Title

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Journal

AIDS, 34(11)

ISSN

0269-9370

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Publication Date

2020-09-01

DOI

10.1097/qad.0000000000002595

Peer reviewed

HHS Public Access

Author manuscript

AIDS. Author manuscript; available in PMC 2021 October 13.

Published in final edited form as:

AIDS. 2020 September 01; 34(11): 1665–1671. doi:10.1097/QAD.0000000000002595.

Internalized HIV Stigma Predicts Subsequent Viremia in US HIV Patients through Depressive Symptoms and ART Adherence

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Abstract

Objective: We sought to examine the prospective association between internalized HIV stigma and unsuppressed viral load and to investigate whether this relationship was sequentially mediated by depressive symptoms and antiretroviral (ART) adherence.

Design: Longitudinal study in a multi-site observational clinical cohort.

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Authorship Statement: Designed the study (KAC, MOJ, TBN), contributed substantially to data acquisition, analysis or interpretation (all authors), drafted the manuscript (KAC, TBN, SD,) critically revised the manuscript for important intellectual content (NL, JS, MJM, HCM, RF, WCM, RDM, KHM, SN, MOJ). All authors approved the final version of the published manuscript.

Previous Presentation: Presented in part at the 14th International Conference on HIV Treatment and Prevention Adherence, June 17-19, 2019, Miami Beach, FL.

Methods: The Center for AIDS Research Network of Integrated Clinical Systems (CNICS) Patient-Reported Outcomes (PROs) survey measures internalized HIV stigma yearly using a 4-item assessment (response scale 1= strongly disagree to 5=strongly agree). We obtained PRO, lab, and appointment data from six CNICS sites. We used multivariable logistic regression to examine the association between mean stigma and subsequent viremia. We then used Bayesian sequential mediation to fit a longitudinal sequential path model spanning four time points to test if depressive symptoms at T_1 and ART adherence at T_2 mediated the effect of stigma at T_0 on viral load at T_3, adjusting for baseline covariates.

Results: Between February 2016 – November 2018, 6,859 patients underwent stigma assessment and were 81% cis-men, 38% Black, 16% Latinx, 32% heterosexual-identified, and 49% ≥50 years of age. Mean stigma level was 2.00 (SD 1.08). Stigma was significantly associated with subsequent viremia (aOR =1.16, 95% CI: 1.05-1.28, p 0.004), as were younger age and Black race. The chained indirect effect from stigma to unsuppressed viral load through depressive symptoms and then adherence was significant (standardized β = 0.002; SD = 0.001).

Conclusions: Internalized HIV stigma positively predicts subsequent viremia through depressive symptoms and ART adherence. Addressing the link between stigma and depressive symptoms could help improve viral suppression.

INTRODUCTION

HIV-related stigma exerts a profound influence on quality of life for people living with HIV (PLWH). Increasing evidence shows that HIV-related stigma has deleterious effects on HIV care and treatment engagement.[1] A fairly robust literature supports a link between HIV-related stigma and suboptimal antiretroviral (ART) adherence,[2,3] but limited empirical data exist to elucidate the mechanisms that may account for this relationship. Depressive symptoms and decreased social support are two potential mediators,[4] but analyses to date have been limited by use of cross-sectional designs, lack of viral load outcomes, and stigma scales that combine more than one form of HIV-related stigma, each of which may operate in different ways.

The role of internalized stigma, or, negative self-attitudes and beliefs about living with HIV, is particularly important because it applies to all PLWH, regardless of whether they have disclosed their status. Other forms of stigma, such as enacted stigma (the experience of HIV-related prejudice or discrimination), are predicated on others' knowing one's HIV-positive status, which may not be the case for some PLWH.[5] A cross-sectional mediation analysis conducted in an observational cohort of women in the U.S. demonstrated that internalized stigma predicted less perceived social support, which in turn predicted more depressive symptoms, which were then associated with suboptimal adherence.[6] However, a large cross-sectional assessment of internalized HIV stigma in the Medical Monitoring Project did not find an association between internalized HIV stigma and virologic suppression.[7] These findings illustrate the difficulty of delineating a clear mechanistic pathway between internalized HIV stigma and key care cascade outcomes when working with cross-sectional data, even when datasets include large numbers of PLWH. A recent prospective analysis of African-American women in Chicago, IL, and Birmingham, AL, also showed that internalized HIV stigma was not associated with subsequent viral load outcomes.[8]

Given that importance of understanding the relationship between internalized HIV stigma and HIV viral load, our group sought to leverage the infrastructure of a large, geographically diverse observational cohort of patients in routine HIV care in the US, the CFAR Network of Integrated Clinical Systems (CNICS), to measure internalized stigma and its relationship to key HIV care cascade outcomes. We previously demonstrated that internalized HIV stigma was significantly associated with concurrent viremia and a history of missed primary care visits.[9] Having established this cross-sectional association, the objectives of the current study were to investigate: 1) the prospective association between internalized HIV stigma and subsequent viral load, and; 2) whether depressive symptoms and ART adherence sequentially mediated the relationship between stigma and viral load using a prospective longitudinal design.

METHODS

Study Setting and Participants

The CNICS cohort study uses electronic medical record data from 8 academic HIV clinics across the US: University of Alabama, Birmingham (UAB), University of Washington (UW), University of California, San Diego (UCSD), Case Western Reserve University (CWRU), University of California, San Francisco (UCSF), Fenway Health Center (FCH), University of North Carolina at Chapel Hill (UNC), and Johns Hopkins University (JHU).[10] Patient-reported outcomes (PROs) consisting of validated measures of ART adherence and other psychosocial factors known to impact HIV outcomes are collected via self-administered surveys on touch screen tablets or computers every four to six months as part of primary care.[11] Each CNICS site has institutional review board approval to collect and transmit EMR and PRO data to a coordinating center at UW, where quality checks are performed before de-identified data is provided to investigators.

In February 2016, we added a four-item version of a validated six-item internalized HIV stigma scale[12] into the PRO survey on a yearly basis. The items were “Having HIV makes me feel like a bad person,” “Having HIV is disgusting to me,” “I think less of myself because I have HIV,” and “I feel ashamed of having HIV” (response categories 1= strongly disagree 2=disagree 3=neutral 4=agree 5=strongly agree). We chose the brief version because of the need to decrease burden on clinic flow, given that PROs are administered in routine care rather than in a study setting. A composite stigma score based on the mean of non-missing items was calculated (scores ranged from 1-5) as long as at least 75% (3 items) were non-missing, otherwise the stigma score was set to missing. Our previous investigation of stigma in CNICS using this scale found high Cronbach’s alpha (0.91).[9] The population for this study consisted of CNICS patients from UAB, UW, UCSD, UCSF, FCH, and JHU. Due to variability in the rollout of the stigma measurement, each site had different periods of stigma assessment represented in the final dataset, ranging from 13 to 23 months.

Mediation Model

We created a mediation model that listed the stigma predictor, mediators, and viral load outcome in sequence, beginning with stigma assessment at T_0, followed by subsequent assessment of depressive symptoms at T_1, followed by a subsequent ART adherence

assessment at T_2, and terminating with the first measurement of viral load (T_3) at least 30 days after the adherence assessment or at least 240 days after stigma assessment if subsequent PROs were not available (Figure 1). The 240 day window was chosen because the minimum window between PRO assessments at most CNICS sites is 105 days (105 days + 105 days + 30 days = 240 days).

Outcome

The primary outcome was unsuppressed viral load, defined as a viral load measurement >200 copies/mL at least 30 days after adherence assessment or at least 240 days after stigma assessment.

Predictor

The primary predictor was internalized HIV stigma measured by the scale described above.

Mediators

Additional variables in the mediation model were depressive symptoms and ART adherence. Depressive symptoms were assessed using the Patient Health Questionnaire-9 (PHQ-9).[13] As with stigma, the mean of PHQ-9 items was calculated for participants who had valid data for ≥75% of PHQ-9 items (i.e. ≥7 items) and, to aid in interpretability, the mean was multiplied by 9 to generate a pseudo-sum of depressive symptoms. The composite measure of depressive symptoms was set to missing for participants with ≥3 items missing.

ART adherence was assessed using the visual analogue scale (VAS),[14,15] in which respondents are asked to mark a point on a line between 0-100% that best represents their adherence over the past month, and a single-item self-report measure that uses a 5 point Likert scale ranging from “very poor” to “excellent” to describe ART adherence over the past 4 weeks.[16] ART adherence was operationalized as an observed variable such that “1” indicated 100% adherence on the VAS and “excellent” on the single-item self-report and “0” was defined as either <100% adherence by VAS and any response that was less than “excellent.” We operationalized ART adherence in this way because we sought to differentiate patients into those reporting optimal adherence and those with suboptimal adherence. To maximize rigor, we set a high threshold of being at the top end of both ART adherence measurements.

Covariates

Covariates were age, gender identity, race/ethnicity, sexual orientation, length of time in CNICS, and CNICS site.[9]

Analysis

We computed descriptive statistics for the study population using SAS 9.4. We then fitted a multivariable logistic regression model in M*plus* version 8.4 of stigma and patient characteristic covariates at T_0 predicting unsuppressed viral load measured at T_3 using direct maximum likelihood estimation with robust standard errors and 95% confidence intervals as a measure of precision. We screened for two-way interactions of stigma with each patient characteristic. The BIC statistic was used to compare the fit of models with interactions

included to the main effects only model; the model with the lowest BIC was selected as the better-fitting model. Linearity of the association of continuous stigma scores with the viral load outcome was performed by including restricted cubic splines of stigma in the analysis and testing the significance of the non-linear splines.[17] Using Bayesian sequential mediation (chained indirect effect) analysis, we then used M*plus* to test if depressive symptoms and ART adherence mediated the relationship between stigma and unsuppressed viral load by estimating indirect effects of stigma on viral load through depressive symptoms and ART adherence, adjusting for T_0 patient characteristic covariates. To further strengthen causal inference with regard to the role of the predictor, mediators, and outcome, we also adjusted for baseline depressive symptoms and ART adherence. All analyses used alpha=0.05 to determine statistical significance.

Cases with missing data on covariates, mediators, or the viral load outcome were included in the analyses under the assumption of being conditionally missing at random (MAR) in the logistic regression via direct maximum likelihood estimation and in the mediation analysis via Bayesian estimation. For logistic regression results, we report the adjusted odds ratio (aOR), its 95% confidence interval and *p*-value. Bayesian analysis based on the multivariate probit distribution was chosen to fit the mediation model because it can incorporate both continuous and binary mediators and outcomes in a chained sequence and yield appropriate asymmetric credible intervals for indirect effects. For Bayesian mediation analysis results, we report the unstandardized regression coefficient (*B*) and its 95% credible interval (CI) and the corresponding standardized regression coefficient estimate (β) and its posterior standard deviation (SD) for direct and indirect effects. These coefficients are linear regression coefficients for the regression of continuous depressive symptoms on the stigma composite score and probit regression coefficients for the regression of perfect adherence and viral load onto the stigma composite and depressive symptoms. Statistical significance at the 5% level is achieved if the 95% credible interval for the indirect effect does not include zero. Because M*plus* reports credible intervals to a maximum of three significant digits and the indirect effects based on the products of direct effects in a chained mediation analysis are small, it was necessary to rescale the stigma composite variable by dividing it by 10. This rescaling did not change conclusions regarding which direct and indirect effects were statistically significant.

RESULTS

The study sample consisted of 6,589 CNICS patients with a median time of CNICS of 6 years (Table 1). The distribution of stigma assessments across sites was as follows: UAB (30%), UW (12%), UCSD (29%), UCSF (7%), FHC (9%), and JHU (13%). Nearly half (49%) of patients were ≥50 years if age, 17% identified as cis-female and 3% as gender minority (e.g. transgender, non-binary), 38% were Black and 16% Latinx, and about one-third (32%) identified as heterosexual. ART use was reported by 93% at baseline, among whom 32% reported optimal adherence. Mean stigma score at baseline was 2.00 (SD 1.08) and mean PHQ-9 score was 5.33 (SD 5.97). At baseline, 13% had unsuppressed viral load, and among those with viral load measurements at T_3, ~9% had unsuppressed viral load. The median time from stigma assessment to viral load measurement was 476 days (IQR 364-595 days).

With regard to logistic regression results, the BIC statistic indicated that the main effects only logistic regression model was preferred to each competing model containing a two-way interaction, so the main effects-only model was chosen as the final model. The null hypothesis of linearity was not rejected ($p = 0.83$), so non-linear spline terms were dropped to yield a parsimonious and readily interpretable main-effects only model. In this model (Table 2), stigma positively predicted unsuppressed viral load such that a unit increase in mean stigma resulted in a 16% increase in the odds of unsuppressed viral load (aOR =1.16, 95% CI: 1.05-1.28, p 0.004). Of participant characteristics, younger age and Black race also positively predicted unsuppressed viral load. No other patient characteristics significantly predicted unsuppressed viral load.

For our second objective, the sequential mediation model resulted in one significant indirect effect. Adjusting for baseline covariates, the indirect effect was from stigma to unsuppressed viral load, through depressive symptoms and then by adherence (unstandardized $B = 0.018$; 95%CI = 0.001, 0.047; standardized β = 0.002; SD = 0.001). Stigma did not have a direct effect on unsuppressed viral load (unstandardized $B = 0.133$; 95%CI = −0.524, 0.804; standardized β = 0.012; SD= 0.031), indicating that the effect of stigma on unsuppressed viral load was expressed through depressive symptoms and adherence only. Figure 2 displays the standardized estimates in path diagram format.

DISCUSSION

This study makes an important contribution to the existing literature, as results demonstrated that internalized HIV stigma predicted unsuppressed viral load and that this relationship was sequentially mediated by depressive symptoms and then ART adherence. Further, our study is the first to examine the prospective association between internalized HIV stigma and viral load in a large, geographically diverse cohort of patients in routine HIV care in the US. While several cross-sectional studies have found that depressive symptoms mediate the relationship between internalized HIV stigma and ART adherence in women in the US,[6,18] our analysis uses a sample with diverse race/ethnicity, gender and sexual identity, longitudinal data and the biologic endpoint of viral load. Although levels of stigma in our study were not high, our results demonstrate that to move the needle on “getting to zero” it will be necessary to focus on the segment of the clinic population that does endorse stigma, even if it is a relatively small number of patients.

While we provide evidence for a pathway between internalized HIV stigma and viral load that has been outlined in several conceptual frameworks,[19,20] we did not have access to other important variables, such as social support,[6,21] which was not available in the dataset. However, investigators have now developed and validated a perceived social support scale within CNICS, including a short three-item version that can allow routine use in clinical care.[22] Understanding the role of substance use vis a vis HIV-related stigma is another crucial line of inquiry. We chose not to adjust for the use of alcohol or illicit drugs in our models because we believed that these variables might function as effect modifiers. Our current analysis lays the groundwork to advance a nuanced exploration of substance use and stigma within CNICS.

The main limitations of our analysis are that we were only able to assess one form of HIV-related stigma, though we did use a well-validated scale,[9,12] and that we were unable to assess the contribution of intersectional stigma, i.e. the convergence of multiple stigmatized identities, including those related to race/ethnicity, sexual or gender identity, or substance use. [23–25] We acknowledge that individuals who complete the PROs may be different than those who do not, however, in our prior cross-sectional analysis of stigma and viral load we used inverse probability weighting to upweight the responses of those with demographic profiles similar to non-responders, finding no difference from the results of unweighted models.[9] We also note that CNICS is a mature cohort with high levels of baseline viral suppression so our findings are most applicable to patients in care at similar clinics – further, we recognize that individuals with high levels of internalized HIV stigma may not come to HIV clinics at all; if this is the case then we are underestimating the magnitude of the relationship between internalized HIV stigma and unsuppressed viral load.

Our analysis may also be limited by the inability to address unmeasured confounders. Cases with partial data were included in our models under the missing at random (MAR) assumption, conditional on the other variables included in the analysis, maximizing the generalizability of the findings. However, if non-response or loss to follow-up occurred for reasons unrelated to the observed variables, bias could have occurred. With regard to measurement of depressive symptoms, we are unable in this analysis to account for referrals to mental health services, which could impact subsequent PHQ-9 scores. Finally, while our assumption that higher levels of stigma lead to higher levels of depressive symptoms is very plausible, it is possible those with high levels of depressive symptoms are more susceptible to negative self-views and that depression could precede stigma on the causal pathway.

In sum, we found that internalized HIV stigma had a modest but significant effect on the likelihood of subsequent viremia and that this relationship was mediated by depressive symptoms and ART adherence. Interventions that account for the role of internalized HIV stigma when addressing depressive symptoms are urgently needed.

Conflicts of Interest and Source of Funding:

None related to this manuscript. Dr. Christopoulos has received investigator-initiated grant support from Gilead Sciences and has served as a Medical Advisory Board member for Gilead. This work was supported by National Institutes of Health R01 MH102198-S1 and R24 AI067039.

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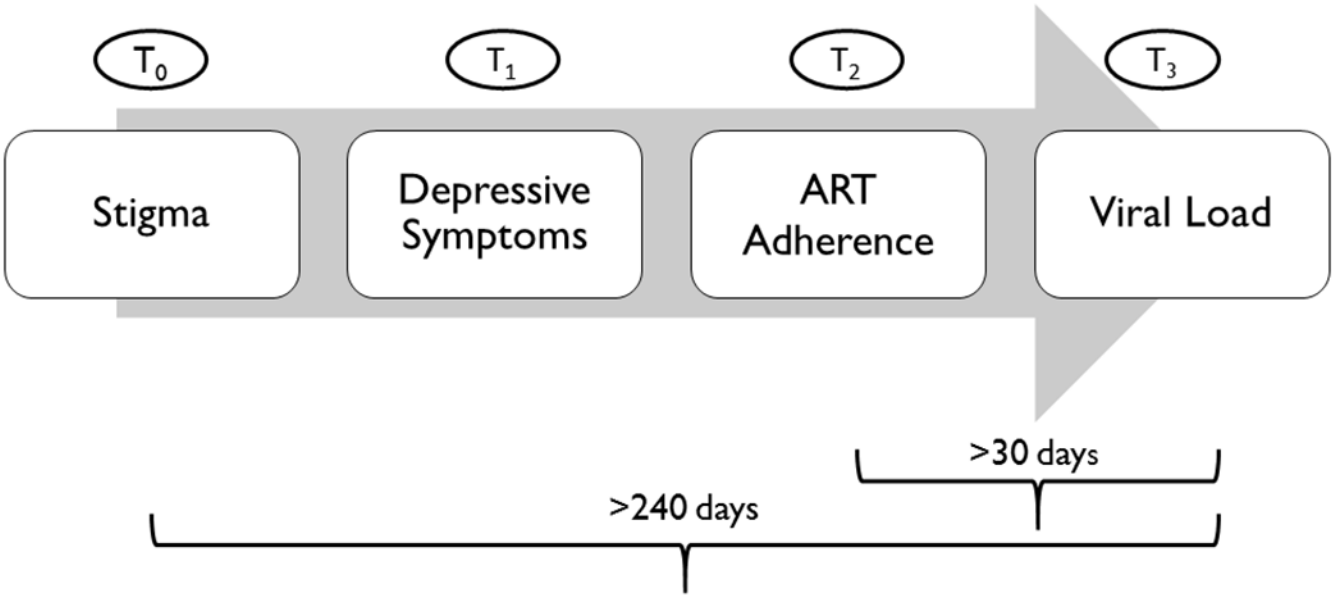

Figure 1.
Measurement Time Points for Internalized HIV Stigma, Depressive Symptoms, Antiretroviral Adherence and Viral Load

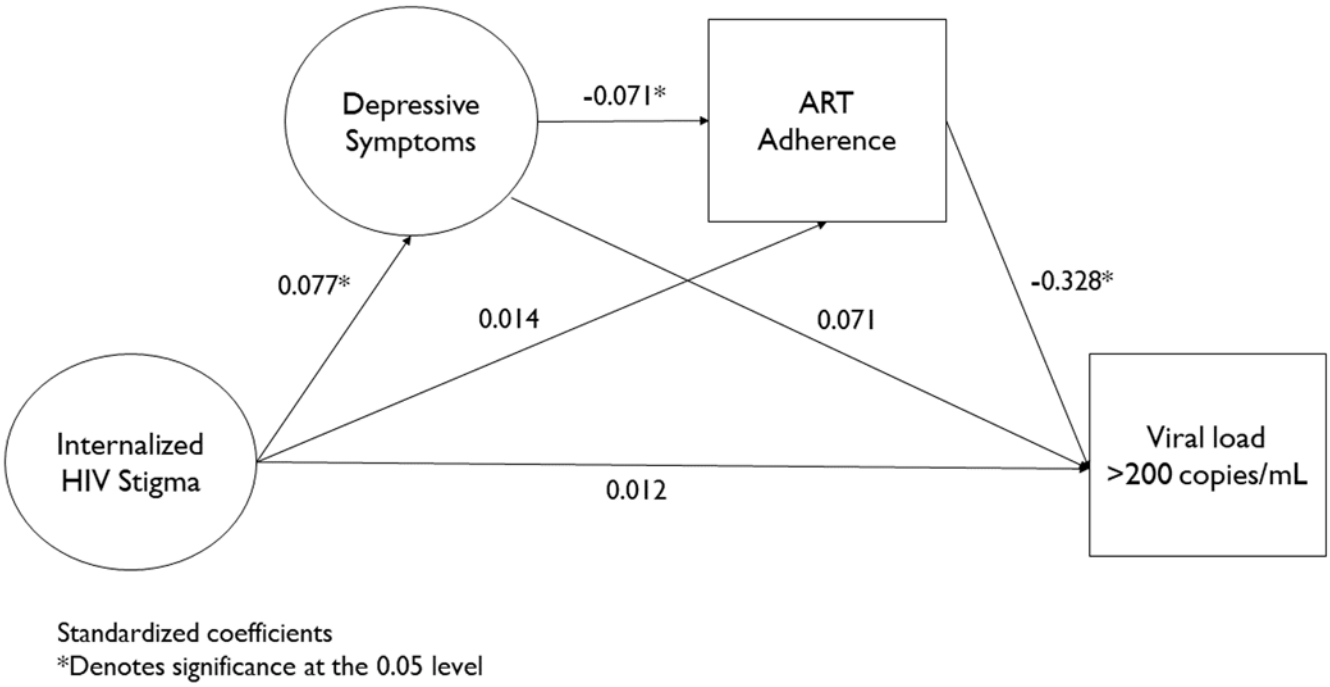

Figure 2.

Longitudinal Sequential Path Model of the Association between Internalized HIV Stigma and Unsuppressed Viral Load through the Mediators of Depressive Symptoms and Antiretroviral Adherence

Table 1.

Demographic and Clinical Characteristics of Patients Undergoing Stigma Assessment in Routine HIV Care in Six Clinics across the United States (N=6,589)

Characteristic	N (%)
Site	
UCSD	1880 (28.5)
UAB	1975 (30.0)
UW	793 (12.0)
FENWAY	613 (9.3)
JH	884 (13.4)
UCSF	444 (6.7)
Age at Index	
19–29 years	527 (8.0)
30–39 years	1213 (18.4)
40–49 years	1651 (25.1)
50 or more years	3198 (48.5)
Current gender	
Cis-male	5303 (80.5)
Cis-female	1112 (16.9)
Gender minority	174 (2.6)
Race	
Black	2474 (37.8)
White	2717 (41.5)
Latinx	1048 (16.0)
Other	304 (4.7)
Heterosexual orientation	2040 (31.6)
ART use at baseline	5812 (92.5)
Optimal adherence at baseline	1,776 (31.9)
Baseline CD4, median (IQR) *	573 (375, 806)
Detectable viral load (>200 copies/mL) †	805 (13.3)
Years in CNICS, median (IQR)	6.3 (2.7, 11.4)
PHQ-9 score at baseline, median (IQR)	3.38 (0, 8.0)
Stigma at baseline, mean (SD)	2.00 (1.08)

Note:

* CD4 closest to stigma assessment from 180 days before to 90 days after, available N=3,746

† Viral load closest to stigma assessment +/− 90 day window, available N=6,061

Other available Ns as follows: N=6,544 for race/ethnicity; N=6,458 for sexual identity; N=5,560 for optimal adherence based on N=5,812 for ART use at baseline; N=6,315 for baseline PHQ-9 score

Table 2.

Association between Internalized HIV Stigma and Unsuppressed Viral Load for Patients Undergoing Stigma Assessment in Six Clinics across the United States (N = 6,589)

Characteristic	Adjusted Odds Ratio (95% CI)	*p*
Stigma	1.16 (1.05–1.28)	0.004
Age, years		
18-29	2.41 (1.49–3.89)	<0.001
30-39	2.11 (1.49–3.01)	<0.001
40-49	2.04 (1.53–2.73)	<0.001
≥50	Reference	--
Gender Identity		
Cis-female	1.29 (0.93–1.80)	0.13
Gender minority	0.72 (0.30–1.74)	0.47
Cis-male	Reference	--
Race/Ethnicity		
Black	1.84 (1.35–2.50)	<0.001
Latinx	1.19 (0.77–1.83)	0.44
Other	1.25 (0.65–2.43)	0.50
White	Reference	--
Sexual Identity		
Heterosexual	1.14 (0.83–1.57)	0.42
Sexual Minority	Reference	--
Years in CNICS	1.01 (0.99–1.03)	0.52

Odds ratios are adjusted for CNICS site but results not shown for clarity.

Wald test for overall age effect: $\chi^2(3) = 29.94$, $p < .0001$

Wald test for overall race/ethnicity effect: $\chi^2(3) = 14.90$, $p = .002$

Wald test for overall gender effect: $\chi^2(2) = 3.16$, $p = .21$.