Title

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Social inequalities contribute to racial/ethnic disparities in depressive symptomology among men who have sex with men

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Abstract

Purpose—Racial/ethnic minorities experience disproportionate rates of depressive symptoms in the United States. The magnitude that underlying factors-such as social inequalities-contribute to these symptoms is unknown. We sought to identify exposures that explain racial/ethnic differences in clinically significant depressive symptomology among men who have sex with men (MSM).

Methods—Data from the Multicenter AIDS Cohort Study (MACS), a prospective cohort study, were used to examine clinically significant symptoms of depression (Center for Epidemiologic Studies Depression Scale score   20) among non-Latinx White, non-Latinx Black, and Latinx MSM. We included 44,823 person-visits by 1,729 MSM seen in the study sites of Baltimore/ Washington, DC; Chicago; Pittsburgh/Columbus; and Los Angeles from 2000–2017. Regression models estimated the percentage of depressive symptom risk explained by social, treatment, and health-related variables related to race/ethnicity. Machine-learning methods were used to predict the impact of mitigating differences in determinants of depressive symptoms by race/ethnicity.

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Results—At the most recent non-missing MACS visit, 16% of non-Latinx White MSM reported clinically significant depressive symptoms, compared to 22% of non-Latinx Black and 25% of Latinx men. We found that income and social-environmental stress were the largest contributors to racial/ethnic disparities in risk for depressive symptoms. Similarly, setting the prevalence of these two exposures to be equal across racial/ethnic groups was estimated to be most effective at reducing levels of clinically significant depressive symptoms.

Conclusion—Results suggested that reducing socioeconomic inequalities and stressful experiences may be effective public health targets to decrease racial/ethnic disparities in depressive symptoms among MSM.

INTRODUCTION

Depression is one of the leading causes of disability in the United States [1, 2]. Racial/ethnic minority populations in the United States experience a larger burden of depression than their non-Latinx White peers [3]. Among older adults (55–65 years), the odds of depression are 16% higher for Black and 44% higher for Latinx compared with White individuals [4]. Striking racial/ethnic disparities also exist in the diagnosis and treatment of depression [5–8]. One study found that physicians were less likely to detect depression in non-Latinx Black and Latinx (58% and 71% less likely, respectively) than in non-Latinx White individuals, despite controlling for previous depression diagnoses [5]. Similarly, non-Latinx White people living with HIV were more likely to receive treatment with antidepressants compared with non-Latinx Black and Latinx peers living with HIV [8].

A number of potential explanations for racial/ethnic disparities in depressive symptoms have been posited. These include underlying racial/ethnic differences in low self-esteem [9], undertreatment with antidepressants [8], absence of health insurance coverage and poor access to care [4], lifestyle factors such as smoking and exercise [4], socioeconomic inequalities and negative stereotypes [10], and discrimination [10–12]. A systematic review of racial/ethnic discrimination and health [13] found that discrimination was associated with a range of mental health outcomes, including depression. Effectively intervening on racial/ethnic disparities in depressive symptoms requires a clear understanding of the factors that drive differences in depressive symptom risk.

Men who have sex with men (MSM) are more likely to experience depressive symptoms compared to their heterosexual counterparts [14]. The age-adjusted prevalence of a lifetime major depressive episode is far higher in MSM (18.7%) than in heterosexual men (8.0%) [15]. Risk factors for depression, including drug use, HIV infection, stigma, and poor health status, are more prevalent in MSM populations.

Minority stress theory posits that individuals who identify as minorities are more likely to experience discrimination, which increases the risk of stress. These elevated stress levels can result in negative health outcomes, including depression, which may help to explain the

higher likelihood of depressive symptoms among MSM as compared to heterosexual men, or among racial/ethnic minorities as compared to Whites [16]. Minority stress theory, however, does not fully take into account the experiences of individuals who represent multiple minorities. These people may be subject to stress from various sources of discrimination, or may develop resilience from their experience within multiply marginalized groups [16, 17]. In order to reconcile this shortcoming, the minority stress theory has been considered in conjunction with the theory of intersectionality [16, 17]. According to the theory of intersectionality, individuals' experiences occur at the intersection of their multiple social identities [18]. MSM of different racial/ethnic minority groups may thus experience discrimination and stress both from their sexual and racial/ethnic minority status [16], and these may synergistically increase risk for depressive symptoms beyond that of individuals with only one of these social identities.

Additionally, racial/ethnic disparities have been well documented in the timing of highly active antiretroviral therapy (HAART) initiation and aggressiveness of HIV treatment [19]. Higher risk for depressive symptoms among racial/ethnic minority MSM may also occur at the intersection of HIV-related and psychosocial factors. Distrust in HIV care providers by people living with HIV could lead to reduced HAART adherence and poorer control of HIV infection [20], thereby exacerbating depressive symptoms.

To our knowledge, few prior studies have focused on racial/ethnic differences in depressive symptoms specifically among MSM [21]. Friedman et al. (2014) reported that Latinx MSM had a higher likelihood of depressive symptoms than White MSM [22]. A study of long-term patterns of depression in MSM and women living with HIV found that Black MSM had greater odds of moderate risk for depression [23]. This study, however, did not explore contributing factors to racial/ethnic differences in depressive symptoms.

Identifying the primary drivers of depressive symptom disparities by race/ethnicity can highlight the ways in which these risk factors for depression contribute to the overall burden of depressive symptoms. In this study, we aimed to quantify racial/ethnic disparities in clinically significant depressive symptoms among MSM and examine social, treatment, and health-related factors that account for these differences. We further simulated the impact of interventions on different risk factors to determine the most effective targets to reduce racial/ethnic disparities in clinically significant depressive symptoms.

METHODS

Study sample and design

The Multicenter AIDS Cohort Study (MACS) is a prospective cohort study of primarily MSM who are either seropositive for HIV type 1 or at risk for HIV infection. This ongoing study began recruiting across centers in Baltimore, Maryland/Washington, DC; Chicago, Illinois; Pittsburgh, Pennsylvania/Columbus, Ohio; and Los Angeles, California in 1984, with subsequent periods of open enrollment. Data collection from semiannual visits includes standardized questionnaires, physical examinations, and biospecimen collection. The MACS parent study design, inclusion criteria, and enrollment has been previously described in detail [24, 25]. Additional inclusion criteria described below were specific to our study.

We restricted the study population to non-Latinx White, non-Latinx Black, and Latinx MSM, defined by self-report. The MACS collects data on race/ethnicity according to the 2015 National Institutes of Health definition [26]. We selected these racial/ethnic categories because they represent the most common races/ethnicities in the MACS and U.S. population [27]. We additionally limited inclusion to men who had completed a subset of MACS questions that captured information on social-environmental stress and social mobility (Long Term Health Effects of Methamphetamine use in the MACS; R01DA022936). These questions were administered during MACS visits 49 and 50 (April 1, 2008-March 31, 2009) and were offered to all participants seen at any of the four study centers during this time period. Variables utilized from the substudy recorded past experiences and were treated as time-constant in our analyses. We included only person-visits with Center for Epidemiologic Studies Depression Scale (CES-D) scores recorded after the year 2000 to capture current trends in depressive symptoms; this time period also corresponds to the modern era for HIV therapy. We used all data that met inclusion criteria and that were collected during the MACS study visits through September 30, 2017.

Assessment of depressive symptoms

We defined clinically significant depressive symptoms as a CES-D score greater than or equal to 20 at a person-visit. CES-D scores greater than or equal to 16 have historically been shown to correspond to clinically significant depressive symptoms [28, 29]. A study that examined CES-D scores among MACS participants found that a cutoff of 20 had a higher specificity relative to a cutoff of 16 [30].

Determinants of depressive symptoms

We selected determinants of depressive symptoms based on prior literature and expert opinion, and we included important social, treatment, and health exposures. Social exposures included baseline education, MACS site, illicit drug use, self-reported income, health insurance coverage, social-environmental stress from sources considered to be strongly related to race/ethnicity (defined as the neighborhood, crime, racism, or police ever having caused stress), sum of parental educational attainment, and social mobility. Baseline educational attainment was categorized as high school or less, some college, college graduate, and at least some postgraduate education. MACS site, to account for unmeasured sociodemographic factors that may vary by location, was categorized into Baltimore, Maryland/Washington, DC; Chicago, Illinois; Pittsburgh, Pennsylvania/Columbus, Ohio; and Los Angeles, California. An individual was classified as having taken illicit drugs since the last MACS visit if he had at least weekly use of poppers, crack, methamphetamine, cocaine, heroin, speedball, and/or ecstasy or used two or more of the above listed drugs. Self-reported information on annual income was collected at each MACS visit and categorized as less than $20,000; $20,000 to $39,999; $40,000 to $59,999; and $60,000 or more. Possession of health insurance was coded as binary. Parental educational attainment was calculated by summing a participant's father's and mother's highest level of education while the participant was in high school. Social mobility was calculated by first averaging the highest level of education of a participant's father and mother when the participant was in high school, and then subtracting this value from the participant's education level reported at his baseline visit.

The MACS has had multiple recruitment waves with changing demographic characteristics. Thus, we also explored recruitment cohort as it represents other unmeasured social factors. Recruitment cohort was categorized based on the MACS recruitment waves captured in this study: 1984, 1987, and 2002.

We considered antidepressant medication use as a treatment variable, and this was coded as binary. Health-related factors included family history of depression, burden of physical comorbidities, recent episodes of insomnia, and HIV serostatus. Family history of depression was coded as binary, with a positive value assigned if a relative had ever been diagnosed as having depression. Relatives were defined as the biological mother, father, brothers, and sisters. Burden of physical comorbidities reported was categorized as zero, one, and two or more comorbidities. Six possible comorbid conditions were included: hepatitis C infection, high blood pressure, diabetes, dyslipidemia, kidney disease, and cancer. Recent episodes of insomnia were coded as binary, with a positive response indicating experience with insomnia or problems sleeping since the last MACS visit. HIV serostatus was treated as binary, and coded as seropositive or seronegative. We drew data on these factors from the same visit as the depressive symptom assessment.

Because HIV infection and treatment may impact both depressive symptom risk and social/health factors, we examined a subset of men living with HIV. Among this group, we considered HIV treatment factors, including adherence to HAART and reported use of HAART at a MACS visit. Adherence to HAART since the last MACS visit was defined as less than 75%, 75% to 94%, 95% to 99%, and 100% adherence. Reported use of HAART at a MACS visit was treated as binary. HIV-related health factors included CD4 cell count at HAART initiation and viral copy-years following HAART initiation. CD4 cell count at HAART initiation was defined as the number of CD4 cells an individual had when he initiated HAART. Viral copy-years following HAART initiation served as a cumulative measure of viral load after initiating HAART.

More detailed definitions of examined factors are described in the Appendix. Once these variables were defined and created, we omitted missing values in analyses relevant to the variables. Using values drawn from an individual's most recent non-missing MACS visit, we compared variables between racial/ethnic groups using χ^2 tests or Fisher exact tests when sample sizes were small. We compared the medians of continuous variables with the Kruskal-Wallis test.

Statistical analysis

Factors that account for racial/ethnic disparities in depressive symptom risk.

—We modeled the probability of reporting clinically significant depressive symptoms using logbinomial regression models. Comparing nested models, we estimated the partial coefficient of determination (partial R^2). Due to difficulties in estimating the partial R^2 in repeated-measures models, in this analysis observations from the same individual were treated as uncorrelated. Multiple approaches were explored for estimating the partial R^2 and conclusions were similar across all methods. The partial R^2 value assessed the racial/ethnic disparity in the risk for clinically significant depressive symptoms remaining after accounting for other determinants of depressive symptoms. Models were adjusted for age.

Smaller partial R^2 values suggested that race/ethnicity did not explain much additional variation in the risk for clinically significant depressive symptoms beyond what was already accounted for by a particular social, treatment, or health determinant. In other words, a small partial R^2 value indicated the importance of a social, treatment, or health factor in determining racial/ethnic disparities in clinically significant depressive symptom risk.

We also estimated the partial R^2 value for each social, treatment, and health factor, after adjusting for age and race/ethnicity. This allowed us to quantify the amount of clinically significant depressive symptom variation each exposure explained after removing the effects of age and race/ethnicity. This estimate provided an indication of the strength of the association between each exposure and clinically significant depressive symptoms.

Impact of intervening on determinants of depressive symptoms.—Many factors likely contribute to racial/ethnic disparities in depressive symptoms but may not be important targets for intervention due to low prevalence or weak associations. We built classification trees with a supervised machine-learning algorithm [31] to predict the probability of reporting clinically significant depressive symptoms at a MACS visit. We made predictions within each racial/ethnic group as a function of various determinants of depressive symptomology. For each determinant, we simulated effects of changes in a variety of public health intervention targets by setting values across racial/ethnic groups to be equal, using the most favorable distribution observed. This allowed us to assess the probable impact of mitigating different depressive symptom determinants on racial/ethnic disparities. For example, if the sample size were 100 men per race/ethnicity group and the distribution of illicit drug use was 30% for non-Latinx White and non-Latinx Black and 15% for Latinx men, we would randomly select 15 non-Latinx White and 15 non-Latinx Black illicit drug users to have their illicit drug use status changed to nonusers. This would set the prevalence of illicit drug use to be 15% across all races/ethnicities. Then we would predict the risk for clinically significant depressive symptoms. We repeated this analysis among the subset of people living with HIV to assess the contribution of HIV-specific exposures.

All analyses were performed in SAS version 9.4 [32]. Two-sided P values less than 0.05 were considered statistically significant.

RESULTS

The study population consisted of 1,729 MSM (contributing 44,823 person-visits), out of a total of 1,842 MACS men who had completed the subset of questions capturing social-environmental stress and social mobility. Exclusions leading to this reduced sample size included 22 men of Other race/ethnicity, 71 men without a history of sex with men, and 20 men who were not followed up after the year 2000. Table 1 displays characteristics of the study population at participants' most recent non-missing MACS visit, stratified by race/ethnicity.

There was a statistically significant racial/ethnic difference in reporting clinically significant depressive symptoms at the most recent MACS visit. Twenty-five percent of Latinx participants reported a CES-D score greater than or equal to 20 compared with 22% of non-

Latinx Black and 16% of non-Latinx White participants ($P < 0.01$). A statistically significant difference between the racial/ethnic groups existed for all social exposure variables. A larger proportion of non-Latinx White participants reported greater baseline educational attainment, annual income, and possession of health insurance. Non-Latinx White participants also had a higher median age and summed parental educational attainment at the most recent MACS visit compared with non-Latinx Black and Latinx participants. A greater percentage of non-Latinx Black participants reported high levels of social-environmental stress (21%) relative to Latinx (19%) and non-Latinx White (6%) participants ($P < 0.01$) (Table 1).

There were racial/ethnic differences in health and disease variables. Non-Latinx White participants (20%) were most likely to report antidepressant medication use at their most recent MACS visit ($P < 0.01$). Latinx participants had the lowest rates of antidepressant medication use (8%) (Table 1).

Non-Latinx Black participants had the lowest prevalence of a family history of depression and insomnia. The comorbidity burden was lowest among Latinx participants compared with non-Latinx Black and non-Latinx White participants ($P < 0.01$). At the most recent non-missing MACS visit, there was a lower proportion of non-Latinx White participants living with HIV (41%) compared with non-Latinx Black (68%) and Latinx (65%) participants ($P < 0.01$) (Table 1).

Factors that account for racial/ethnic disparities in depressive symptom risk

Table 2 presents the percentage of clinically significant depressive symptom variation explained by race/ethnicity after adjusting for age and other social, treatment, and health exposures. When accounting only for age, race/ethnicity explained 0.9% of the remaining variation in clinically significant depressive symptoms. The largest contributors to racial/ethnic disparities in clinically significant depressive symptoms were social factors. These factors were the only exposures that reduced the percentage of depressive symptom variation explained by race/ethnicity to less than 0.8. Self-reported annual income and social-environmental stress contributed the most to differences in the prevalence of clinically significant depressive symptoms by race/ethnicity (Table 2). When income and social-environmental stress were both included in the regression model, race/ethnicity explained only 0.3% of the remaining variation in clinically significant depressive symptomology. Parental educational attainment and insomnia experience were the two exposures that explained the most variation in clinically significant depressive symptomology after accounting for age and race/ethnicity.

Among individuals living with HIV, the results were similar. Race/ethnicity explained 0.6% of the remaining variation in clinically significant depressive symptoms after accounting for age. Social exposures explained far more of the racial/ethnic disparity in the prevalence of clinically significant depressive symptoms than HIV infection-related variables. After controlling for age and race/ethnicity, the most variation in clinically significant depressive symptoms was explained by insomnia experience and HAART adherence.

Impact of intervening on determinants of depressive symptoms

Table 3 displays the predicted probability of reporting clinically significant depressive symptoms at a MACS visit, stratified by race/ethnicity. The greatest reduction in racial/ethnic disparities in the risk for clinically significant depressive symptoms resulted from setting income and social-environmental stress levels to be equivalent across racial/ethnic groups. If all participants had income levels similar to non-Latinx White participants, the prevalence of clinically significant depressive symptoms was estimated to decline by an absolute difference of 1.1% among non-Latinx Black and 6.2% among Latinx participants. Similarly, setting social-environmental stress levels to reflect those of non-Latinx White participants reduced the prevalence of clinically significant depressive symptoms by an estimated 3.2% among non-Latinx Black and 3.3% among Latinx participants.

At the most recent non-missing MACS visit, all races/ethnicities demonstrated an association between higher income and lower prevalence of clinically significant depressive symptoms (Figure 1), and between social-environmental stress experience and an elevated likelihood of reporting clinically significant depressive symptoms (Figure 2). This lends credence to the intervention simulation, which indicated that increasing income and decreasing stress experiences would reduce clinically significant depressive symptom prevalence. Setting both income and social-environment stress levels as equivalent across racial/ethnic groups resulted in an absolute decrease of clinically significant depressive symptom prevalence estimated at 6.3% among non-Latinx Black and 11.5% among Latinx participants.

The reductions in clinically significant depressive symptom prevalence observed for income and stress were larger than those estimated for health- and treatment-related variables: If levels of insomnia were set to those of non-Latinx Black participants, clinically significant depressive symptom prevalence was estimated to decline by only 0.2% among non-Latinx White and 1.7% among Latinx participants.

Results were similar among men living with HIV. Increasing adherence to HAART to levels observed in non-Latinx White people living with HIV reduced clinically significant depressive symptom prevalence by an estimated 0.2% among non-Latinx Black MSM living with HIV but made no difference among Latinx participants living with HIV (Table 3).

We conducted sensitivity analyses using a clinically significant depressive symptom definition of a CES-D score greater than or equal to 16 at a person-visit, as this is a threshold commonly used in the literature. Findings were comparable (results available from authors upon request).

DISCUSSION

To our knowledge, this is the first study that has attempted to quantify the amount to which underlying exposures are associated with racial/ethnic disparities in clinically significant depressive symptoms among MSM. Our findings identify clear disparities in clinically significant depressive symptoms within the MACS and highlight the degree to which other social and health service factors likely beget those differences. Social factors contributed far

more to racial/ethnic disparities in symptoms of depression than other health or disease factors examined. Self-reported annual income and social-environmental stress had the greatest impact on reducing the variance in clinically significant depressive symptoms associated with race/ethnicity. Manipulating the distribution of these variables also decreased clinically significant depressive symptom prevalence by the largest amount.

Income and social-environmental stress are patterned by underlying discrimination and institutional forms of racism, such as residential segregation [33, 34]. Areas with a high concentration of racial/ethnic minorities are frequently economically disadvantaged, with reduced access to key financial resources [33, 35]. Additionally, racial/ethnic minorities are disproportionately exposed to social-environmental stressors, such as violent crime [36–38], police violence [39, 40], and grossly inequitable rates of incarceration [41, 42]-the War on Drugs and ethnic profiling of Latinx people being two demonstrable examples [34, 43]. Prior studies have linked low socioeconomic status and elevated stress levels to depressive outcomes [44–47]. Racial/ethnic minorities experiencing these socioenvironmental conditions at an elevated rate may help to explain observed disparities in clinically significant depressive symptomology.

Limitations

Although this study had many strengths, including its use of a large and robust dataset of health outcomes among MSM, it was not without limitations. Exposure data collected through questionnaires were retrospectively recalled by participants (rather than collected in real-time), which may have influenced accuracy. This could bias findings towards or away from the null, depending on the nature of the relationships between exposure recall, race/ethnicity, and depressive symptoms [48, 49]. Additionally, some of the questions composing the CES-D may operate differently within distinct racial/ethnic groups [50]. Perreira et al. (2005) found that the full CES-D factor structure varied by race/ethnicity and immigrant generation in adolescents, and they proposed using a five-item CES-D for this study population [51]. Similarly, older Black adults were more likely to report interpersonal problems in the CES-D despite similar depressive symptomology [52]. Any CES-D measurement variance by race/ethnicity in the MACS could have introduced bias into this study. Finally, in this study all men with a self-identified Latinx ethnicity were grouped into one single category, as small sample size in this group prevented further stratification. Analyzing all Latinxs in a single ethnic category may have masked important distinctions in depressive symptom risk between Latinxs of different races, or among unique Latinx subgroups. Despite these limitations, our findings have important implications for public health policy and disparities in depressive symptoms warrant further study.

Recommendations for interventions to reduce disparities in depressive symptoms

Short-term public health interventions.—Health-promoting actions can be tailored to address the needs of specific racial/ethnic groups [53, 54]. Additionally, case management can help patients to navigate the health care system. Both approaches have shown to be particularly effective at reducing racial/ethnic disparities in depressive symptoms [53]. Criminal justice reform is also necessary to decrease social-environmental stressors for racial/ethnic minorities. A recent study demonstrated that police killings of unarmed Black

Americans negatively impacts the mental health of Black individuals, but not White individuals [55]. Other interventions have focused on teaching racial/ethnic minorities coping strategies to counter experiences with discrimination, though these strategies do not address the fundamental problem of racism and should be considered a temporary solution, not a primary public health goal. A culturally-tailored pilot program that trained Black MSM living with HIV to cope with discrimination showed increased use of several positive coping strategies among individuals who were trained [56].

Long-term public health interventions.—Intervention strategies should focus on social factors among racial/ethnic minorities, such as increasing income and reducing social-environmental stress. To do so, we must address structural racism. Structural racism is commonly understood as historical and contemporary societal-level imbalances in power and opportunity among racial/ethnic groups that act to form and strengthen discriminatory beliefs and actions [34, 57]. One recommended approach to confront structural racism involves a three-stage process of (1) acknowledging the presence of racism in societal structures, (2) understanding how it is operating, and then (3) developing a community-specific plan to address the identified issues [58]. Redevelopment and investment in historically disadvantaged neighborhoods is another approach, with redevelopment efforts tailored to the specific location [34]. A 2010 study demonstrated the efficacy of investing in underprivileged areas and people. In this work, homeless/unstably housed persons living with HIV, the majority of whom were male racial/ethnic minorities, were provided housing assistance [59]. Six months after receiving this assistance, individuals reported fewer depressive symptoms and lower stress levels [59]. Structural racism can also be incorporated into educational curriculums-particularly in medical and public health schools-where it is currently given little to no attention [34, 60].

Directions for future research.—We must better understand the association between racism and mental health, such as how racist experiences translate into health outcomes [10]. A relatively recent avenue of research has focused on the impact of racism on stress and the corresponding effect on allostatic load [61, 62]. Allostatic load, the overstimulation of the body's stress response, has negative health consequences [63], including altered brain function and development of depression [62]. Researchers should continue to investigate the sociobiological connection between racism, allostatic load, and mental health, especially in the context of intersectionality. Additionally, further research is needed to understand how evidence-based psychological treatments for depression-such as Cognitive Behavioral Therapy and Dialectic Behavior Therapy-can be used in conjunction with efforts to address social barriers disproportionately experienced by racial/ethnic minorities (e.g. violent crime, racism and discrimination, and police violence).

In this study, we showed that social exposures have strong associations with observed racial/ethnic disparities in clinically significant depressive symptoms among MSM. This suggests that efforts to combat these disparities should focus on addressing social factors. We identified income and social-environmental stress as two particularly important targets for intervention. Such actions are critically needed in the United States at a time where discrimination, xenophobia, and blatant racism are openly used as tools for political gain

[64, 65]. Efforts are particularly vital among populations existing at the intersection of multiply marginalized groups, such as racial/ethnic minority MSM living with HIV. While confronting racism deeply entrenched in U.S. society is certainly an uphill battle, it is a worthy-and in fact, necessary-cause among public health practitioners if equity (health and otherwise) for all is ever to be achieved.

ACKNOWLEDGMENTS

APPENDIX

Baseline educational attainment was categorized as high school or less, some college, college graduate, and at least some postgraduate education. Categories were modeled after [66]. Recruitment center location was categorized into the four active Multicenter AIDS Cohort Study (MACS) sites: Baltimore, Maryland/Washington, DC; Chicago, Illinois; Pittsburgh, Pennsylvania/Columbus, Ohio; and Los Angeles, California. Recruitment cohort was coded into categories based on the MACS recruitment waves captured in this study: 1984, 1987, and 2002. The latest MACS recruitment wave, initiated in 2010, was not included in this study because the social-environmental stress and social mobility MACS questions were asked before the 2010 recruitment began and individuals who did not respond to the social-environmental stress and social mobility questions were excluded.

Illicit drug use since the last MACS visit was coded as binary, with an individual classified as having taken illicit drugs if they had at least weekly use of poppers, crack,

methamphetamine, cocaine, heroin, speedball, and/or ecstasy since the last MACS visit or used two or more of the above listed drugs since the last MACS visit. This definition came from [66].

Self-reported annual income at a MACS visit was categorized as less than $20,000; $20,000 to $39,999; $40,000 to $59,999; and $60,000 or more, with categories adapted from [66]. Nearest-neighbor interpolation was used to fill in missing incomes with values within six months of the missing data. Possession of health insurance during a MACS visit was coded as binary. Social-environmental stress was treated as binary and adapted from the Urban Life Stress Scale [67]. A participant was coded as positive for stress if they reported that the neighborhood environment, crime/violence, racism/discrimination, or police relations had ever caused a lot or extreme stress.

Sum parental educational attainment was calculated by summing a participant's father's and mother's highest level of education while the participant was in high school. The participant's father's and mother's education levels were coded as 0 = high school or less; 1 = junior college or trade school; 2 = college graduate; 3 = postgraduate education; and missing = missing visit, no father/mother while participant was in high school, participant does not know, or participant refused to answer. Therefore, the generated sum parental educational attainment variable ranged from zero to six.

Social mobility served as a proxy for the social opportunities taken advantage of by a participant and was calculated by first averaging the highest level of education of a participant's father and mother while the participant was in high school, and then subtracting this value from the participant's education level reported at their baseline visit. Because both the average of the highest level of education of a participant's father and mother and a participant's baseline education ranged from zero to three, the generated social mobility variable ranged from negative three to three, with negative numbers indicating reduced mobility and positive numbers indicating increased mobility.

Use of antidepressant medication at a MACS visit was coded as binary. Adherence to highly active antiretroviral therapy (HAART) since last MACS visit among persons living with HIV was defined as less than 75%, 75% to 94%, 95% to 99%, and 100% adherence, as determined through self-report. This definition was adapted from [68]. Reported use of HAART by an individual living with HIV at a MACS visit was treated as binary, with any type of HAART (protease inhibitors, integrase inhibitors, etc.) being coded as positive.

Family history of depression, a non-modifiable risk factor for depression [69], was coded as binary, with a positive value being assigned if a participant reported that a relative had ever been diagnosed as having depression. Relatives were defined as the biological mother, father, brothers, and sisters.

Burden of comorbidities reported at a MACS visit was categorized as zero comorbidities, one comorbidity, and two or more comorbidities. Six possible comorbid conditions were included: hepatitis C infection (defined as detectable hepatitis C RNA in serum); high blood pressure (defined as systolic pressure > 140 mmHg or diastolic pressure > 90 mmHg); diabetes (defined as fasting glucose 126 mg/dL or a self-report of previous clinical

diabetes diagnosis with use of medication); dyslipidemia (defined as either a fasting total cholesterol 200 mg/dL, low-density lipoprotein 130 mg/dL, high-density lipoprotein < 40 mg/dL, triglycerides 150 mg/dL, or use of lipid-lowering medications with self-report of a previous clinical diagnosis); kidney disease (defined as an estimated glomerular filtration rate < 60 mL/min/1.73 m^2 body surface area or a urine protein-to-creatinine ratio 200 mg protein/1 g creatinine); or cancer (defined as diagnosis of any cancer type at, or within a year of, the MACS study visit). These comorbid conditions were selected and defined in the study [70].

Insomnia, a risk factor for depression [71], was coded as binary. An individual who reported insomnia or problems sleeping since the last MACS visit was treated as a positive response. HIV serostatus was treated as binary, with an individual being coded as seropositive or seronegative at a MACS visit. HIV serostatus was determined through enzyme-linked immunosorbent assay (ELISA), confirmed by Western blot [24].

CD4 cell count at HAART initiation captured the number of CD4 cells, as determined by flow cytometry, an individual living with HIV had when they initiated HAART (or the earliest MACS visit post-HAART initiation). Viral copy-years following HAART initiation served as a cumulative measure of a person living with HIV's viral load after they began taking HAART. Viral load was measured via Roche assays. The definition and equation of viral copy-years came from the study [72], and the variable is conceptually similar to pack-years for smoking. Before calculation of viral copy-years, missing viral load values were filled in using multiple imputation with the Markov chain Monte Carlo method [73]. Age, current CD4 cell count, and HAART treatment history at a MACS visit were used as predictor variables.

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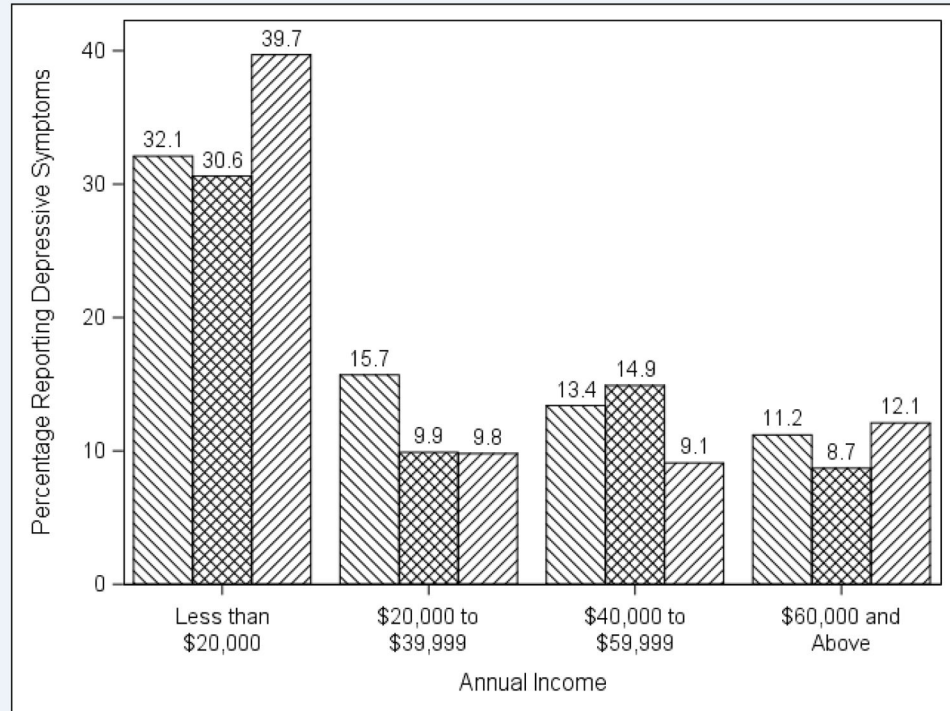
Fig. 1.
Percentage of 1,729 men who have sex with men reporting clinically significant depressive symptoms (Center for Epidemiologic Studies Depression Scale score 20) at the most recent non-missing Multicenter AIDS Cohort Study visit (2000–2017), by race/ethnicity and self-reported annual income. Downward hash represents non-Latinx White men. Cross hash represents non-Latinx Black men. Upward hash represents Latinx men. There is an observable trend in decreasing clinically significant depressive symptom prevalence as annual income increases within all races/ethnicities. The prevalence of clinically significant depressive symptoms is notably high in the lowest annual income category of less than $20,000.
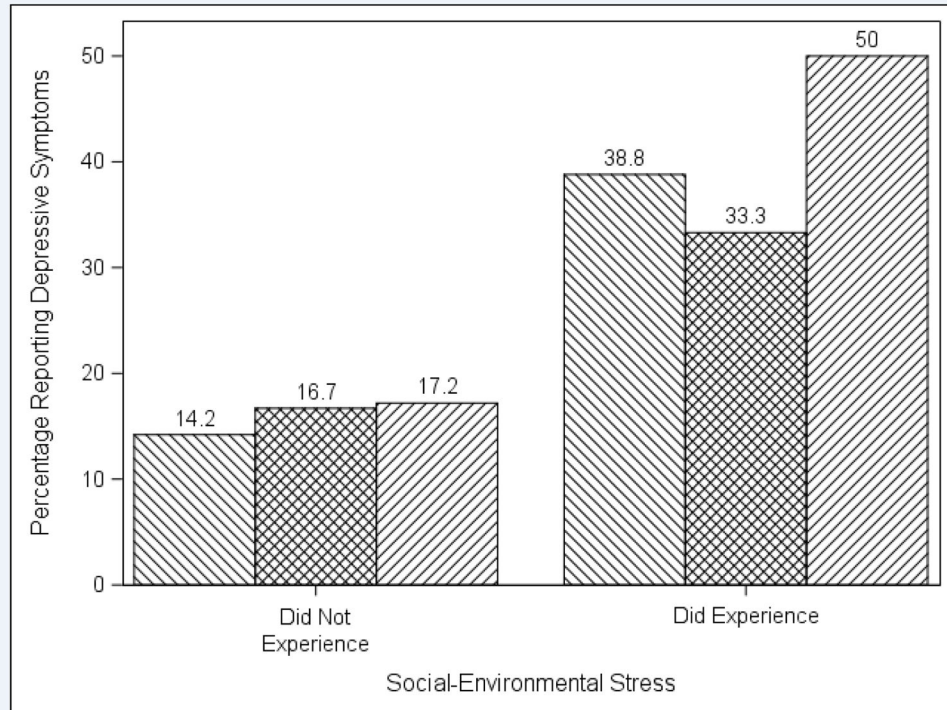
Fig. 2.
Percentage of 1,729 men who have sex with men reporting clinically significant depressive symptoms (Center for Epidemiologic Studies Depression Scale score 20) at the most recent non-missing Multicenter AIDS Cohort Study visit (2000–2017), by race/ethnicity and social-environmental stress experience. Downward hash represents non-Latinx White men. Cross hash represents non-Latinx Black men. Upward hash represents Latinx men. There is a significant increase in the likelihood of reporting clinically significant depressive symptoms between those who have never experienced social-environmental stress and those who have. This relationship is constant across all races/ethnicities.

Table 1.

Characteristics of 1,729 MSM[a] at the Most Recent Non-Missing MACS Visit, 2000–2017[b].

Most recent characteristic	Non-Latinx White	Non-Latinx Black	Latinx	P value
	Column% (N participants[c])			
Outcome				
Clinically significant depressive symptoms (CES-D 20)				<0.01
No	84 (994)	78 (285)	75 (133)	
Yes	16 (193)	22 (79)	25 (45)	
Social				
Baseline educational attainment				<0.01
High school or less	9 (110)	34 (125)	35 (62)	
Some college	25 (293)	35 (128)	32 (57)	
College graduate	26 (303)	15 (55)	17 (30)	
At least some postgraduate education	41 (481)	15 (56)	16 (29)	
Recruitment center location				<0.01
Baltimore, MD/Washington, DC	26 (306)	28 (103)	11 (19)	
Chicago, IL	15 (177)	38 (138)	15 (27)	
Pittsburgh, PA/Columbus, OH	32 (381)	15 (55)	5 (9)	
Los Angeles, CA	27 (323)	19 (68)	69 (123)	
Recruitment cohort				<0.01
1984	76 (903)	9 (31)	20 (36)	
1987	4 (47)	17 (61)	4 (7)	
2002	20 (237)	75 (272)	76 (135)	
Illicit drug use since last visit				0.03
No	90 (1068)	86 (312)	85 (152)	
Yes	10 (119)	14 (52)	15 (26)	
Self-reported annual income				<0.01
Less than $20,000	17 (187)	53 (186)	41 (68)	
$20,000 to $39,999	21 (229)	20 (71)	25 (41)	
$40,000 to $59,999	16 (179)	13 (47)	13 (22)	
$60,000 and above	45 (492)	13 (46)	20 (33)	
Possession of health insurance				<0.01
No	2 (13)	7 (19)	12 (14)	
Yes	98 (807)	93 (272)	88 (100)	
Social-environmental stress[d]				<0.01
No	94 (1031)	79 (270)	81 (134)	
Yes	6 (67)	21 (72)	19 (32)	
Treatment				
Taking antidepressant medication				<0.01
No	80 (951)	85 (310)	92 (163)	
Yes	20 (236)	15 (54)	8 (15)	

Most recent characteristic	Non-Latinx White	Non-Latinx Black	Latinx	P value
Adherence to HAART since last visit				<0.01
Less than 75%	2 (7)	2 (5)	5 (5)	
75% to 94%	4 (19)	11 (23)	14 (13)	
95% to 99%	46 (199)	41 (87)	45 (43)	
100%	48 (210)	46 (97)	36 (35)	
Reported use of HAART				0.15
No	8 (37)	11 (26)	13 (15)	
Yes	92 (442)	89 (218)	87 (100)	
Health				
Family history of depression				<0.01
No	36 (422)	53 (194)	49 (88)	
Yes	64 (762)	47 (169)	51 (90)	
Burden of comorbidities				<0.01
0 comorbidities	21 (254)	28 (101)	40 (71)	
1 comorbidity	44 (528)	37 (135)	41 (73)	
2+ comorbidities	34 (405)	35 (128)	19 (34)	
Experienced insomnia since last visit				0.06
No	70 (572)	77 (224)	70 (80)	
Yes	30 (247)	23 (67)	30 (34)	
HIV serostatus				<0.01
Seronegative	59 (698)	32 (116)	35 (62)	
Seropositive	41 (489)	68 (248)	65 (116)	
	Median (IQR)			
Social				
Age (years)	62.8 (56.9–68.5)	55.2 (49.7–59.8)	51.0 (44.1–57.0)	<0.01
Sumparental educational attainment	1.0 (0.0–2.0)	0.0 (0.0–2.0)	0.0 (0.0–1.0)	<0.01
Social mobility	1.0 (0.0–2.0)	0.0 (0.0–1.0)	1.0 (0.0–2.0)	<0.01
Health				
CD4 cell count at HAART initiation	487.5 (319.0–728.0)	467.0 (326.0–678.0)	479.0 (331.0–601.0)	0.26
Viral copy-years following HAART initiation	3,170.5 (292.1–44,745.2)	28,189.4 (833.6–203,720.3)	18,467.5 (337.0–137,619.2)	<0.01
Total number of participants	1187	364	178	

CES-D = Center for Epidemiologic Studies Depression Scale; HAART = highly active antiretroviral therapy; IQR = interquartile range; MACS = Multicenter AIDS Cohort Study; MSM = men who have sex with men.

[a] Participants restricted to non-Latinx White, non-Latinx Black, and Latinx MSM who had responded to questions capturing social-environmental stress and social mobility.

[b] Person-visits restricted to those with CES-D scores recorded after the year 2000.

[c] Due to missing values, participant frequencies within individual variable categories may not sum to total participant counts.

[d] Defined as the neighborhood, crime, racism, or police ever having caused stress.

Table 2.

Percentage of Clinically Significant Depressive Symptom Variation Explained by Race/Ethnicity after Separately Adjusting for each of the Other Exposures Within 1,729 MSM[a], 2000–2017[b].

Exposure accounted for (along with age) in the models	Race/Ethnicity Partial R^2, %[c]		Exposure Partial R^2, %[d]	
	All participants	People living with HIV	All participants	People living with HIV
Social				
Baseline educational attainment	0.5	0.3	0.8	0.6
Recruitment center location	0.8	0.4	0.4	0.3
Recruitment cohort	0.6	0.3	0.2	0.4
Illicit drug use since last MACS visit	0.9	0.7	0.3	0.5
Self-reported annual income at a MACS visit	0.1	0.2	8.2	7.6
Possession of health insurance at a MACS visit	0.7	0.5	4.3	4.0
Social-environmental stress[e]	0.4	0.3	8.5	10.4
Sumparental educational attainment	0.5	0.2	10.4	12.2
Social mobility	0.5	0.3	8.3	9.4
Treatment				
Reported use of antidepressants at a MACS visit	1.3	1.1	3.7	3.4
Adherence to HAART taken since last MACS visit	NA	0.5	NA	26.9
Reported use of HAART at a MACS visit	NA	0.6	NA	0.9
Health				
Family history of depression	1.1	0.8	1.1	1.0
Burden of comorbidities at a MACS visit	0.9	0.6	0.1	0.0
Experienced insomnia since last MACS visit	0.8	0.5	12.0	13.3
HIV serostatus	0.8	NA	0.1	NA
CD4 cell count at HAART initiation	NA	0.6	NA	6.1
Viral copy-years following HAART initiation	NA	0.7	NA	2.4

HAART = highly active antiretroviral therapy; MACS = Multicenter AIDS Cohort Study; MSM = men who have sex with men; NA = not applicable.

[a]Participants restricted to non-Latinx White, non-Latinx Black, and Latinx MSM who had responded to questions capturing social-environmental stress and social mobility.

[b]Person-visits restricted to those with Center for Epidemiologic Studies Depression Scale scores recorded after the year 2000.

[c]Partial R^2 value for race/ethnicity, controlling for age and an exposure. Smaller partial R^2 values indicate that race/ethnicity did not explain much additional clinically significant depressive symptom variation beyond what was already accounted for by age and the exposure.

[d]Partial R^2 value for an exposure, controlling for age and race/ethnicity. Larger partial R^2 values indicate that the exposure explained more clinically significant depressive symptom variation after accounting for the effects of age and race/ethnicity.

[e]Defined as the neighborhood, crime, racism, or police ever having caused stress.

Table 3.

Predicted Prevalence[a] (%) of Clinically Significant Depressive Symptoms (CES-D 20) at a MACS Visit Within 1,729 MSM[b], 2000–2017[c].

Variable distribution[d]	Predicted[e]	Baseline education set[f]	Illicit drug use set[g]	Annual income set[f]	Health insurance set[f]	Stress set[f]	Parental education set[f]	Social mobility set[f]	Anti-depressants set[f]	HAART adherence set[f]	Use of HAART set[g]
All participants											
Non-Latinx White	9.4	9.4	9.3	9.4	9.4	9.4	9.4	9.4	9.4	NA	NA
Non-Latinx Black	22.2	21.2	21.6	21.1	22.1	19.0	23.1	22.8	23.6	NA	NA
Latinx	26.3	26.6	26.3	20.1	26.6	23.0	26.1	27.4	27.7	NA	NA
People living with HIV											
Non-Latinx White	14.0	14.0	13.9	14.0	14.0	14.0	14.0	14.0	14.0	14.0	14.0
Non-Latinx Black	20.4	19.6	20.1	20.2	20.4	16.9	20.6	20.6	22.7	20.2	20.4
Latinx	30.1	29.9	30.1	24.9	29.8	24.1	27.1	29.4	33.7	30.1	30.1

Variable distribution[d]	Predicted[e]	Family history set[h]	Comorbidities set[g]	Insomnia set[h]	Serostatus set[f]	CD4 cell count set[f]	Viral copy-years set[f]
All participants							
Non-Latinx White	9.4	9.6	9.6	9.2	9.4	NA	NA
Non-Latinx Black	22.2	22.2	22.6	22.2	23.4	NA	NA
Latinx	26.3	26.8	26.3	24.6	27.0	NA	NA
People living with HIV							
Non-Latinx White	14.0	13.8	14.1	13.9	NA	14.0	14.0
Non-Latinx Black	20.4	20.4	20.4	20.4	NA	21.8	19.6
Latinx	30.1	30.1	30.1	29.3	NA	29.0	29.9

CES-D = Center for Epidemiologic Studies Depression Scale; HAART = highly active antiretroviral therapy; MACS = Multicenter AIDS Cohort Study; MSM = men who have sex with men; NA = not applicable.

[a] Prevalence of reporting clinically significant depressive symptoms predicted through a classification tree.

[b] Restricted to non-Latinx White, non-Latinx Black, and Latinx MSM who had responded to questions capturing social-environmental stress and social mobility.

[c] Person-visits restricted to those with CES-D scores recorded after the year 2000.

[d] Set variables are adjusted to the distribution of the racial/ethnic group with the most health-beneficial, naturally occurring distribution.

[e] Predicted column provides the probability of reporting clinically significant depressive symptoms as predicted with the variable distributions as they exist in MACS.

[f] Variable set to distribution of non-Latinx White men.

[g] Variable set to distribution of Latinx men.

*h*Variable set to distribution of non-Latinx Black men.