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## Disparities in Achieving and Sustaining Viral Suppression among a Large Cohort of HIV-Infected Persons in Care Washington, DC

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### Conflict of Interest

The authors have no conflicts to declare.

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**Abstract**

One goal of the HIV care continuum is achieving viral suppression (VS), yet disparities in suppression exist among subpopulations of HIV-infected persons. We sought to identify disparities in both the ability to achieve and sustain VS among an urban cohort of HIV-infected persons in care. Data from HIV-infected persons enrolled at the 13 DC Cohort study clinical sites between January 2011 and June 2014 were analyzed. Univariate and multivariate logistic regression were conducted to identify factors associated with achieving VS (viral load <200 copies/ml) at least once, and Kaplan–Meier (KM) curves and Cox proportional hazards models were used to identify factors associated with sustaining VS and time to virologic failure (VL ≥ 200 copies/ml after achievement of VS). Among the 4,311 participants 95.4% were either virally suppressed at study enrollment or able to achieve VS during the follow-up period. In multivariate analyses, achieving VS was significantly associated with age (aOR: 1.04; 95% CI: 1.03–1.06 per 5-year increase) and having a higher CD4 (aOR: 1.05, 95% CI 1.04–1.06 per 100 cells/mm<sup>3</sup>). Patients infected through perinatal transmission were less likely to achieve VS compared to MSM patients (aOR: 0.63, 95% CI 0.51–0.79). Once achieved, most participants (74.4%) sustained VS during follow-up. Blacks and perinatally-infected persons were less likely to have sustained VS in KM survival analysis (log rank chi-square  $p < 0.001$  for both) compared to other races and risk groups. Earlier time to failure was observed among females, Blacks, publically insured, perinatally infected, those with longer-standing HIV infection, and those with diagnoses of mental health issues or depression. Among this HIV-infected cohort, most people achieved and maintained VS; however, disparities exist with regard to patient age, race, HIV transmission risk, and co-morbid conditions. Identifying populations with disparate outcomes allows for appropriate targeting of resources to improve outcomes along the care continuum.

## Keywords

viral suppression; care continuum; HIV; disparities; cohort

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## Introduction

The ultimate goal of the HIV care continuum is the ability to achieve and sustain viral suppression (VS) as this can result in improved individual and population-level outcomes with reductions in comorbidities, mortality, and reduced risks of HIV transmission to others. (Cohen et al., 2011) Thus, achieving and sustaining VS is essential to meet the care continuum goals and successfully implement HIV prevention approaches such as treatment as prevention.

Data from the United States highlights gaps along the continuum with as few as 30% of persons being able to achieve VS. (United States Centers for Disease Control and Prevention, 2014a) Disparate outcomes have been observed along the continuum and it is well documented that blacks, women, men who have sex with men (MSM), and youth are disproportionately impacted by HIV and have lower rates of suppression. (Hall et al., 2012; Mugavero et al., 2009; Zaroni & Mayer, 2014; T. P. Giordano, Hartman, Gifford, Backus, & Morgan, 2009; T. P. Giordano et al., 2005; Olatosi, Probst, Stoskopf, Martin, & Duffus, 2009; Torian & Wiewel, 2011) Accordingly, one of the National HIV AIDS Strategy (NHAS) goals is to reduce HIV-related health disparities. (United States Office of National AIDS Policy, 2010) To reach this goal, identifying groups at higher risk of failing to achieve and maintain suppression is necessary to inform the development of targeted interventions.

In Washington DC, where the HIV prevalence is 2.5%, blacks, women, and MSM represent 75%, 27%, and 44% of persons living with HIV, respectively (District of Columbia HIV/AIDS, Hepatitis, STD, TB Administration, 2014) Upon measurement of DC's care continuum, an estimated 57% of persons have achieved VS and only 46% remain suppressed over time. (District of Columbia HIV/AIDS, Hepatitis, STD, TB & Administration, 2014) However, more detailed analysis of subpopulations has not been performed to confirm and monitor the relative success of subpopulations in achieving the NHAS goals.

In response to the high burden of HIV disease in Washington, DC in 2011, the DC Cohort study was launched to provide a platform from which to develop strategies and programs to reduce the burden of HIV/AIDS on the nation's capital. The DC Cohort is a longitudinal observational cohort study of HIV-infected persons receiving care at 13 clinical sites in DC and is able to provide timely data to monitor the quality of HIV care being provided. (Castel, 2012; Greenberg et al., 2015) Briefly, after obtaining informed consent, data are routinely collected from electronic medical records (EMR) on patients' socio-demographics, general medical and HIV transmission risk factors, and HIV/AIDS diagnosis dates; these data are supplemented with manual abstraction of historical data. Additionally, information on clinical encounters, diagnoses including hepatitis, treatments, and laboratory tests including CD4 cell counts, viral load (VL), and resistance, are collected on all participants. As an observational cohort, follow-up visits, and therefore data collection, correspond with the frequency of clinical visits. Monthly, data are imported into a centralized database via an

internet application called Discovere®, and processed into analytic files via SAS 9.4 (Cary, NC).

The objectives of this analysis were to identify disparities in the achievement of VS and sustained VS over time among DC Cohort participants so that population-based interventions to improve care could be based on specific, real-time data relevant to the HIV care environment in Washington, DC.

## Methods

### Data source

For this analysis, DC Cohort participants enrolled in the study between January 1, 2011 and June 15, 2014 were included. VL information collected through September 15, 2014 was also included. Among the 6,162 persons enrolled during this time period, the analysis was restricted to persons who were antiretroviral treatment experienced at enrollment and had at least two reported VL values at least 60 days apart over the course of the study (n= 4,311). Persons whose ARV status at enrollment was not known were excluded from the analysis. All data reported, including behavioral risk factors and clinical diagnoses, were abstracted from the EMR. Data on persons who refuse to be in the Cohort are routinely collected and, thus far, persons refusing to participate are significantly more likely to be female, white, and have public insurance.

### Definitions

VS was defined as having at least one VL test result less than 200 copies/ml; a commonly used cutoff for measuring suppression in other population-based HIV VS analyses. (Panel on Antiretroviral Guidelines for Adults and Adolescents, 2014; Benator et al., 2014; United States Centers for Disease Control and Prevention, 2014a; United States Centers for Disease Control and Prevention, 2014b; United States Department of Health and Human Services, 2015; Yehia et al., 2015) Among those who achieved VS, sustained VS was defined as having all subsequent VL values less than 200 copies/ml after achieving suppression. Virologic failure (VF) was defined as having a VL that was greater than or equal to 200 copies/ml after achieving suppression.

### Statistical Analysis

Univariate analyses, using chi-square and Wilcoxon rank sum tests, were conducted describing the characteristics of DC Cohort participants and assessing differences between those who achieved suppression and those who did not.

For each six-month time interval between January 1, 2011, and June 15, 2014, we calculated trends in the percentage of persons on ARVs and the percentage with a suppressed VL, median VL among those not suppressed, and the percentage of VLs greater than 100,000 copies/ml among those who were not suppressed. Univariate and multivariate Cox proportional hazards analyses were performed to identify factors at enrollment that were associated with achieving VS. Similarly, we assessed factors associated with time to earlier VF among patients who were suppressed at enrollment or during observation. For this

analysis, observation time began at the date of enrollment or VS, and all covariates were measured at that time. All variables were included in the multivariate models regardless of significance.

To assess the time to VF, Kaplan-Meier survival curves were generated among those participants who were suppressed at study enrollment or thereafter. Mean time in months from the time of suppression to the first date of “failure”, defined as a VL test result of 200 copies/ml or greater, or last VL test date, was calculated stratifying by race and HIV transmission mode.

Given the inherent variability in the 13 clinics, for all analyses, site type (hospital vs. community based clinic) was adjusted for as a random effect. P-values less than or equal to 0.05 were considered significant and all analyses were performed in SAS 9.4 (Cary, NC).

## Results

Among the 4,311 participants enrolled between January 1, 2011, and June 15, 2014, 4,111 (95.4 %) were either virally suppressed at study enrollment or able to achieve VS during the follow-up period (Table 1). Most participants were male (75.5%), non-Hispanic black (73.9%), MSM (39.6%), and publically insured (65.7%), and the median age at enrollment was 47.5 years (data not shown). At the time of study enrollment, among those achieving suppression, the median time since HIV diagnosis was 11.2 years; participants had a median CD4 count of 534 cells/ $\mu$ l. Compared to participants who achieved VS, a significantly higher proportion of persons not suppressed were younger, female, black, unstably housed, publically insured, perinatally infected, and had lower CD4 counts at enrollment ( $p < 0.05$  for all). There were no statistically significant differences between the two groups with respect to length of HIV diagnosis, history of alcohol or substance abuse, and other co-morbid conditions such as hepatitis B or C, mental health diagnoses, or HIV care facility.

When VS was measured over a maximum of a three-year observation period, high proportions of participants were on ART and suppressed at each time interval (Figure 1). For each six-month interval, 94.4% to 97.6% of participants were on ART with 81.2% to 83.8% of participants suppressed at each time point. Among those participants not virally suppressed, the median VL ranged between 2,262 copies/ml to 4,611 copies/ml with as many as 21.3% having VL results at or above 100,000 copies/ml. Further, among those with VL of 100,000 copies/ml or more, 86.0–99.0% were prescribed ART at the time of their high VL test, indicative of poorly controlled HIV replication, potential treatment failure, and/or poor medication adherence.

In the unadjusted Cox proportional hazards model, among participants with baseline CD4 count and known years since HIV diagnosis ( $n=4,141$ ), older age, male sex, non-Hispanic white race/ethnicity, private insurance, MSM, and higher CD4 count were all significantly associated with earlier time to achieving VS ( $p < 0.05$ ) (Table 2). However, in the adjusted multivariate model, perinatal infection was significantly associated with longer time to VS and only older age and higher CD4 count at enrollment remained significantly associated with earlier achievement of VS. After adjustment for other factors, for each five-year

increase in age, there was a 4% increase in achieving VS and for every 100-cell/ $\mu$ l increase in CD4 count, there was a 5% increase in achieving VS.

Among participants whose VL was suppressed at enrollment or who achieved suppression during observation, 4,024 had at least one subsequent VL test result after VS. Participants who sustained VS had an average of 6.47 VLs recorded after suppression with a mean testing rate of 3.33 tests per person-year (95% CI:3.28–3.37); those who did not sustain VS had an average of 3.99 VLs recorded with a mean testing rate of 3.83 tests per person-year (95% CI:3.71–3.95). Using Kaplan-Meier analysis, most participants (n=2,994; 74.4%) remained VS throughout the study period (Figure 2a); 29.0% of black participants were unable to sustain VS, with a mean time to VF of 31.5 months compared to those of other races (15.7%, with mean time to failure of 34.9 months) (log rank chi-square,  $p < 0.0001$ ). When stratified by mode of transmission, time to VF was lowest for perinatally infected participants (mean time to failure of 18.7 months with 38.8% experiencing failure), followed by those infected through other modes of transmission and heterosexuals. MSM had the longest mean time to failure of 32.9 months with only 22.2% experiencing VF (log rank chi-square,  $p < 0.001$ ) (Figure 2b).

In univariate Cox proportional hazards models among patients VS during observation with known CD4 count and years HIV-diagnosed who had at least one subsequent VL recorded (n=3,921), older age and higher CD4 count at the time of suppression were associated with longer times to VF, while female sex, non-Hispanic black and race/ethnicity, temporary or no housing, public insurance, heterosexual, injection drug use, or perinatal mode of HIV transmission, greater number of years HIV-diagnosed, alcohol use, substance abuse, and mental health disorders, were all associated with earlier time to VF (Table 3). After adjustment for other factors, age, sex, race/ethnicity, insurance, mode of HIV transmission, years HIV-diagnosed, CD4 count, and mental health/depression remained significantly associated with time to VF.

## Discussion

Among a large urban cohort of HIV-infected persons in care, the majority of patients were able to achieve and maintain VS. The rates of VS observed among DC Cohort participants were higher than those observed in other national studies (Hall et al., 2013; Muthulingam, Chin, Hsu, Scheer, & Schwarcz, 2013) as well as those measured using local surveillance data. (District of Columbia HIV/AIDS, Hepatitis, STD, TB Administration, 2014) This may be explained in part by the fact that DC Cohort participants may reflect a subset of HIV-infected persons who are more fully engaged in HIV care and have been living with HIV for a relatively long period of time. Earlier diagnosis of HIV and subsequent treatment initiation may also explain the high rates of suppression. (Silverberg et al., 2006) This may be particularly true in Washington, DC where access to HIV care and treatment is facilitated by high rates of insurance coverage, DC Department of Health initiatives such as “treatment on demand”, and an AIDS Drug Assistance Program that has never had a waiting list. (District of Columbia Department of Health, 2014, personal communication May 26, 2015) Observed high rates of VS coupled with reductions in HIV deaths and declines in incident infections

(District of Columbia HIV/AIDS, Hepatitis, STD, TB Administration, 2014) may be reflective of the impact of these city-wide public health initiatives.

However, consistent with previous studies, in our study population blacks, younger persons, and participants infected perinatally or through heterosexual contact were less likely to, or took longer to achieve VS, and were more likely to experience VF. Younger age is a well-documented risk factor for poor engagement in care and subsequently poorer outcomes including VS. Adolescents and young adults have been found to have steep drop offs along the care continuum and lower rates of VS compared to their older counterparts.(Adeyemi, Livak, McLoyd, Smith, & French, 2013; Hall et al., 2013; Whiteside et al., 2014; Yehia, Fleishman, Metlay, Moore, & Gebo, 2012; Zandoni & Mayer, 2014) Issues such as treatment fatigue, disclosure, lack of social support, and stigmatization may lead to periods of non-adherence and may help explain this finding, particularly among those perinatally infected, given the long duration of infection.(Abramowitz et al., 2009; Giannattasio et al., 2011; Reisner et al., 2009; Williams et al., 2006) Additionally, older persons may be more engaged in care due to the management of other chronic health conditions perhaps resulting in a more regular relationship with their HIV provider and may subsequently be more adherent. (Crawford, Sanderson, Breheny, Fleming & Thornton, 2014; Yehia et al., 2015)

While race/ethnicity did not remain significantly associated with achieving VS after adjusting for other factors, it was associated with higher rates of VF. Lower rates of achieved and sustained VS, as well as higher rates of VF, have also been observed among blacks in other HIV cohorts. (Adeyemi, Livak, McLoyd, Smith, & French, 2013; Anastos et al., 2000; Beer, Oster, Mattson, Skarbinski, for the Medical Monitoring Monitoring Project, 2014; Gant et al., 2014; Gifford et al., 2000; Gulick et al., 2006; Hall et al., 2013; Hartzell, Spooner, Howard, Wegner, & Wortmann, 2007; Lucas, Chaisson, & Moore, 1999; Lucas, Chaisson, & Moore, 2003; McFall et al., 2013; Pence et al., 2008; Yehia et al., 2012) In contrast, in other studies, no major differences by race were observed with respect to virologic or immunologic outcomes; however, racial minorities did have a longer time to treatment initiation than whites.(Jensen-Fangel et al., 2002) Explanations for these observed disparities have been attributed to cultural factors (Gifford et al., 2000), health literacy (Beer et al., 2014), and provider cultural competency (Saha et al., 2013). Higher rates of VF among blacks may be due to lower visit adherence, (Howe et al., 2014) poor ART adherence,(Beer et al., 2014; Giordano et al., 2010; Mugavero et al., 2009; Schackman et al., 2007) depression, (McFall et al., 2013) and lower socioeconomic status.(McFall et al., 2013) Thus, interventions addressing these factors may result in improved ART initiation, adherence, and clinical outcomes among racial and ethnic minorities.(Howe et al., 2014)

Although our results indicate that heterosexually infected persons were less likely to sustain VS compared to MSM, there is less consensus as to the effect of mode of transmission on VS. Heterosexually infected males have been less likely to achieve VS compared to infected MSM. (Hall et al., 2013) However, other studies have found no difference in VS rates by transmission category in multivariate analysis. (Hanna et al., 2013; Muthulingam, Chin, Hsu, Scheer, & Schwarcz, 2013) While many successful interventions exist focused on HIV prevention, engagement and retention in care among HIV-infected MSM (Bouris et al., 2013; Hightow-Weideman, Smith, Valera, Matthews, Lyons, 2011; Maulsby et al., 2013),



fewer interventions focus specifically on heterosexuals. Identifying individual, social, or structural factors such as increasing patient contact, medical case management, or testing technological interventions, may help to improve engagement, retention, and VS among heterosexuals and other HIV-infected persons. (Gardner et al., 2014; Higa, Marks, Crepaz, Liau, Lyles, 2012; IAPAC, 2015; Ko, Liu, Lai, Pai, Ko, 2013; Thompson et al., 2012)

While the majority of DC Cohort participants were able to achieve VS, intermittent episodes of high viremia (at least one VL greater than 100,000 copies/ml) among those unable to maintain VS were observed among 24% of participants during the study period. Persons with persistently and intermittently high VLs are at higher risk for developing resistant virus (Lucas et al., 1999) and transmitting virus. (Terzian et al., 2012) In this analysis, unsustained virologic control was associated with structural factors including insurance, and unstable housing. These data thus can inform public health decisions regarding strategies to reduce the costs associated with HIV care and reduce HIV associated morbidities. In Washington, DC successful interventions to address these structural barriers have included implementation of the Affordable Care Act, inclusive of early Medicaid expansion, as well as the availability of programs such as the Housing Opportunities for Persons Living with HIV/AIDS (HOPWA). Integration of mental health services with HIV care programs has also been shown to improve retention in care among persons with mental health and depression diagnoses (Sin & DiMatteo, 2014; Pyne et al., 2011; Safren et al., 2009) Further analysis of factors such as ART regimens, retention in care, medication adherence, and the development of drug resistance, as well as clinic-level factors such as the availability of supportive ancillary services, may also provide further insight into understanding these observed treatment failures.

There are several limitations in our analysis. The data presented here only reflect those HIV-infected persons in care and who agreed to participate in the DC Cohort study. Furthermore, the disparities in VS identified are representative of a cohort of HIV-infected individuals who seek care regularly and are most likely highly engaged in care, treatment experienced, and therefore more likely to achieve suppression. Data on persons who were lost to follow-up, died, transferred care to a non-DC Cohort clinic or did not have laboratory testing were censored at the time of these events; however, additional analyses found that these participants accounted for less than 3 percent of participants (data not shown). Although data on ART prescription was provided, the accuracy of ART start dates prior to study enrollment was limited hence we did not include time on ART as a covariate in our analysis. We did not have ART adherence data to further explain some of the outcomes observed with respect to suppression and virologic failure. Finally, this paper presents data on only the first three and a half years of follow up.

Despite these limitations, strengths of this analysis include that it provides a longitudinal representation of HIV care in a major urban area on more than one third of persons living with HIV in Washington, DC. Additionally, the DC Cohort includes data on a diverse group of persons receiving HIV care in a variety of clinical settings. Through the DC Cohort, HIV providers receive data in aggregate form that can more readily be analyzed thereby allowing clinicians to take more timely action to improve the quality of patient care. In the future, sites that are deemed as underperforming may be able to work with the District of Columbia

Department of Health to receive additional support and resources through the development and implementation of targeted individual, structural, and population-level interventions. The ability to maintain suppression over long periods of time will require careful population-based monitoring, which the DC Cohort study is uniquely designed to provide. While this current analysis includes only the first 4,311 patients for whom longitudinal data were available, this population represents 85% of the patients at participating clinics who were approached.

In conclusion, high percentages of individuals in this observational cohort of HIV-infected persons receiving care achieved and sustained VS. We identified disparities in VS and identified subcategories of persons who remain under-treated and are potentially contributing to the ongoing spread of HIV in the city. Efforts to identify persons with disparate outcomes will allow for appropriate targeting of resources to improve VS and achieve national goals aimed at reducing health disparities and maximizing outcomes along the care continuum.

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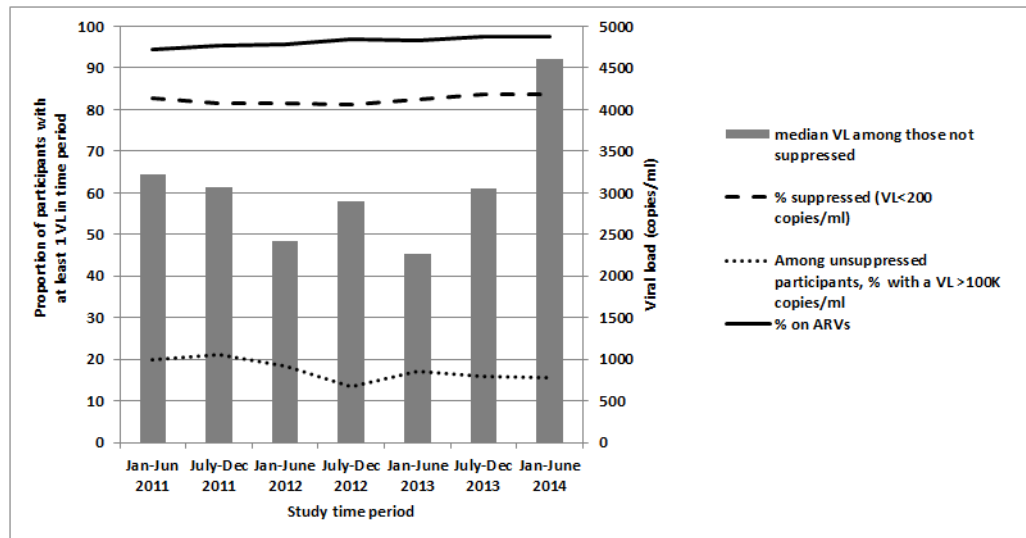
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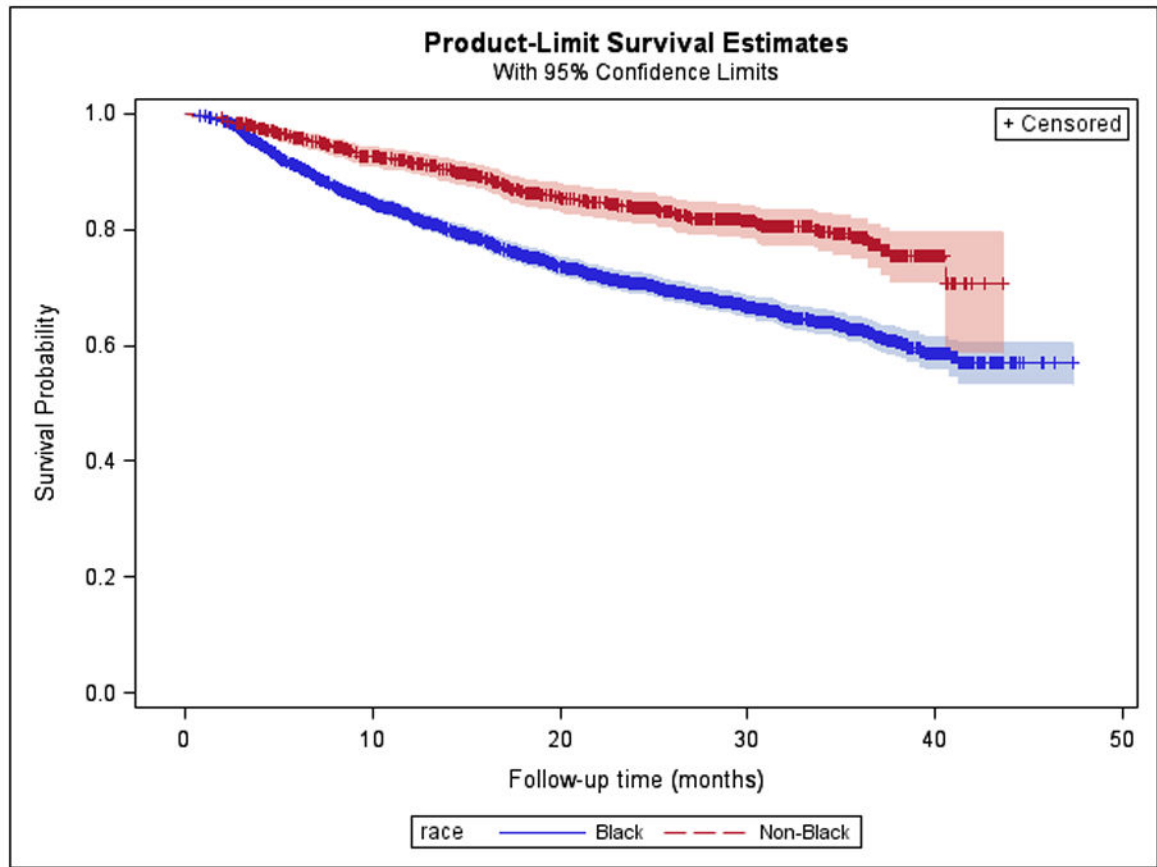
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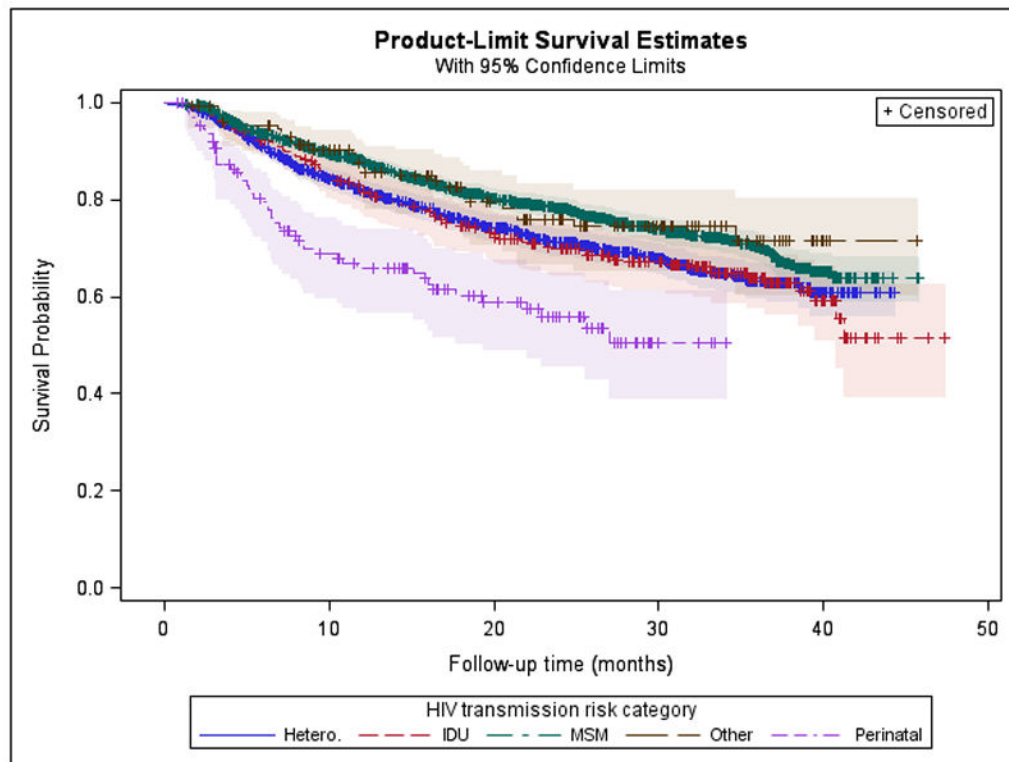


**Figure 1. Trends in Antiretroviral Use and Viral Suppression among DC Cohort participants, January 2011–June 2014**

This figure presents the proportion of participants who were on antiretroviral (ARV) treatment, had at least one VL test result, and were suppressed (VL < 200 copies/ml) at each 6-month interval between January 2011 and June 2014. Among those who were not suppressed, the median VL and percentage of participants who had a VL over 100,000 copies/ml are also shown.







**Figure 2. Kaplan-Meier Curves of Sustained Viral Suppression by Race and Mode of Transmission**

**2A.** The mean time to virologic failure (defined as a VL>200 copies/ml after achieving suppression) among black participants was 31.5 months compared to those of other races (34.9 months) (log rank chi-square,  $p<0.0001$ ). **2B.** The mean time to virologic failure for participants infected through perinatal transmission was 18.7 months, and for those infected through high-risk heterosexual contact it was 29.6 months compared to participants infected through MSM (32.9 months) (log rank chi-square,  $p<0.001$ ).

**Table 1**Characteristics of ARV-experienced DC Cohort Participants by Viral Suppression Status (N=4,311).<sup>1</sup>

Characteristics <sup>2</sup>	Participants Ever Achieving VS (n=4,111)	Participants Not Achieving VS (n=200)	P-value <sup>3</sup>
<b>Age (yrs) (median, IQR)</b>	47.9 (38.8, 55.0)	39.6 (21.8, 47.8)	<b>&lt;0.001</b>
<b>Sex at birth, n (%)</b>			
Male	3131 (76.2)	122 (61.0)	<b>&lt;0.001</b>
Female	980 (23.8)	78 (39.0)	
<b>Race/Ethnicity, n (%)</b>			
Non-Hispanic black	3,07 (73.1)	180 (90.0)	<b>&lt;0.001</b>
Non-Hispanic white	698 (17.0)	11 (5.5)	
Hispanic	179 (4.4)	4 (2.0)	
Other <sup>4</sup>	73 (1.8)	5 (2.5)	
Unknown	154 (3.7)	0 (0.0)	
<b>Housing status, n (%)</b>			
Permanent/stable	3480 (84.7)	154 (77.0)	<b>0.022</b>
Temporary/unstable	345 (8.4)	27 (13.5)	
Homeless	54 (1.3)	5 (2.5)	
Other/Unknown	232 (5.6)	14 (7.0)	
<b>Insurance status, n (%)</b>			
Public	2682 (65.2)	151 (75.5)	<b>&lt;0.001</b>
Private	1218 (29.6)	33 (16.5)	
Other	99 (2.4)	9 (4.5)	
Unknown	112 (2.7)	7 (3.5)	
<b>Mode of transmission, n (%)</b>			
MSM	1654 (40.2)	53 (26.5)	<b>&lt;0.001</b>
High risk heterosexual	1142 (27.8)	61 (30.5)	
IDU	310 (7.5)	10 (5.0)	
Perinatal	132 (3.2)	42 (21.0)	
MSM/IDU	55 (1.3)	2 (1.0)	
Other	77 (1.9)	3 (1.5)	
Unknown	741 (18.0)	29 (14.5)	
<b>Years HIV positive (median, IQR)</b>	11.2 (5.5, 17.4)	11.3 (5.8, 17.8)	0.88
<b>CD4 at enrollment (cells/<math>\mu</math>l) (median, IQR)</b>	534 (356, 737)	297 (106, 470)	<b>&lt;0.001</b>
<b>Alcohol abuse, n (%)</b>			
No	2,576 (62.7)	121 (60.5)	0.79
Yes	442 (10.8)	24 (12.0)	
Missing	1,093 (26.6)	55 (27.5)	
<b>History of substance abuse, n (%)</b>			
No	2,219 (54.0)	102 (51.0)	0.70
Yes	695 (16.9)	37 (18.5)	
Missing	1,197 (29.1)	61 (30.5)	

Characteristics <sup>2</sup>	Participants Ever Achieving VS (n=4,111)	Participants Not Achieving VS (n=200)	P-value <sup>3</sup>
<b>Hepatitis C status, n (%)</b>			
Negative	3,562 (86.6)	181 (90.5)	0.12
Positive	549 (13.4)	19 (9.5)	
<b>Hepatitis B status, n (%)</b>			
Negative	3,982 (96.9)	196 (98.0)	0.36
Positive	129 (3.1)	4 (2.0)	
<b>Mental Health/Depression, n (%)</b>			
No	2,781 (67.6)	145 (72.5)	0.15
Yes	1,330 (32.4)	55 (27)	
<b>HIV care facility</b>			
Hospital	2,245 (54.6)	115 (57.5)	0.42
Community-based organization	1,866 (45.4)	85 (42.5)	

<sup>1</sup>This includes participants who had at least two viral load measurements during the study period and were antiretroviral (ARV) treatment experienced.

<sup>2</sup>Housing, insurance status, alcohol abuse, substance abuse, hepatitis B, hepatitis C, and mental health/depression were measured at the time of study enrollment. Hepatitis B, C, and mental health/depression were based on ICD9 coding.

<sup>3</sup>Chi-square or Wilcoxon test; significant p-values <0.05 are bolded.

<sup>4</sup>Other race includes mixed race individuals, Asians, Alaska Natives, American Indians, Native Hawaiians, and Pacific Islanders.

**Table 2**Factors Associated with Achieving Viral Suppression (N=4,141).<sup>1</sup>

Characteristic	HR (95%CI)	aHR (95%CI) <sup>2</sup>
<b>Age (per 5 yrs)</b>	<b>1.05 (1.04, 1.06)</b>	<b>1.04 (1.03, 1.06)</b>
<b>Sex at birth</b>		
Male	ref	ref
Female	<b>0.88 (0.81, 0.94)</b>	0.95 (0.87, 1.03)
<b>Race/Ethnicity</b>		
Non-Hispanic black	<b>0.82 (0.75, 0.89)</b>	0.93 (0.84, 1.02)
Non-Hispanic white	ref	ref
Hispanic	0.96 (0.81, 1.13)	1.08 (0.91, 1.28)
Other	0.86 (0.67, 1.10)	0.96 (0.75, 1.24)
Unknown	1.01 (0.85, 1.21)	1.05 (0.88, 1.26)
<b>Housing status</b>		
Permanent/stable	ref	ref
Temporary/unstable	0.91 (0.81, 1.02)	0.97 (0.86, 1.09)
Homeless	0.89 (0.68, 1.16)	0.93 (0.70, 1.22)
Other/Unknown	0.99 (0.87, 1.14)	0.99 (0.85, 1.14)
<b>Insurance status</b>		
Public	<b>0.87 (0.82, 0.94)</b>	0.94 (0.87, 1.02)
Private	ref	ref
Other	0.85 (0.69, 1.04)	0.90, 0.73, 1.11)
Unknown	0.91 (0.75, 1.11)	0.91 (0.74, 1.12)
<b>Mode of transmission</b>		
MSM	ref	ref
High risk heterosexual	<b>0.91 (0.84, 0.98)</b>	0.93 (0.84, 1.02)
IDU	0.99 (0.87, 1.12)	0.98 (0.84, 1.13)
Perinatal	<b>0.53 (0.44, 0.64)</b>	<b>0.63 (0.51, 0.79)</b>
MSM/IDU	1.09 (0.83, 1.43)	1.10 (0.84, 1.45)
Other	0.98 (0.77, 1.23)	0.98 (0.78, 1.25)
Unknown	0.97 (0.89, 1.06)	0.96 (0.88, 1.06)
<b>Years HIV positive (per 5 years)</b>	1.01 (0.99, 1.03)	0.98 (0.96, 1.01)
<b>CD4 at enrollment (per 100 cells/microliter)</b>	<b>1.05 (1.04, 1.06)</b>	<b>1.05 (1.04, 1.06)</b>
<b>Alcohol abuse</b>		
No	ref	ref
Yes	0.98 (0.89, 1.09)	0.99 (0.88, 1.11)
Unknown	0.98 (0.91, 1.05)	1.00 (0.90, 1.10)
<b>History of substance abuse</b>		
No	ref	ref
Yes	0.97 (0.89, 1.06)	1.00 (0.91, 1.09)
Unknown	1.01 (0.94, 1.09)	1.02 (0.93, 1.12)
<b>Hepatitis C status</b>		

Characteristic	HR (95%CI)	aHR (95%CI) <sup>2</sup>
Negative	ref	ref
Positive	1.05 (0.96, 1.15)	0.99 (0.89, 1.10)
<b>Hepatitis B status</b>		
Negative	ref	ref
Positive	1.03 (0.86, 1.23)	1.04 (0.87, 1.25)
<b>Mental Health/Depression</b>		
No	ref	ref
Yes	1.03 (0.96, 1.10)	1.00 (0.93, 1.07)

<sup>1</sup> Excludes participants with missing baseline CD4 or years HIV-diagnosed. Clinic type (hospital vs. CBO) was adjusted for as a random effect in the model.

<sup>2</sup> Adjusted for all other variables in the model. Significant hazard ratios and 95% confidence intervals are bolded.

**Table 3**Factors Associated with Earlier Time to Virologic Failure (N=3,921).<sup>1</sup>

Characteristic	HR (95%CI)	aHR (95%CI) <sup>2</sup>
<b>Age (per 5 yrs)</b>	<b>0.93 (0.91, 0.96)</b>	<b>0.92 (0.89, 0.95)</b>
<b>Sex at birth</b>		
Male	ref	ref
Female	<b>1.52 (1.33, 1.75)</b>	<b>1.32 (1.12, 1.56)</b>
<b>Race/Ethnicity</b>		
Non-Hispanic black	<b>2.18 (1.77, 2.69)</b>	<b>1.75 (1.40, 2.19)</b>
Non-Hispanic white	ref	ref
Hispanic	1.42 (0.95, 2.12)	1.26 (0.83, 1.89)
Other	1.34 (0.74, 2.44)	1.21 (0.66, 2.21)
Unknown	<b>1.54 (1.03, 2.31)</b>	<b>1.69 (1.12, 2.55)</b>
<b>Housing status</b>		
Permanent/stable	ref	ref
Temporary/unstable	<b>1.53 (1.18, 1.99)</b>	1.19 (0.91, 1.57)
Homeless	<b>1.76 (1.10, 2.81)</b>	1.58 (0.97, 2.56)
Other/Unknown	<b>0.71 (0.62, 0.81)</b>	0.87 (0.71, 1.07)
<b>Insurance status</b>		
Public	<b>1.91 (1.59, 2.29)</b>	<b>1.31 (1.07, 1.60)</b>
Private	ref	ref
Other	0.96 (0.79, 1.16)	0.85 (0.65, 1.10)
Unknown	1.24 (0.61, 2.52)	0.84 (0.41, 1.74)
<b>Mode of transmission</b>		
MSM	ref	ref
High risk heterosexual	<b>1.31 (1.12, 1.52)</b>	1.07 (0.88, 1.29)
IDU	<b>1.36 (1.09, 1.70)</b>	1.17 (0.90, 1.53)
MSM/IDU	0.75 (0.40, 1.40)	0.64 (0.34, 1.21)
Perinatal	<b>2.55 (1.87, 3.47)</b>	<b>1.65 (1.11, 2.44)</b>
Other	1.16 (0.72, 1.87)	1.10 (0.68, 1.77)
Unknown	1.16 (0.97, 1.40)	1.07 (0.87, 1.30)
<b>Years HIV positive (per 5 years)</b>	<b>1.06 (1.02, 1.11)</b>	<b>1.12 (1.07, 1.17)</b>
<b>CD4 at viral suppression (per 100 cells/μL)</b>	<b>0.90 (0.88, 0.92)</b>	<b>0.89 (0.87, 0.91)</b>
<b>Alcohol abuse</b>		
No	ref	ref
Yes	<b>1.36 (1.12, 1.66)</b>	1.17 (0.93, 1.46)
Unknown	<b>1.30 (1.13, 1.49)</b>	<b>1.25 (1.03, 1.51)</b>
<b>History of substance abuse</b>		
No	ref	ref
Yes	<b>1.36 (1.15, 1.60)</b>	1.11 (0.93, 1.33)
Unknown	1.01 (0.87, 1.18)	0.88 (0.73, 1.07)
<b>Hepatitis C status</b>		

Characteristic	HR (95%CI)	aHR (95%CI) <sup>2</sup>
Negative	ref	ref
Positive	1.11 (0.90, 1.37)	0.82 (0.64, 1.04)
<b>Hepatitis B status</b>		
Negative	ref	ref
Positive	1.04 (0.69, 1.55)	0.92 (0.61, 1.39)
<b>Mental Health/Depression</b>		
No	ref	ref
Yes	<b>1.47 (1.28, 1.70)</b>	<b>1.24 (1.06, 1.45)</b>

<sup>1</sup> Includes participants who were suppressed at study enrollment or achieved suppression during observation; excludes participants with unknown CD4 cell count or years HIV-diagnosed. Clinic type (hospital vs. CBO) was adjusted for as a random effect in the model.

<sup>2</sup> Adjusted for all other variables in the model. Significant hazard ratios and 95% confidence intervals are bolded.

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