Table 4 shows any statistically significant reduction in the $I^2$ below the corrected $I^2$ of 34% - an indication that the

pooled effect does not vary by any of the variables examined. We also ran a meta-regression using the six variables examined in the subgroup analysis. None of the variables showed any statistically significant effect on the outcome variable, confirming the finding of the subgroup analysis that there is no significant heterogeneity in the remaining studies included in this meta-analysis.

Table 4: Subgroup Analysis for Studies included in the Secondary Analysis

| Variable | No. of Studies in the Meta-Analysis | OR (95% CI) | $I^2$ Parameter | P-Value |
|---|---|---|---|---|
| **CVD Definition** | **12** | | | |
| D-Dimer | 2 | 1.27 (1.22-1.32) | 67 | 0.08 |
| Hypertension | 4 | 1.25 (1.17-1.33) | 23 | 0.27 |
| Unspecified CVD | 2 | 1.36 (1.06-1.74) | 65 | 0.09 |
| Carotid Artery | 2 | 1.29 (0.96-1.72) | 67 | 0.08 |
| Other CVD | 2 | 1.28 (1.08-1.50) | 69 | 0.07 |
| **Study Design** | **12** | | | |
| Longitudinal Studies | 8 | 1.28 (1.16-1.41) | 35 | 0.15 |
| Cross-Sectional Studies | 4 | 1.25 (1.03-1.52) | 48 | 0.12 |
| **Study Location** | **12** | | | |
| National Studies | 6 | 1.27 (1.22-1.32) | 56 | 0.04 |
| Regional Studies | 6 | 1.25 (1.18-1.33) | 1 | 0.41 |
| **Gender** | **12** | | | |
| M&F Studies | 6 | 1.27 (1.22-1.32) | 27 | 0.23 |
| Female Studies | 4 | 1.20 (1.05-1.37) | 51 | 0.10 |
| Male Studies | 2 | 1.26 (1.17-1.36) | 66 | 0.08 |
| **Age** | **12** | | | |
| 35-45 Years | 9 | 1.26 (1.21-1.31) | 24 | 0.23 |
| 46-56 Years | 3 | 1.28 (1.19-1.38 | 65 | 0.06 |
| **Study Size** | **12** | | | |
| Under 10K | 10 | 1.26 (1.19-1.34) | 45 | 0.06 |
| Over 10K | 2 | 1.26 (1.21-1.31) | 0 | 0.56 |

## DISCUSSION AND CONCLUSION

This review provides a more comprehensive and updated assessment of the black-white differences in the risk for cardiovascular disease among people with HIV/AIDS in the United States. The last published review by Oramasionwu et al. (2012) found inconclusive evidence regarding the interaction between race, HIV/AIDS, and CVD. Our study examined this question by reviewing a larger and more recent sample of studies by not just conducting a systematic review, as Oramasionwu and her team did, but, in addition, conducting a meta-analysis. This enabled us to bring together a number of separately conducted studies, sometimes with conflicting findings, and synthesizing, pooling them quantitatively, and re-analyzing their results to assess the combined effect of race on our outcome variable. Meta-analysis has been considered as one of the highest levels of evidence (Garg et al., 2016). The robustness of meta-analysis comes from the fact that, by combining the samples of individual studies, the overall sample size is increased, thereby improving the statistical power of the analysis as well as the precision of the estimates of the outcome variable (Akobeng, 2005).

Our results suggest that blacks who are living with HIV/AIDS have a higher risk for CVD compared to whites in the same group. Specifically, our study found that blacks with HIV/AIDS had a 26% higher risk of CVD than their white counterparts. In this sense, our findings do not support the overall conclusion by Oramasionwu et al. (2012) that there is limited evidence regarding the interaction between race, HIV/AIDS, and CVD. A possible reason why our general finding is different from that of Oramasionwu et al. (2012) is their rather small sample size of only 5 studies and their failure to assess the pooled effect of race from the contradictory studies included in their review. In addition, given the periods when the studies in the Oramasionwu et al. (2012) review were conducted (1993-2005), and considering the increasing racial health disparities in the United States, it is possible that our findings reflect the changing landscape of CVD health disparities in the HIV/AIDS population.

Two significant changes have occurred in the U.S. population between the period of the studies included in the Oramasionwu et al, (2012) review and ours. In the first place, there has been a marked change in the size of racial and ethnic minority groups in the U.S. population, resulting in a more diverse population and an increase in the percentage of the minority U.S. population (Colby & Ortman, 2015). Such a population shift underscores the importance of monitoring the health status and health behavior of these groups (Kurian & Carderelli, 2007). Secondly, racial disparities in cardiovascular disease have continued to increase in the United States since the Oramasionwu et al. (2012) review (American Heart Association, 2016) as have the prevalence of many of the risk factors associated with cardiovascular disease in the black population. The studies we have reviewed capture these more recent trends and, therefore, provide a more up-to-date assessment of the phenomenon.

Racial disparities in health date back to some of the earliest health records in the United States and show that blacks have poorer health than whites across various health indicators (Williams & Sternthal, 2010). The dominant view of early research on racial health disparities was that the disparities reflected innate biological differences between the racial groups (Krieger, 1987). However, in spite of attempts to address the biological forces that were seen as the causes of such disparities, racial inequalities in health have continued to persist (Williams, 1997; Lahelma & Rahkonen, 1997; Mitchell, 2015) or to widen (Orsi et al., 2010). The fact that health disparities still persist raises important questions about the origins of these disparities in the structural, cultural, and personal conditions of life (Schnittker & McLeod, 2005). Sociological contribution to the understanding of health disparities date back to the writings of scholars like W.E.B. Du Bois (Du Bois, 1967). A central argument in Du Bois' explanation was that social factors, rather than inherent biological traits, caused racial health disparities. Other studies have echoed Du Bois by associating these disparities with increased poverty or socioeconomic inequality, disproportionate incarceration, fewer educational opportunities, adverse social environments, and residential segregation (LaVeist, 1989; Link & Phalen, 1995; Williams & Collins, 1995; Hayward et al., 2000; Lanier & Sutton, 2013).

As Schnittcker & McLeod (2005) point out, the causes of health disparities are not *just* social *or* psychological but *both* social *and* psychological (emphasis added). Similarly, it can be argued that, contrary to the paradigmatic schism created by differing perspectives on racial health disparities, the causes of the disparities, such as those we found in the risk for CVD in people with HIV/AIDS, are not *just* biomedical *or* social. They are *both* biomedical *and* social. The confluence of HIV/AIDS and CVD highlights a need to examine a merged perspective that is broad-based, integrative, and provides a more comprehensive understanding of reasons for the racial health inequalities. In fact, as Dankwa-Mullan et al. (2010) aptly point out, the current

landscape of health disparities science (should) include(s) an understanding of the complex associations between biological and nonbiological determinants of health. These sentiments were also echoed by Williams et al. (2010) who argued that research on racial differences in health should seek to understand how social exposures, or determinants, combine with biology to affect the social distribution of disease. "Otherwise, we run the risk of creating false antitheses of mutually exclusive explanations where they do not apply" (Lahelma & Rahkonen, 1997).

To understand the reasons why blacks living with HIV/AIDS experience higher risk for CVD relative to their white counterparts, it is important to consider two main factors. First, as has been well established in much of the health literature, blacks are disproportionately affected by CVD compared to non-Hispanic whites. While this disparity has been attributed to a host of behavioral, social, economic, environmental, and structural factors (Ma & Ma, 2015; Golden et al,. 2015; American Heart Association, 2014; Sharma et al., 2004), there is also evidence that biological factors play an important determining role in African Americans' differential vulnerability to CVD (Cooper, 2004; Gibbons, 2004; Schisler, et al., 2009). It is conceivable that when this genetic predisposing intersects with the risk factors for HIV/AIDS, the resultant effect is an increased risk for CVD among blacks, thus compounding the racial disparities observed. Population groups, like African Americans, with higher baseline risks for diseases such as CVD, are more likely to exhibit higher risks for HIV/AIDS coinfections (Kaufman, 2008). It is therefore conceivable that the disproportionate burden of HIV/AIDS among blacks increases their vulnerability to CVD.

Second, studies examining the effect of HIV therapy on CVD risks have concluded that people who are exposed to certain antiretroviral therapies, particularly protease-inhibiter (PI)-based regimens, tend to experience elevated risks for CVD due to side effects from the HIV medication and inflammatory effects of the virus (Lang, 2015). Is it possible that different antiretroviral therapies will work differently in different racial groups? Even though our study was not able to directly examine this question due to lack of adequate information from the studies we reviewed, several health studies have answered this question affirmatively. For instance, in their study examining racial differences in response to ART for HIV-infection, Ribuardo et al. (2013) found that blacks had a 40% virologic failure compared to whites. In other words, HIV therapy worked differently in blacks than it did in whites. Racial differences in response to medication have also been found in the treatment of other health conditions. For instance, in a study on hypertension in patients with coronary artery disease, Steinberg (2013) found that many of the black patients required more hypertension medication at higher doses to achieve the desired goal. On the other hand, these goals were achieved at much lower doses among Hispanic and white patients. Literature on pharmacological genetics has also uncovered significant differences among racial and ethnic groups in the metabolism, clinical effectiveness, and side-effect profiles of many drugs (Burroughs, 2002; Wood, 2001). Based on this literature, it is reasonable to conclude that, among other possible reasons, the black-white differences in the risk for CVD observed in our study is because HIV therapy has more negative CVD-related outcomes among blacks than in whites. If true, this would highlight the need to apply these findings to HIV/AIDS treatment and for HIV/AIDS practitioners to develop and follow treatment protocols that consider individual risk factor profiles.

Limitations

Our study had three main limitations. First, since our review only included studies that were identified through MEDLINE, it is possible that if our study had included other relevant studies in abstract-format only and others that may have been indexed in other databases it may

have led to a different conclusion than the one we made in our study. Limiting our review to full-text studies indexed in MEDLINE was in an attempt to replicate the previous review by Oramasionwu et al. (2012). Future reviews could overcome this limitation by including relevant studies that are unpublished, in abstract-format only, and in indexed in additional databases. Second, by limiting our review to studies that only examined U.S. populations, our findings cannot be broadly generalized. A much broader generalization can be achieved if future studies can examine global data. Finally, our study did not compare racial disparities in CVD among the HIV-infected blacks and non-Hispanic whites to the overall disparities of CVD in the general population (i.e. HIV-uninfected people). While our review has concluded that there is a black-white disparity in the risk for CVD among people with HIV/AIDS, there is a possibility that the detected disparity may be explained by existing disparities in the general population and that the disparity we detected may be different (either smaller or larger) than existing disparities. The results of our review do not provide a basis for determining this one way or another since our review was limited by studies that did not separate their study participants by HIV-status. This provides a new direction for future research in this area.

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