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## Social capital, depressive symptoms, and HIV viral suppression among young Black, gay, bisexual and other men who have sex with men living with HIV

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### Abstract

Social capital, the sum of an individual's resource-containing social network connections, has been proposed as a facilitator of successful HIV care engagement. We explored relationships between social capital, psychological covariates (depression, stigma and internalized homonegativity), and viral suppression in a sample of young Black gay, bisexual and other men who have sex with men (YB-GBMSM). We recruited 81 HIV-positive YB-GBMSM 18–24 years of age from a clinic setting. Participants completed a cross-sectional survey, and HIV-1 viral load (VL) measurements were extracted from the medical record. Sixty-five percent (65%) were virally suppressed (HIV-1 VL < 40 copies/ml). Forty-seven percent (47%) had a positive depression screen. Depressive symptoms affected viral suppression differently in YB-GBMSM with lower vs. higher social capital ( $p = 0.046$ , test for statistical interaction between depression and social

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#### Compliance with Ethical Standards

All authors disclose no potential conflicts of interest. All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee (Emory IRB and Grady Research Oversight Committee) and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. Informed consent was obtained from all individual participants included in the study.

capital). The odds of viral suppression among YB-GBMSM with lower social capital was 93% lower among those with depressive symptoms (OR= 0.07, p= 0.002); however, there was no association between depressive symptoms and viral suppression among those with higher social capital. Our results suggest that social capital may buffer the strong negative effects of depressive symptoms on clinical outcomes in YB-GBMSM living with HIV. In addition to treating depression, there is a role for interventions to augment social capital among YB-GBMSM living with HIV as a strategy for enhancing care engagement.

### Keywords

social capital; engagement in care; youth; HIV

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## INTRODUCTION

*Social capital*, defined here as “the net worth of an individual’s resource-containing reciprocal, and trustworthy social network connections” (1, 2), has been proposed as a potentially powerful facilitator of care engagement for people living with HIV (3, 4). Despite high levels of interest in social capital internationally, there is relatively little research focused on social capital among key HIV-affected populations in the United States (US), such as young Black gay, bisexual, and other men who have sex with men (YB-GBMSM). YB-GBMSM have the highest HIV incidence rates of any demographic group in the US (5), and they are also at risk for suboptimal engagement outcomes across the HIV care continuum, including lower rates of linkage to care, retention in care, and viral suppression relative to older, White, or female individuals living with HIV (6, 7). Given the critical importance of engagement in HIV care for both individual and public health, identifying facilitators of care engagement for YB-GBMSM living with HIV could be critically important for policy, programming and intervention development.

Although social capital *per se* remains underexplored among YB-GBMSM living with HIV in the US, there is ample evidence of the significance of social relationships in their lives. Previous research has identified biological family, friends, and other natural mentors as important social connections and sources of support for YB-GBMSM (8, 9). At the dyadic level, social relationships have been successfully used to enhance medication adherence and retention in care among YB-GBMSM through identification of a support person within their existing networks (10, 11). Additionally, researchers have described unique endogenous social structures within YB-GBMSM communities such as the house/ball scene (in which groups called “houses” composed primarily of YB-GBMSM compete against one another in underground dance events known as “balls”) and surrogate “gay families” that provide important social resources for their members (12, 13). Taken together, this body of research provides indirect evidence to support the potential role of social capital in enhancing HIV care engagement in this population.

Although this topic is understudied in the US, research highlighting the role of social capital in HIV care has previously been conducted in diverse international settings. A multi-country ethnographic study in sub-Saharan Africa, for example, found social capital to be both a

direct and indirect facilitator of care engagement among people living with HIV (14, 15). The direct effects of social capital were illustrated by individuals describing use of social connections to address concrete barriers to care such as transportation and financial needs. At other times, social capital also acted indirectly as a buffer; for example, when supportive social connections helped people to cope with the adverse mental health consequences of stigmatizing community attitudes. Studies of women and children living with HIV in Ethiopia and Kenya have similarly highlighted the positive role of social capital in coping and resilience in these key populations (16, 17). Importantly, social capital is not only an important influence on care engagement, it is also a potentially modifiable resource that can serve as an intervention target (3, 18). Several types of interventions have been developed to enhance social capital among populations infected with, or at risk for HIV; these include peer-based interventions, community empowerment interventions, group-based microfinance interventions and support group interventions (3).

To date, social capital has not been specifically linked to HIV care outcomes among YB-GBMSM in the US. Inquiry into the functioning of social capital among YB-GBMSM may help to identify novel targets for innovative interventions aiming to improve HIV care engagement among YB-GBMSM. We therefore sought to explore direct and indirect effects of social capital in a clinic-recruited sample of YB-GBMSM living with HIV.

### Theoretical Background

As noted above, we define social capital from the individual's perspective as "the net worth of an individual's resource-containing reciprocal, and trustworthy social network connections" (1, 2). Social capital is a multidimensional construct that includes structural, cognitive and functional features of social networks (19). Features such as the size and diversity of a person's network, as well as the socioeconomic and other resources owned by contacts in that network, all have a bearing on the social capital that can be derived therein (20, 21). Social capital can further be divided into two main subtypes: *bonding capital* and *bridging capital* (22). Bonding capital refers to resources accessed within social groups whose members are relatively similar in terms of sociodemographic factors (e.g., emotional support from close friends of similar cultural and economic backgrounds), while bridging capital refers to resources derived from cross-identity connections (e.g., information about a job opportunity from a co-worker who is not from the same demographic group) (22). Both bonding and bridging capital have been theorized to have positive impacts on health outcomes.

A few conceptual distinctions are worth noting. Our focus on personally owned social capital (inherent in an individual's network connections) differs from that of scholars who measure social capital and its effects at the community level (e.g., focusing on the levels of social cohesion and civic participation within communities) (19, 23, 24). Although both personal and community-level social capital have important implications for HIV, we were interested in individual-level clinical outcomes for the purpose of this analysis and therefore chose to focus on the role of *personal* social capital in HIV care engagement. Finally, it is worth noting the distinction between social capital and social support, as these are closely related but separate constructs. Social capital is a description of the resources (including

emotional social support, but also other factors such as information channels or social credentials) available in an individual's social network (25). On the other hand, social support measures tend to focus more on an individual's satisfaction with the support they receive, as opposed to assessing the potential for support in the network as a whole (26–28).

In our review of the literature on social capital and HIV care outcomes, several factors emerged as potentially important covariates that should be included in our analysis: namely, depression and stigma. Depressive symptoms have repeatedly been demonstrated to impede effective engagement in care among people living with HIV. Prior research has delineated negative relationships between depression and a range of outcomes along the HIV care continuum, including medication adherence/acceptance (29–31), missed clinic visits (32), and viral suppression (31). Depression is also a common mental health problem, with a disproportionately high prevalence among youth living with HIV (29). Of note, prior research in other vulnerable populations, such as homeless individuals and victims of natural disasters, suggests that social capital can have a significant and beneficial impact on levels of depressive symptoms (33–35).

Stigma is another factor that is important for HIV care engagement and may be related to depression and social capital among YB-GBMSM and other people living with HIV. Ware et al.'s work in sub-Saharan Africa specifically cited stigma as a major barrier to care that appeared to be buffered by social capital (14). It is important to note that GBMSM are often subject to multiple stigmas, including HIV-specific stigma as well as stigma related to same-sex identity. Aspects of both HIV stigma and internalized homonegativity have been negatively associated with medical appointment adherence among YB-GBMSM in our previous research (36, 37).

The current analysis is an exploratory study that was designed to examine associations between social capital and HIV viral suppression (the ultimate measure of engagement along the HIV care continuum) among YB-GBMSM. We hypothesized that social capital would be positively associated with HIV viral load suppression, and therefore designed the study to address the following objectives: (1) To determine whether social capital (total social capital, bonding social capital and bridging social capital) had a positive direct association with HIV viral load suppression; (2) To determine whether any relationship between social capital and HIV viral load suppression was confounded by depressive symptoms, HIV stigma, and/or internalized homonegativity. (3) To determine whether social capital might indirectly impact (act as an effect modifier) between psychosocial covariates (depressive symptoms, stigma, internalized homonegativity) and HIV viral load suppression.

## METHODS

We recruited 81 YB-GBMSM living with HIV from a pediatric/adolescent clinic in a large Southeastern city in the United States, between November 2015 and July 2016. Potential participants were approached during their visits to medical providers or other support staff in the clinic. Patients who self-identified as Black and male, reported a history of ever having sex with a male partner, and had been in care at the clinic for at least one year, were invited to participate. This was largely a convenience sample; however, in an effort to recruit a

sample that included incompletely engaged individuals, we stratified our recruitment so that half of the participants had missed more than 25% of their scheduled visits within the previous year, while the other half had not. Participants completed a one-time Audio Computer Assisted Self Interview (ACASI) that included measures of social capital, depressive symptoms, and other psychosocial constructs. A trained graduate research assistant subsequently abstracted clinical data from the patient's electronic medical record (EMR), including scheduled and missed appointments as well as the most recent viral load measurement.

## Measures

**Social Capital**—We modified Chen's Personal Social Capital Scale (2) in order to measure social capital in our participants. This scale contains 10 items with 49 sub-items; each sub-item is scored on a Likert-type scale ranging from 1 to 5 and each item's score is the mean of the sub-item scores. Total scale scores therefore range from 10 (lowest social capital) to 50 (highest social capital). The Personal Social Capital Scale is theoretically based and contains subscales that measure bonding capital and bridging capital. Of note, however, this measure was originally developed for use among adults in China and therefore contained items not likely to apply in our context.

In order to modify the scale for use among YB-GBMSM, our study team (consisting of researchers with experience working directly with YB-GBMSM in our local community) first examined each scale item and changed wording that was unlikely to be relevant for our participants or that might be difficult to understand (e.g., references to "country fellows"). We also added items that seemed likely to be important for our participants based on our prior work with YB-GBMSM (e.g., questions about lesbian, gay, bisexual, transgender and queer [LGBTQ] organizations and college fraternities). Next, we conducted individual cognitive interviews with a convenience sample of five YB-GBMSM to solicit input on the readability, clarity and content of the scale. We added items and clarified wording based on their feedback. Finally, we piloted the modified scale in an online sample of  $n=204$  geographically diverse YB-GBMSM aged 18–29 recruited via a popular social networking website that caters specifically to Black GBMSM, and found the scale to have excellent reliability in that sample ( $\alpha=0.88$ ); the same was true in the current sample ( $\alpha=0.86$ ).

**Depression**—We utilized the Centers for Epidemiologic Studies-Depression Revised version (CESD-R) to measure depressive symptoms (38). The CESD-R is a 20-item scale that requires participants to characterize their frequency of depressive symptoms using a 5-point Likert scale ranging from 0 (*not at all or less than 1 day last week*) to 4 (*nearly every day for two weeks*). A score of 16 is consistent with clinically significant depressive symptoms. (39) The CESD-R has demonstrated validity and reliability in numerous other studies of people living with HIV (40). Reliability was excellent in our sample as well ( $\alpha=0.91$ ).

**HIV Stigma**—This construct was measured using the Revised HIV Stigma scale for youth, a 10-item scale that asks participants to rate their agreement with various statements about attitudes towards people with HIV, disclosure concerns, and negative self-image (41). The

scale utilizes a 4-point Likert scale ranging from 1 (*strongly agree*) to 4 (*strongly disagree*) and was reliable in our sample ( $\alpha = 0.84$ ).

**Internalized Homonegativity**—We utilized Mayfield’s Internalized Homonegativity Inventory (IHNI) to measure this construct (42). This measure asks participants to rate their agreement with a series of statements about their feelings regarding their own sexual orientation, using a 6-point Likert scale ranging from 1 (*strongly disagree*) to 6 (*strongly agree*). The IHNI had excellent reliability in our sample ( $\alpha=0.92$ ).

**Viral Suppression**—Viral suppression was defined here as HIV-1 RNA (viral load) below the limit of detection for the assay used in the clinical encounter. The most commonly used assays have lower limits of either 20 or 40 copies/mL, depending on the patient’s insurance provider (or lack thereof), which in turn determines the laboratory that ultimately performs the test. We utilized the participant’s most recent viral load measurement, which was in most cases collected on the date of the survey, and which was always within 90 days of the survey date.

## Analysis

The potential association of each of the factors in Table 2 with the outcome (viral suppression) was evaluated using logistic regression modeling. Odds ratios (ORs) were calculated to measure the degree of association between risk factors and viral suppression. Continuous predictors were included in the logistic regression models assuming a linear relationship between these predictors and viral suppression. Additionally, continuous predictors were dichotomized at the median, assuming the relationship between the predictor and viral suppression was flat within the intervals.

Due to the limited number of patients without viral suppression and concern for model overfitting, covariates included in multivariable logistic regression analyses were limited to main effects. Covariate selection was driven by available knowledge, theoretical expectations, and biological plausibility of potential confounders, taking into consideration the hypothesis of interest. The adjusted OR and its 95% confidence interval were calculated for each risk factor in the presence of others in the final models.

Subgroup analyses were used to evaluate potential moderating effects of total, bonding and bridging social capital on the relationship between depressive symptoms and viral suppression. The effect of total social capital (and separately, bonding social capital and bridging social capital) was investigated by including the statistical interaction between depressive symptoms (CESD-R: <16 or  $\geq 16$ ) and social capital in a logistic regression model.

## RESULTS

### Sample Characteristics

Our sample ranged in age from 18–24 years (mean=22, SD= 1.5). A large majority (84%) described their sexual orientation as gay, with few participants self-identifying as bisexual, straight/heterosexual or questioning/unsure. Most had completed at least a high school

diploma or General Education Development (GED – high school equivalency) certification, and many had started college or technical school as well. Two-thirds reported current employment. In terms of their engagement in care, half of the sample had missed over 25% of their appointments, and 71.6% had missed at least one appointment. In spite of this, 56/81 (69.1%) self-reported very good or excellent adherence, and 53/81 (65.4%) were virally suppressed at the time of the survey (Table 1).

The prevalence of depressive symptoms was high in our sample. Nearly half (47%) of our participants scored above the CESD-R cutoff for depressive symptoms. The mean social capital score was 25 (SD=6.4), which is comparable to results among Chinese respondents in Chen's original scale validation study (2).

### Bivariable analyses

Participants with lower total social capital (below the median of 25) were less likely to be virally suppressed compared to participants with higher total social capital (59% vs. 73%, OR 0.54, 95% CI 0.28–1.04;  $p=0.06$ , Table 2); this finding approached but did not quite reach statistical significance. We also analyzed the subscales of social capital to test their separate relationships with viral suppression and found that low bonding social capital (below the median of 12) retained this relationship with viral suppression (OR 0.54, 95% CI 0.28–1.04;  $p=0.06$ ), while bridging social capital did not appear to be related (OR 0.96, 95% CI 0.39–2.41;  $p=0.93$ ). Participants with depressive symptoms (CESD-R  $\geq 16$ ) were less likely to be virally suppressed (18/38, 47%) compared to participants who did not have depressive symptoms (35/43, 81%; OR 0.21, 95% CI 0.10–0.42;  $p<0.01$ ). Several other baseline demographic characteristics summarized in Table 2 (i.e. older age, housing stability within last 6 months) also displayed significant associations with viral suppression.

### Multivariable analyses

From multivariable logistic regression, only depressive symptoms remained associated with viral suppression (Table 3). Total social capital, HIV stigma, internalized homonegativity, bonding social capital (data not shown) and bridging social capital (data not shown) were not independent predictors of viral suppression. The adjusted odds of viral suppression was significantly lower in participants with depressive symptoms compared to participants who were not depressed (adjusted OR 0.26, 95% CI 0.09–0.78;  $p = 0.02$ , Table 3).

### Subgroup analyses

We found evidence of effect modification by total social capital on the association between depressive symptoms and viral suppression. When comparing YB-GBMSM with lower total social capital (below the median score of 25;  $N=41$ ) to YB-GBMSM with higher total social capital (above 25;  $N=40$ ), the interaction was significant (Table 4, test for statistical interaction between depressive symptoms and total social capital,  $p=0.046$ ). There was no association between depressive symptoms and viral suppression among those with higher total social capital (OR 0.63, 95% CI 0.15–2.59;  $p=0.52$ ). However, the odds of viral suppression among YB-GBMSM with lower total social capital was 93% lower in depressed participants compared to participants without depressive symptoms (OR 0.07, 95% CI 0.01–0.37;  $p=0.002$ ).

We also examined potential moderating effects of the social capital subscales (bonding and bridging capital) on the relationship between depressive symptoms and viral suppression. In these cases, the interaction was not significant; neither bonding nor bridging capital alone moderated the relationship between depressive symptoms and viral suppression (test for statistical interaction between depressive symptoms and bonding social capital,  $p=0.31$ ; test for statistical interaction between depressive symptoms and bonding social capital,  $p=0.11$ ; data not shown).

## DISCUSSION

The prevalence of depressive symptoms in our sample was high, even compared with other studies of US youth living with HIV (29, 43, 44). The strong association between depressive symptoms and lack of viral suppression, which has been well documented in other studies of youth living with HIV (43, 45), was further supported here in our specific study sample of YB-GBMSM. There also appeared to be a trend towards a direct association between level of social capital (particularly the bonding capital component) and the likelihood of viral suppression. Importantly, for patients with depressive symptoms, social capital was a protective factor – the detrimental effect of depressive symptoms on viral suppression was worse for those with low social capital. These results were consistent with our theoretical expectations, and with several other studies that examined related study questions in similar populations (46, 47).

The stronger association of bonding social capital (relative to bridging social capital) with viral suppression was a notable finding. Previous research on the influence of bonding and bridging capital on health has yielded conflicting results. The importance of bonding social capital has been demonstrated for mental health in particular (19, 48). However, others have also highlighted stress and strain that can result from the close social networks that yield bonding social capital, as such networks may produce conflicts, envy, or more onerous obligations for their members (20, 49). On the other hand, certain scholars have highlighted the relative importance of bridging capital, or “weak ties” that link individuals to resources they would not normally be able to access. For example, a study focused on health information seeking among Black adults highlighted the importance of bridging ties to healthcare professionals as integral to the process of seeking healthcare information (50). For our sample of YB-GBMSM, however, the associations with bonding social capital, appeared to be more positive than negative (and stronger than associations with bridging capital) for the health outcome in question. Our findings are consistent with some, but not all, literature in the field, highlighting the population-specific and disease-specific nature of these constructs and conceptual relationships.

The function of social capital as a buffer against depressive symptoms is consistent with a prior US study that found social network factors (including size of emotional, financial and medical support networks) to be significantly associated with fewer depressive symptoms in a mixed-serostatus sample of Black men who have sex with men (MSM) participating in the HIV Prevention Trials Network (HPTN) 061 protocol (46). Although this study did not label their predictor variables as social capital *per se*, the size of networks and different support functions described by the authors align well with specific items in our measure (e.g., items



assessing network size and items assessing the presence of specific assets such as “medical or health knowledge”, “large wealth”, etc.) and with the concept of social capital more generally.

In another very pertinent study, Friedman et al. utilized data from the Multicenter AIDS Cohort Study (MACS) of MSM living with HIV, and found that functional social support (a single item measuring the number of people an individual could “count on”) was an effect modifier between concomitant syndemic indicators (including depression) and viral suppression (47). Again, the authors did not frame their functional social support item in terms of social capital theory; however, this measurement of the size of one’s help network correlates directly with items on our social capital measure. We should note, however, that there are important differences between this study and ours; the majority of the MACS cohort is white and over 40 years of age. Nevertheless, the similar findings in terms of the effect modifying relationship support the validity of our results and the theoretical relationships they suggest.

### Limitations and Strengths

Our study has several limitations. Our sample size was small, limiting our ability to make statistical inferences. This is in part because we initially powered our study on retention in care, and may therefore have been underpowered to detect smaller effect sizes on viral suppression. Additionally, the recruitment of patients directly from the clinic setting skewed our sample towards those who were more easily able to maintain some level of care engagement. To offset this bias, we did purposively recruit our sample so that half of our participants were less compliant youth (who had missed 25% or more of their visits in the last year). Still, all participants were at least engaged in care enough to come to a clinic visit, and future studies should aim to recruit from the community to reach a wider range of YB-GBMSM. Finally, we recruited from a single site and cannot gauge how generalizable our results may or may not be to other geographic or clinical settings – multisite investigations are indicated in the future.

Still, several strengths bear mention. In our assessment of care engagement, we were able to utilize biological measurements obtained from the EMR abstraction, as opposed to relying on participant self-report. Our analysis was culturally specific: to measure social capital, we utilized a scale that we had previously adapted and tested specifically in YB-GBMSM – this and all reported measures had excellent reliability in our sample. Finally, this analysis focused not only on barriers to care (e.g., depressive symptoms) but also began to demonstrate the role of social capital – an important, modifiable resilience factor with the potential to improve outcomes for YB-GBMSM living with HIV.

### Conclusions

Our results suggest a potential role for interventions that augment social capital among YB-GBMSM living with HIV, particularly for those with depressive symptoms. Interventions have previously been developed to intentionally create social capital in non-U.S. settings; these include community empowerment interventions, group-based microfinance, support groups, and peer-led interventions (3, 18, 51, 52). To our knowledge, however, no such

interventions have been specifically developed for YB-GBMSM living with HIV in the United States. Future interventions aiming to improve social capital with this population will need to be culturally tailored, as the nature and role of social capital varies considerably in different cultural and demographic groups (53). However, we believe that social capital-based interventions hold significant promise for YB-GBMSM. The appeal of a social capital-based intervention is that it naturally lends itself to a strengths-based approach – such interventions would not need to create new social networks *de novo*, but could instead build upon the naturally existing social structures and mentoring relationships that YB-GBMSM often describe (8, 13). By augmenting resources that YB-GBMSM already possess, a social capital-based approach has the potential to be a feasible and sustainable way to enhance care engagement in this population.

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**Table 1**

## Baseline Characteristics (n = 81)

Age (years) mean $\pm$ SD	22.4 $\pm$ 1.6
Sexual Orientation	
Gay or homosexual	68 (84.0%)
Bisexual	9 (11.1%)
Heterosexual/Straight	1 (1.2%)
Questioning/Unsure	3 (3.7%)
Education	
Did not complete HS	10 (12.3%)
HS/GED/Post HS	71 (87.7%)
Current Employment	54 (66.7%)
Missed appointments	
Missed more than 25% appointments	41 (50.6%)
At least one appointment missed	51 (71.6%)
Self-reported good or excellent adherence	56 (69.1%)
Virologically suppressed	53 (65.4%)

Unless otherwise noted, continuous variables are reported as mean  $\pm$  SD and categorical variables are reported as no. (%).

**Table 2**

Bivariable logistic regression analysis of variables potentially associated with viral suppression

		Prevalence of viral suppression	Odds ratio [95% CI]	p
ALL patients		53/81 (65%)		
Age	<23	20/36 (56%)	0.46 [0.24,0.88 ]	0.019
	23	33/45 (73%)	reference	
Education	did not complete HS	5/10 (50%)	0.48 [0.19,1.23]	0.13
	HS/GED/Post HS	48/71 (68%)	reference	
Working	Yes	39/54 (72%)	2.41 [1.22,4.77 ]	0.011
	No	14/27 (52%)	reference	
Sexual Orientation	bisexual, heterosexual/straight, questioning/unsure	7/13 (54%)	0.56 [0.24,1.31 ]	0.18
	homosexual/gay	46/68 (68%)	reference	
Housing				0.0002
	Lives alone	31/41 (76%)	reference	
	Living with family	14/19 (74%)	0.90 [0.37,2.18 ]	
	Other	8/21 (38%)	0.20 [0.09,0.44 ]	
Moved in last 6 months	No	28/36 (78%)	2.8 [1.4,5.6 ]	0.0036
	Yes	25/45 (56%)	reference	
Total Social Capital (median =25)	<25	24/41 (59%)	0.54 [0.28,1.04 ]	0.06
	25	29/40 (73%)	reference	
Bonding Social Capital (median 12)	<12	24/41 (59%)	0.54 [0.28,1.04 ]	0.06
	12	29/40 (73%)	reference	
Bridging Social Capital (median 13.5)	<13.5	26/40 (65%)	0.96 [0.39,2.41 ]	0.93
	13.5	27/41 (66%)	reference	
HIV Stigma (median 26)	<26	28/38 (74%)	2.02 [0.79,5.17 ]	0.14
	26	25/43 (58%)	reference	
Internalized Homonegativity (median 48)	<48	30/39 (77%)	2.75 [1.05,7.2 ]	0.04
	48	23/42 (55%)	reference	
Depressive Symptoms (CESD-R)	<16 Not Depressed	35/43 (81%)	reference	
	16 Depressed	18/38 (47%)	0.21 [0.1,0.42 ]	<.0001

Multivariable logistic regression analysis of variables potentially associated with viral suppression

**Table 3**

<b>Risk Factor</b>	<b>β</b>	<b>SE</b>	<b>OR [95% CI]</b>	<b>P</b>	
Total social capital (median 25)	<25 vs 25	-0.3173	0.5211	0.73 [0.26,2.02]	0.54
Depressive symptoms (<16 or 16)	Yes vs No	-1.3366	0.5567	0.26 [0.09,0.78]	0.016
HIV stigma (median 26)	<26 vs 26	-0.1526	0.612	0.80 [0.26,2.85]	0.80
Internalized Homonegativity (median 48)	<48 vs 48	0.671	0.581	1.96 [0.63,6.11]	0.248

Abbreviations: β, estimated regression coefficient; SE, standard error; OR, odds ratio; CI, confidence interval.



**Table 4**

Logistic regression model with total social capital and depressive symptoms plus the interaction as risk factors potentially associated with viral suppression

Subgroup	Odds Ratio (95% CI)	P Value
Depressed: Total social capital (<25 vs ≥25)	0.27 (0.07, 1.05)	0.06
Not Depressed: Total social capital (<25 vs ≥25)	2.53 (0.45, 14.3)	0.30
Low social capital (depressed/not depressed)	0.07 (0.01, 0.37)	0.002
High social capital (depressed/not depressed)	0.63 (0.15, 2.59)	0.52

P value for interaction between total social capital and depressive symptoms = **0.046**

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