Published in final edited form as:

Lancet HIV. 2017 July ; 4(7): e311–e320. doi:10.1016/S2352-3018(17)30067-X.

Isolating the sources of racial disparities in HIV prevalence among men who have sex with men (MSM) in Atlanta, GA: A modeling study

Steven M. Goodreau, PhD,

Department of Anthropology, University of Washington, Seattle WA, USA

Eli S. Rosenberg, PhD,

Department of Epidemiology, Emory University Rollins School of Public Health, Atlanta GA, USA

Samuel M. Jenness, PhD,

Department of Epidemiology, Emory University Rollins School of Public Health, Atlanta GA, USA

Nicole Luisi, MPH,

Department of Epidemiology, Emory University Rollins School of Public Health, Atlanta GA, USA

Sarah E. Stansfield, MA,

Department of Anthropology, University of Washington, Seattle WA, USA

Gregorio A. Millett, MPH, and

amfAR, The Foundation for AIDS Research, New York, NY, USA

Patrick S. Sullivan, PhD

Department of Epidemiology, Emory University Rollins School of Public Health, Atlanta GA, USA.

[Full professor]

Abstract

Background—In the United States, men who have sex with men (MSM) are a most-affected risk group, with Black MSM (BMSM) experiencing markedly higher HIV prevalence than White MSM (WMSM). We created a simulation model to jointly evaluate the strength of existing hypotheses and data in explaining these disparities.

Methods—A dynamic, stochastic agent-based network model of BMSM and WMSM ages 18-39 in Atlanta incorporated race-specific individual and dyadic-level prevention and risk behaviors, network attributes, and care patterns. We estimated parameters from two Atlanta-based studies of

Corresponding author: Steven M. Goodreau, Dept. of Anthropology, Campus Box 353100, University of Washington, Seattle, WA 98195, USA, goodreau@uw.edu, +1 (206) 685-3870.

Contributors:

SMG led the design and coding of the model, conducted some of the simulations, led the analysis of the simulations and co-led the writing.

ESR contributed to the design of the model, led the analysis of the source data to parametrize the model, and co-led the writing.

SMJ contributed to the design and coding of the model, the analysis of the source data and simulations, and the writing.

NL contributed to the design of the model and the analysis of the source data to parametrize the model

SES conducted some of the simulations and contributed to the analysis of the results

GAM contributed to the conceptualization of the question, analysis and the writing

PSS contributed to the design of the model, the analysis of the source data and simulations, and the writing

this population (n=1,117), supplemented by the literature. We modeled the ability for racial assortativity to generate or sustain HIV prevalence disparities, alone as well as in conjunction with scenarios of observed racial patterns in behavioral, care, and susceptibility parameters.

Findings—Race-assortative mixing alone could not sustain a pre-existing disparity. Differences in care cascade, stigma-related behaviors, and CCR5 genotypes each contributed considerably to the disparity, but nearly half (45%) of the disparity remained unexplained. A scenario evaluating race-specific reporting differences was the only one to yield Black HIV prevalence similar to that observed.

Interpretation—Racial assortativity is an inadequate explanation for observed disparities. Work to close the gap in the care cascade by race is imperative, as are efforts to increase serodiscussion and strengthen relationships among Black MSM especially. Further work is urgently needed to identify other sources and pathways of this disparity, integrate concomitant STI epidemics into models, and understand reasons for racial differences in behavioral reporting.

Funding—NIH R21HD075662;RC1MD004370;R01MH085600;R01HD067111;R01AI083060;R01HD068395;P30AI027757;P30AI050409;R24HD042828.

Introduction

Men who have sex with men (MSM) account for most new HIV diagnoses in the United States.¹ Concentrations of HIV infections among MSM are highest in the US South, where Georgia is uniquely high in both HIV prevalence and cases.² The MSM HIV epidemic is characterized by marked, longstanding Black/White racial disparities in HIV prevalence and incidence; a recent Atlanta study estimated prevalence of 43% among Black MSM (BMSM) and 13% among White MSM (WMSM), a 3.3-fold difference.^{3,4} Numerous explanations for the disproportionate impact among BMSM have been offered, and thoroughly reviewed.^{5,6} These include distal structural factors such as poverty, stigma, and institutionalized racism; however, the proximate causal pathways through which these factors enact disparate epidemics have proven challenging to elucidate. For structural factors to cause differentials in HIV incidence, they must mediate one or both more proximate causes: (1) the frequency with which HIV-negative individuals are potentially exposed to HIV and (2) the probability of transmission given exposure. The latter comprises factors associated with either the HIV-negative partner (e.g., circumcision status) or HIV-positive partner (e.g., viral suppression). Much literature around MSM race disparities focuses on self-reported individual risk behaviors (e.g., number of sex partners, substance use), but their explanatory power is limited since most behaviors are not higher among BMSM.^{5,6} Inadequate HIV suppression among partners of HIV-negative MSM places them at increased risk of acquisition, with racial disparities in the care continuum likely contributing to racially-disparate HIV incidence.⁷ Few studies have examined susceptibility differences, save for the protective CCR5-32 mutation, which is more prevalent among WMSM.⁸ Differences in sexual network properties are potential explanatory higher-order risk behaviors, although the primary evidence for meaningful differences by race are mixed.^{4,6,9,10}

Gay- and HIV-related stigma may also affect the health of BMSM, influencing their apparent and real HIV risks by shortening partnership durations and suppressing pre-sexual

HIV status discussion (serodiscussion). Stigma and mistrust of research may cause greater underreporting of risk-enhancing behaviors by BMSM; evidence suggests that correction for under-reporting leads to equivalent, rather than lower, risks for BMSM compared to WMSM.¹¹

Race-assortative mixing (the tendency to select same-race partners) can enable disparities arising from other factors to remain concentrated within racial groups. Partner race is a strong explanatory factor in incidence studies,^{12,13} suggesting a role in facilitating persistence for existing disparities.^{6,7,10} However, race-assortative mixing *alone* likely cannot sustain a disparity arising from other sources. Modeling theory, including work on HIV/STIs, predicts that a given epidemic typically heads towards a fixed equilibrium, regardless of its current state.¹⁴ For each sub-population, this entails a specific prevalence representing the point at which incident infections are balanced by deaths among HIV-infected persons. We have built a web tool illustrating this to build further intuition in the context of a simpler model (<https://sgoodreau.shinyapps.io/equilibria>), and elaborated on the theory in the Supplement (pp. 22-25). The theory suggests that, if race-assortative mixing alone cannot generate a disparity, it also cannot sustain one indefinitely, let alone cause increasing disparities over time. However, exceptions to this theory exist, and epidemic dynamics can be slow for lifelong infections like HIV.¹⁵ The ability for reported levels of race-assortative mixing, in combination with other reported proximate factors, to generate observed HIV racial disparities among MSM or maintain them over the long timeframes they have been observed, has not been assessed. One study using data-driven models to examine this question for young MSM over 15 years found that racial differences in HIV incidence narrowed over the course of the simulated epidemic.¹⁶

Dynamic network models are well-positioned to evaluate multiple proposed mechanisms for their ability to generate or sustain disparities, and have been called for.⁷ We implemented a model parameterized by data from young MSM in Atlanta, Georgia to answer two questions:

Question 1. Assuming that disparities emerged from some unmeasured differences early in the epidemic, how long could they be sustained under observed race-assortative mixing, with or without other observed racial differences?

Question 2. How much of the observed 3 3-fold racial disparity in HIV prevalence could be generated by specific measured proximate factors, alone or in combination?

We further probe our findings for Question 2 to consider the potential impact of behavior misclassification induced by societal-level factors.

Methods

Model design

We used dynamic, stochastic network models that extend earlier work.¹⁷ We summarize key features here; the Online Supplement provides additional information. We began with 10,000 MSM, each possessing fixed (e.g. race, circumcision status, CCR5 32) and dynamic (e.g. age, infection status) attributes. HIV-infected men possessed additional dynamic attributes (e.g. diagnosis status, treatment status, stage, viral load). We simulated multiple

transitions for each man by week, concurrently with relational dynamics (Table 1). Models simulated three contact networks for anal intercourse: main partnerships, casual partnerships with repeat contacts, and one-time contacts. These employed separable-temporal ERGMs,¹⁸ implemented in the R package suite *statnet* (www.statnet.org/trac/wiki) and *EpiModel* (www.epimodel.org). These methods allow fine control over numerous relational structures, including dependencies among having partners of each type; Table 2 includes modeled relational structures. Behavioral, demographic, and care-continuum parameters varied by race, or for counterfactual analyses were averaged across race. We modeled each scenario 16 times, with race-specific outcomes tracked until reaching equilibrium prevalence (Supplement p. 2).

Data sources

We obtained race-specific risk and prevention behaviors and network attributes from two studies of HIV/STI disparities in BMSM and WMSM in Atlanta, conducted 2010-2014 (Table 2). Involvement was a prospective HIV incidence cohort (n=803); the MAN Project was a cross-sectional chain-referral sexual networks study (n=314).^{4,13,19} Both used venue-time-space sampling (MAN Project for network-seed-level respondents). We included MSM from the baseline visit of Involvement and seeds from the MAN Project. Since these disparity-focused studies addressed MSM aged 18-39, the main timeframe over which HIV disparities appear, our model does also.

Participants completed self-administered computer-based questionnaires assessing demographics and prevention and risk behaviors for respondents and their most recent sex partners (Involvement, up to 5 in the previous 6 months; MAN Project, up to 10 in 12 months).⁴ We measured dyadic behaviors for pre-sexual, ongoing relationship, and last-sex time periods. Analyses included only dyads in which anal intercourse (AI) occurred at least once (n=2,626 dyads). We obtained treatment and efficacy parameters from published literature (Supplement p. 18). We adopted the susceptibility estimate for CCR5 32 heterozygotes from the one MSM-specific study,⁸ which found a stronger effect than heterosexuals studies have, because our model focused on MSM and our goal was to estimate maximum potential explanatory power for hypothesized factors.

BMSM reported lower levels of sexual risk behaviors, consistent with the literature and reports from the two source studies.^{3,4,13,19} However, BMSM's mean relationship durations were consistently lower, as was serodiscussion, possibly reflecting differences owing to stigma.²⁰ We thus combined sexual behaviors into two groups: stigma-associated behaviors that generally favor greater transmission for BMSM [*Stigma-associated behaviors*] and the remaining majority of sexual behaviors reported at higher levels by WMSM [*Majority of sexual behaviors*]. Table 3 outlines scenarios; all included race-assortative mixing at reported levels (~90% of relationships within-race across all partner types).

Our model does not aim to project the future, nor recreate past temporal trajectories, since the latter would require highly detailed historical data that do not exist. Rather, our aims are framed within a context where disparities in HIV burden have long existed at high levels, and where disparities in many determinants have been examined in isolation across various study designs. For Question 1, simulation duration has an explicit meaning, while for

Question 2 we focus on equilibria, which represents the maximum disparity a scenario can generate or sustain. Intervention models often include a calibration step in which model parameter(s) are varied to obtain historical prevalence before beginning intervention rollout. Here, we instead by design estimated all parameters from data sources, without calibration, since the ability of observed factors to generate observed prevalence by race and the disparity between them is precisely the objective.

For Question 1, we specified networks in which BMSM and WMSM have 43% and 13% prevalence, respectively, matching that observed in Involvement.⁴ We set all behaviors for BMSM and WMSM to between-race mean values (*Null* scenario), with reported levels of race-assortativity. We repeated the simulations with all parameters set to observed race-specific values (*As-observed*).

For Question 2, we created initial networks in which BMSM and WMSM both have 5% HIV prevalence and conducted the simulation under all scenarios in Table 3. Every scenario included race-assortativity. By grouping the modeled factors and assigning either observed race-specific values or mean values averaged across races, we probed the disparity generated by all hypothesized factors together (*As-observed*), each factor group in isolation, and combinations of factor groups. The final scenario set BMSM sexual behaviors equal to WMSM, to probe the potential impact of misreporting owing to higher social desirability bias for minority respondents.^{11,21,22} We began these runs with low, equal prevalence to test whether these scenarios could *generate* a disparity. We then repeated select runs starting with observed race-specific prevalence to confirm the final disparity was insensitive to initial prevalence, as predicted by modeling theory (Supplement pp. 22-25).

Role of the Funding Source

The funder of the study had no role in study design, analysis, or interpretation, or in writing of the report. The corresponding author had access to de-identified data used in the study and had final responsibility for the decision to submit for publication.

Results

Figure 1a-b demonstrates the potential for observed levels of race-assortative mixing to sustain pre-existing HIV prevalence disparities, holding all other factors equal by race (*Null* scenario). Initially, incidence is higher for BMSM, given the difference in partners' HIV prevalence. Subsequently, incidence and prevalence converge, with prevalence disparity reducing to half the initial disparity in 6 7 years, and by over 90% in 22 years. Figure 1c-d shows the same outcomes with all parameters set to observed race-specific values (*As-observed*). Disparities disappear even more quickly (prevalence disparity reducing by 50% and 90% in 3 3 years and 9 7 years, respectively), and equilibrium prevalence is higher for WMSM than BMSM.

Table 4 shows results for Question 2. Figure 2a-b shows the race-specific prevalences (*x*-axis=BMSM, *y*-axis=WMSM) for the same models, relative to observed Involvement results. Leaving all parameters *As-observed* by race yields mean BMSM and WMSM HIV prevalence values identical to those obtained using *As-Observed* parameters seeded with

observed prevalences. The same is true for the *Null* scenario. This supports the extension of basic modeling theory to our more complex case; the set of race-specific equilibrium prevalences is independent of initial (non-zero) prevalence, and these scenarios cannot sustain pre-existing disparities larger than those they generate. We provide an additional example and detail in the Supplement (p. 22-25).

We next consider the power of each factor group in explaining the observed disparity. Differences in the *Care continuum*, *CCR5-32* and *Stigma-associated behaviors* each explained a substantial disparity. However, *Majority of sexual behaviors* did the opposite, yielding substantially lower BSM prevalence. Racial differences in *Residual determinants* (circumcision, background mortality) yielded only a slight disparity. Most scenarios yielded WSM prevalence close to observed prevalence, but *Majority of sexual behaviors* overestimated it. BSM prevalence was underestimated by all models.

Combining factor sets, close to half of the disparity was explainable by *Biomedical determinants*, and about one-sixth by *Care and disclosure*. *All sexual behaviors* yielded the same magnitude reverse disparity as *Majority of sexual behaviors* alone. The *All BSM risk factors* scenario assessed *a priori* race-specific factor values that would favor higher BSM prevalence; this yielded a large disparity, and is the only scenario to include individual runs with a disparity within the observed data's confidence interval. The various factor sets generally yielded WSM prevalence close to observed, except for *All sexual behaviors*. Some factor sets yielded BSM prevalence values closer to observed prevalence than factor groups individually; all were still underestimates, however.

In a scenario probing *Misclassification of risk behaviors*, keeping all values *As-observed* by race but assigning BSM equivalent to WSM for *Majority of sexual behaviors*, mean BSM prevalence closely matched the observed 43.4%. However, WSM prevalence was higher than observed, generating a smaller-than-expected disparity.

Discussion

We used data from disparities-focused Atlanta studies and a previously-described model structure¹⁷ to ask whether the possible factors that have emerged in the scientific literature to date are sufficient to explain observed disparities. We find that these hypothesized explanatory factors accounted for, at most, 56% of observed disparities.

We demonstrated that, all else being equal, race-assortative mixing patterns in MSM cannot generate or sustain a racial disparity, let alone promote increased disparities over time. This suggests that race-assortativity along with pre-existing prevalence disparities is an insufficient explanation for population-level race disparities in incidence. However, it is important to distinguish the lack of population-level explanatory ability from that of prevalence among partner populations to predict short-term race-specific individual-level risk. In incidence studies¹³, HIV prevalence in specific partner pools (e.g., Black partners¹³, older partners¹²) remains a strong individual predictor of HIV acquisition. We urge that such associations not be used to stigmatize groups living with higher prevalence, but rather

reinforce the urgent need for approaches that improve the health of everyone living with or at risk for HIV while respecting the dignity of all impacted communities.^{4,13,23}

Lower HIV care engagement and attainment of viral suppression for BMSM explained a meaningful proportion of disparate HIV prevalence. Others have described the components of care and how social and structural factors, such as poverty and access to transportation, are associated with care outcomes.³ These findings underscore the urgency for achieving the U.S. National HIV/AIDS Strategy (NHAS) goal of increasing access to care, in order to achieve other NHAS goals to reduce new infections and HIV-related disparities.²⁴ Prospective studies are needed to more fully understand reasons for racially-disparate HIV care outcomes among MSM and to develop interventions addressing the entire care continuum for MSM. Despite the importance of HIV care in explaining prevalence disparities,^{3,7} these disparities existed before the availability of effective combination treatments, supporting the contribution of other factors.²⁵

We found that the CCR5-32 mutation had a large potential role in Black/White disparities among MSM. This hinged on the inclusion of a partially protective effect for the heterozygous genotype. A meta-analysis of predominantly small, diverse studies²⁶ failed to show a consistent protective effect for heterozygosity, but importantly did not include the only US MSM and race-focused study that informed our parameter.⁸ Given the immutable nature of genetic differences, this presents a programmatic and messaging challenge. Because other groups with lower HIV prevalence (e.g., Asian-Americans) similarly lack deletion genotypes, the ability for genetic differences alone to impart HIV disparities is likely limited, but we demonstrate their potential to operate in concert with other factors to yield significant disparities.⁶

Relatedly, we did not consider STI as biological co-factors, given the additional model complexity and overlap with causal and associative mechanisms for HIV. Results from Involvement demonstrated high and similar racially-disparate incidence of gonorrhea, chlamydia, and syphilis, and statistical modeling estimated that rectal STI explained 15% of subsequent HIV infection.^{4,27} That work did not consider increased HIV transmission from co-infected MSM. Models incorporating STI might expect to yield greater racial HIV disparities, and are in development.

This analysis lends several new insights into the role of risk behaviors in MSM epidemics. Modeling only the set of risk behaviors at observed levels, generally reported at lower levels for BMSM, yielded an inverse disparity. That this disparity was nearly identical to the scenario with all factors *As-observed* shows the strong leverage for risk behaviors in determining disparities, which masks biomedical factors in creating disparities. When modeling equal, rather than lower, behavioral risks for BMSM relative to WMSM, the direction of disparity was reversed and population-observed BMSM prevalence was obtained, suggesting the potential for underreporting to help explain disparities. Racial differences in reporting have been discounted due to qualitative consistency of reports across time and geography, but recent validation studies have demonstrated underreporting.^{6,11,21,22} Further understanding of the social determinants of underreporting and development of risk biomarkers in MSM studies are needed.²⁸ Two behaviors hypothesized as influenced by

stigma – reduced HIV serodiscussion and shorter partnership duration – operated differently than other behaviors. When considered separately, they yielded a modest disparity, demonstrating a mechanism through which stigma can shape HIV risk for BMSM.

Our model did not explain the full magnitude of disparities observed in surveillance and research studies, and we suggest several possible sources not accounted for in our model, beyond STI. We incorporated the only published nationally-representative estimates of care engagement among MSM by race, derived from CDC datasources, because comparable Atlanta-specific data were unavailable; the latter might reflect greater racial differences in engagement and viral suppression. This highlights the need for improved care continuum surveillance for all racial groups of MSM at all jurisdictional levels. We modeled race-assortative mixing probabilities as uniform within race; individual heterogeneity in the propensity for inter-racial partnerships, attributable to residential or cultural segregation, may amplify disparities.

Although our model represents Black and White MSM in Atlanta, our initial population size ($n=10,000$) is smaller than those communities. However, models for infectious diseases like STIs where contact rates do not scale with population size are generally robust to size above a small threshold (on the order of $n=1e^3$).²⁹ Nevertheless, we re-ran our main scenario at $n=5,000$ and $n=20,000$ (results not shown), and confirmed that results were consistent.

Because this model focused on MSM <40 years old, disparity convergences were faster than would be observed in reality. We did not model HIV pre-exposure prophylaxis (PrEP), an effective biomedical prevention modality, since observed disparities pre-date PrEP approval. Elsewhere we extend our framework to consider PrEP,³⁰ with subsequent work addressing PrEP's impact on racial disparities.

High HIV incidence among BMSM is one of the most pressing public health concerns of our times and a national priority.²⁴ Our model highlights the roles of access to effective HIV care, biological susceptibility, and behaviors, in influencing this risk. We demonstrate that there are residual sources of disparity that remain unexplained and demand further exploration, if we are to achieve health equity. Programs and policies must emphasize expanded access to antiretroviral therapies for HIV treatment and prophylaxis, while seeking to alleviate underlying social determinants and stigmas that shape BMSM's HIV risks. Our results confirm that the goals of the US National HIV/AIDS Strategy, if achieved, hold substantial potential to reduce HIV disparities by race among MSM.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

This project was funded by NIH research grants R21HD075662, RC1MD004370, R01MH085600, R01HD067111, R01AI083060, and R01HD068395. We were aided by additional institutional support came to the UW Center for AIDS Research (P30AI027757), the Emory Center for AIDS Research (P30AI050409), and the UW Center for Studies in Demography and Ecology (R24HD042828). We would like to thank study participants, the Statnet Development Team, the UW Network Modeling Group, the Helen R. Whitely Foundation and the Whitely Center.

References

1. Johnson AS, Hall HI, Hu X, Lansky A, Holtgrave DR, Mermin J. Trends in diagnoses of HIV infection in the United States, 2002–2011. *JAMA*. 2014; 312(4):432–4. [PubMed: 25038362]
2. Rosenberg ES, Grey JA, Sanchez TH, Sullivan PS. Rates of prevalent HIV infection, prevalent diagnoses and new diagnoses among men who have sex with men in US states, metropolitan statistical areas, and counties, 2012–2013. *JMIR Public Health and Surveillance*. in press.
3. Millett GA, Peterson JL, Flores SA, et al. Comparisons of disparities and risks of HIV infection in black and other men who have sex with men in Canada, UK, and USA: a meta-analysis. *Lancet*. 2012; 380(9839):341–8. [PubMed: 22819656]
4. Sullivan PS, Peterson J, Rosenberg ES, et al. Understanding racial HIV/STI disparities in black and white men who have sex with men: a multilevel approach. *PloS one*. 2014; 9(3):e90514. [PubMed: 24608176]
5. Mausbly C, Millett G, Lindsey K, et al. HIV Among Black Men Who Have Sex with Men (MSM) in the United States: A Review of the Literature. *AIDS and behavior*. 2013
6. Millett GA, Peterson JL, Wolitski RJ, Stall R. Greater risk for HIV infection of black men who have sex with men: a critical literature review. *American journal of public health*. 2006; 96(6)
7. Rosenberg ES, Millett GA, Sullivan PS, Del Rio C, Curran JW. Understanding the HIV disparities between black and white men who have sex with men in the USA using the HIV care continuum: a modeling study. *The lancet HIV*. 2014; 1(3):e112–e8. [PubMed: 25530987]
8. Marmor M, Sheppard HW, Donnell D, et al. Homozygous and heterozygous CCR5-Delta32 genotypes are associated with resistance to HIV infection. *Journal of acquired immune deficiency syndromes*. 2001; 27(5):472–81. [PubMed: 11511825]
9. Tieu HV, Nandi V, Hoover DR, et al. Do Sexual Networks of Men Who Have Sex with Men in New York City Differ by Race/Ethnicity? *AIDS patient care and STDs*. 2016; 30(1):39–47. [PubMed: 26745143]
10. Mustanski B, Birkett M, Kuhns LM, Latkin CA, Muth SQ. The Role of Geographic and Network Factors in Racial Disparities in HIV Among Young Men Who have Sex with Men: An Egocentric Network Study. *AIDS and behavior*. 2015; 19(6):1037–47. [PubMed: 25430501]
11. White D, Rosenberg ES, Cooper HL, et al. Racial differences in the validity of self-reported drug use among men who have sex with men in Atlanta, GA. *Drug and alcohol dependence*. 2014; 138:146–53. [PubMed: 24629628]
12. Garofalo R, Hottel AL, Kuhns LM, Gratzler B, Mustanski B. Incidence of HIV infection and Sexually Transmitted Infections and Related Risk Factors among Very Young Men Who Have Sex with Men. *Journal of acquired immune deficiency syndromes*. 2016
13. Sullivan PS, Rosenberg ES, Sanchez TH, et al. Explaining racial disparities in HIV incidence in black and white men who have sex with men in Atlanta, GA: a prospective observational cohort study. *Annals of epidemiology*. 2015; 25(6):445–54. [PubMed: 25911980]
14. Simon CP, Jacquez JA. Reproduction Numbers and the Stability of Equilibria of Si Models for Heterogeneous Populations. *Siam J Appl Math*. 1992; 52(2):541–76.
15. Huang WZ, Cooke KL, Castilochavez C. Stability and Bifurcation for a Multiple-Group Model for the Dynamics of Hiv Aids Transmission. *Siam J Appl Math*. 1992; 52(3):835–54.
16. Beck EC, Birkett M, Armbruster B, Mustanski B. A Data-Driven Simulation of HIV Spread Among Young Men Who Have Sex With Men: Role of Age and Race Mixing and STIs. *Journal of acquired immune deficiency syndromes*. 2015; 70(2):186–94. [PubMed: 26102448]
17. Beyrer C, Baral SD, van Griensven F, et al. Global epidemiology of HIV infection in men who have sex with men. *Lancet*. 2012; 380(9839):367–77. [PubMed: 22819660]
18. Krivitsky PN, Handcock MS. A separable model for dynamic networks. *J R Stat Soc B*. 2014; 76(1):29–46.
19. Hernandez-Romieu AC, Sullivan PS, Rothenberg R, et al. Heterogeneity of HIV Prevalence Among the Sexual Networks of Black and White Men Who Have Sex With Men in Atlanta: Illuminating a Mechanism for Increased HIV Risk for Young Black Men Who Have Sex With Men. *Sexually transmitted diseases*. 2015; 42(9):505–12. [PubMed: 26267877]

20. White D, Grey JA, Gorbach PM, Rothenberg RB, Sullivan PS, Rosenberg ES. Racial Differences in Partnership Attributes, Typologies, and Risk Behaviors Among Men Who Have Sex With Men in Atlanta, Georgia. *Arch Sex Behav*. 2016
21. Hernandez-Romieu AC, Sullivan PS, Rothenberg R, et al. Concordance of Demographic Characteristics, Sexual Behaviors, and Relationship Attributes Among Sex Dyads of Black and White Men Who Have Sex with Men. *Arch Sex Behav*. 2016
22. Piwowar-Manning E, Fogel JM, Laeyendecker O, et al. Failure to identify HIV-infected individuals in a clinical trial using a single HIV rapid test for screening. *HIV clinical trials*. 2014; 15(2):62–8. [PubMed: 24710920]
23. Matthews DD, Smith JC, Brown AL, Malebranche DJ. Reconciling Epidemiology and Social Justice in the Public Health Discourse Around the Sexual Networks of Black Men Who Have Sex With Men. *American journal of public health*. 2016; 106(5):808–14. [PubMed: 26890175]
24. White House Office of National AIDS Policy. Washington, D.C: 2015. The National HIV/AIDS Strategy: updated to 2020. <https://www.aids.gov/federal-resources/national-hiv-aids-strategy/nhas-update.pdf>;
25. Sullivan P, Chu S, Fleming P, Ward J. Changes in AIDS incidence for men who have sex with men, United States 1990-1995. *AIDS*. 1997; 11(13):1641–6. [PubMed: 9365770]
26. Liu S, Kong C, Wu J, Ying H, Zhu H. Effect of CCR5-Delta32 heterozygosity on HIV-1 susceptibility: a meta-analysis. *PloS one*. 2012; 7(4):e35020. [PubMed: 22496885]
27. Kelley C, Vaughan A, Luisi N, et al. The Effect of High Rates of Bacterial Sexually Transmitted Infections on HIV Incidence in a Cohort of Black and White Men who Have Sex with Men in Atlanta, GA. *AIDS research and human retroviruses*. 2015
28. Gallo MF, Steiner MJ, Hobbs MM, Warner L, Jamieson DJ, Macaluso M. Biological markers of sexual activity: tools for improving measurement in HIV/sexually transmitted infection prevention research. *Sexually transmitted diseases*. 2013; 40(6):447–52. [PubMed: 23677018]
29. Carnegie NB, Morris M. Size matters: concurrency and the epidemic potential of HIV in small networks. *PloS one*. 2012; 7(8):e43048. [PubMed: 22937011]
30. Jenness SM, Goodreau SM, Rosenberg E, et al. Impact of the Centers for Disease Control's HIV Preexposure Prophylaxis Guidelines for Men Who Have Sex With Men in the United States. *The Journal of infectious diseases*. 2016

Research in Context

Evidence before this study

Black/White disparities in HIV infection have been a hallmark of the US HIV epidemic for decades, but fully explaining them has been an elusive goal. We searched PubMed for articles published through June 5, 2016 using combinations of keywords “HIV”, “AIDS”, “MSM”, “men who have sex with men,” “disparity”, “disparities”, “race”, “racial”, “ethnic”, “ethnicity”, “Black”, “African American”, “White”, and “Caucasian”. We focused on studies that discussed either the empirical evidence for, or sources and causal mechanisms of, disparities in HIV incidence and prevalence between Black and White MSM in the US. These included countless empirical studies over decades that demonstrate the existence of these disparities. They also included multiple critical literature reviews and meta-analyses that document the existence and magnitude of many potential sources, including racial assortativity; proximal differences in the care cascade, sexual networking, and biological co-factors; and distal factors such as stigma and poverty. However, quantifying the magnitude of disparity that the many proximal sources could generate or sustain over time requires dynamic modeling, a fact that many of the papers acknowledge, and call for. One recent paper developed a data-driven network model to consider disparities in very young MSM over a 15-year time frame, but this paper did not attempt to partition the observed disparities attributed to each of the proposed sources, nor estimate the proportion unexplained. In addition, a series of theoretical modeling papers lays out the expected relationships between the generation and maintenance of disparities, although these have not been verified in the specific context of racial disparities in HIV in US MSM.

Added value of this study

Our study is the first to demonstrate in a dynamic model that a combination of many proposed proximal sources of racial disparities in HIV among US MSM actually generates a reverse disparity. We are able to quantify the amount that each component in combination with race-assortativity contributes to observed or reverse disparities, with the care cascade, biological co-factors, HIV serostatus disclosure and some aspects of partnership dynamics each generating a considerable amount of the observed disparity. We demonstrate that misclassification within other behavioral components would be sufficient to generate observed HIV prevalence among Black MSM. We clarify the relationship between the power of these causes to generate and to sustain disparities, and demonstrate that their ability to sustain pre-existing disparities on their own is short-lived.

Implications of all the available evidence

We provide the most thorough assessment to date of the ability for proposed sources to either generate or sustain observed racial disparities in HIV among US MSM over the long-term. High HIV incidence among Black MSM is one of the most pressing public health concerns of our times, and reducing this burden is a priority in the US National HIV/AIDS Strategy. We provide novel evidence for the relative importance of the proximal sources to this high burden, a necessary first step in determining the effectiveness of efforts to reduce that burden. We also demonstrate how much these

sources are not able to explain, highlighting the areas where more evidence is crucially needed.

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

Figure 1. Changes in race-specific HIV incidence and prevalence over time

These scenarios probe Question 1, the ability for pre-existing disparities that may have been generated early in the epidemic to be sustained, either by assortative mixing alone (*Null model*) or by the full set of race-specific behavioral, biological, demographic and clinical conditions as drawn from our studies and the literature (*As-observed* scenario). Results demonstrate that the disparities cannot be indefinitely sustained, but would begin to noticeably reduce within a few years. Prevalence plots are on left, incidence plots on right. (A-B) *Null model*. (C-D) *As-observed* scenario. Initial HIV prevalence by race is set to that observed in our source data. Individual lines represent each of 16 simulations; thick lines represent means.

Figure 2. Black vs. White HIV prevalence, by model scenario

This figure presents the race-specific HIV prevalence levels generated by each scenario, providing visual insight into whether the challenges in explaining observed disparities are due to systematic under-estimation of BSM prevalence, over-estimation of WSM prevalence, or both. BSM prevalence is shown on the X-axis, WSM prevalence on the Y-axis. Observed point estimates are shown by the small black square, surrounded by confidence limits shown by whiskers. For sake of legibility, individual runs are not shown; shapes represent the convex hull of 16 points reflecting individual runs. Plots are divided across two panels for legibility. (A) First seven scenarios; (B) final five scenarios. For scenario explanations, see Table 3. Many scenarios approximate WSM prevalence; however, the first 12 scenarios all strongly underestimate Black prevalence. This suggests the value in considering a scenario incorporating misclassification of some risk behaviors by Black MSM, based potentially on stigma or mistrust of research. This final scenario captured BSM prevalence, but over-estimated that for WSM.

Table 1**Model transitions**

Process	Notes
Arrivals	Constant rate, equal number of Black and White MSM
Departures due to aging out of model population	Occurs deterministically at age 40.
Departures due to background mortality	Occurs with age- and race-specific all-cause mortality rates.
Departures due to HIV mortality	Occurs as a complex function of time since infection off treatment, on treatment and fully suppressed, and on treatment and partially suppressed.
Main and casual partnership formation	Modeled using ERGMs; occur in patterns that maintain the race- specific cross-sectional distribution of partner counts, race mixing and age mixing from our data.
Main and casual partnership dissolution	Modeled using ERGMs; occur in patterns that maintain the race- specific partnership-type-specific relational durations.
AI within main and casual partnership	Drawn each week for each relationship from a Poisson distribution with means specific to race combination and relational type.
One-time AI contact	Drawn each week with probabilities that maintain race and age mixing, and with individuals' one-time contacts a function of their race, count of ongoing partnerships, and individual propensity.
HIV status disclosure	Modeled at the level of the relational pair; function of the races of the pair, relational type, and whether diagnosis occurs before or after start of relationship.
Condom use	Determined separately for each act; function of races of pair, relation type, and diagnosis and disclosure status of HIV+ partner.
Sexual role selection	Function of individual propensities, which vary by race; bi-directional AI allowed.
Transmission	Probabilities depend on condom use; the positive partner's infection stage, viral load and sexual position; and the negative partner's circumcision status (if insertive) and CCR5 32 genotype.
Viral dynamics	Largely follow those of previous models ¹⁷ and depend on time since infection and treatment status.
HIV testing	Small race-specific percentage of men never test; for those who do, intertest intervals are race-specific.
Treatment initiation	Contingent on diagnosis; timing after diagnosis varies by race.
Treatment cessation and re-initiation	Occur at race-specific rates in order to maintain reported prevalence of treatment and durable treatment by race.
Suppression	May occur at partial or full levels, at race-specific frequencies.

Table 2

Parameters varying by race

Parameter	Black MSM value	White MSM value	Determinant Group
Proportion of men never testing	7.7%	5.2%	Care continuum
Mean inter-test interval for men testing	43 weeks	45 weeks	Care continuum
Percent linked to care within 3 months of diagnosis	71.6%	82.9%	Care continuum
Proportion of diagnosed men who are on ART	22.3%	39.2%	Care continuum
Proportion of currently suppressed men who are durably suppressed for 1 year	58.8%	69.1%	Care continuum
Proportion of men on ART who are suppressed	62.4%	67.7%	Care continuum
CCR5 32 genotype frequencies (32 homozygote, heterozygote, wildtype homozygote)	0.0%, 3.4%, 96.6%	2.1%, 17.6%, 80.3%	CCR5 32
Probability of disclosure of HIV+ status to new main partner at outset of relationship	68.5%	88.9%	Stigma-associated behaviors
Probability of disclosure of HIV+ status to new casual partner at outset of relationship	52.7%	82.8%	Stigma-associated behaviors
Probability of disclosure of HIV+ status to one-time contact	44.5%	69.1%	Stigma-associated behaviors
Momentary degree distributions (i.e. proportion of men with a given number of main and casual relationships at a point in time)	0 cas 1 cas 2 cas 50.6% 15.1% 5.3%	0 cas 1 cas 2 cas 43.5% 18.4% 9.5%	Majority of sexual behaviors
Mean number of one-time AI events per week, for men with a given momentary degree distribution	0 main 1 main 20.7% 6.1% 0 cas 1 cas 2 cas	0 main 1 main 23.3% 3.3% 0 cas 1 cas 2 cas	Majority of sexual behaviors
Quintiles for mean number of of-time AI events per week	0 main 1 main 0.073% 0.091% 0.080% 0.055% 0.052% 0.052%	0 main 1 main 0.057% 0.084% 0.091% 0.057% 0.058% 0.058%	Majority of sexual behaviors
Proportion exclusively insertive	0.00, 0.010, 0.039, 0.074, 0.212	0.00, 0.003, 0.036, 0.068, 0.231	Majority of sexual behaviors
Proportion exclusively receptive	24.2%	22.8%	Majority of sexual behaviors
Circumcision prevalence	32.1%	22.8%	Majority of sexual behaviors
Mortality rates (per year)	87.4%	91.8%	Residual determinants
	0.00159 (18-24)	0.00103 (18-24)	Residual determinants
	0.00225 (25-34)	0.00133 (25-34)	
	0.00348 (35-39)	0.00214 (35-39)	

Black-Black dyad value Black-White dyad value White-White dyad value

Parameter	Black MSM value		White MSM value		Determinant Group
Mean main partnership duration	348 days	372 days	555 days	Stigma-associated behaviors	Stigma-associated behaviors
Mean casual partnership duration	131 days	286 days	144 days	Stigma-associated behaviors	Stigma-associated behaviors
Mean AI acts per week in main partnership	1.19	1.79	1.56	Majority of sexual behaviors	Majority of sexual behaviors
Mean AI acts per week in casual partnership	0.75	1.13	0.98	Majority of sexual behaviors	Majority of sexual behaviors
Base probability of condom use during AI, main partnership	0.38	0.10	0.15	Majority of sexual behaviors	Majority of sexual behaviors
Base probability of condom use during AI, casual partnership	0.39	0.11	0.16	Majority of sexual behaviors	Majority of sexual behaviors
Base probability of condom use during AI, one-time contact	0.49	0.15	0.22	Majority of sexual behaviors	Majority of sexual behaviors
Probability of intra-event role-versatility among two role-versatile MSM	0.42	0.56	0.49	Majority of sexual behaviors	Majority of sexual behaviors
Mean difference in square root of ages, main partnerships	0.42	0.45	0.52	Majority of sexual behaviors	Majority of sexual behaviors
Mean difference in square root of ages, casual partnerships	0.50	0.63	0.63	Majority of sexual behaviors	Majority of sexual behaviors
Mean difference in square root of ages, one-time contacts	0.46	0.59	0.59	Majority of sexual behaviors	Majority of sexual behaviors

For sources and derivations, see Supplement (pp. 3-22)

Table 3

Counterfactual scenarios used to answer Questions 1 and 2

	Factor group					
Dash indicates factors set to between-race mean values Check indicates factors set to observed race-specific values						
	Description	HIV care continuum	CCR5 32	Stigma-assoc behaviors	Majority of sexual behaviors	Residual determinants
	Null (<i>all factors set to between-race mean</i>)	-	-	-	-	-
	As-observed (<i>all factors race-specific</i>)	✓	✓	✓	✓	✓
Factor groups in isolation	Care continuum	✓	-	-	-	-
	CCR5 32	-	✓	-	-	-
	Stigma-associated behaviors (<i>relationship duration, HIV serodiscussion</i>)	-	-	✓	-	-
	Majority of sexual behaviors	-	-	-	✓	-
	Residual determinants (<i>background mortality, circumcision rates</i>)	-	-	-	-	✓
Combined	Biomedical determinants	✓	✓	-	-	-
	Care and disclosure ^a	✓	-	✓ ^a	-	-
	All sexual behaviors	-	-	✓	✓	-
	All BMSM risk factors	✓	✓	✓	-	✓
	Missclassification of risk behaviors	✓	✓	✓	BMSM assigned WMSM values	✓

^aFor this scenario, HIV serodiscussion was the only stigma-associated factor set to observed race-specific values

Note: all scenarios included observed race-assortative mixing

Results for Question 2

Table 4

Scenario Description	HIV prevalence, Black MSM	HIV prevalence, White MSM	HIV prevalence ratios (PR), Black MSM / White MSM	% of disparity explained
Observed (Involvement cohort)	197/454 = 43.4% [38.9% - 48.0%]	46/349 = 13.2% [9.9% - 17.0%]	3.30 [2.47 - 4.40]	
Null	544/5578 = 9.8% [9.4% - 10.0%]	556/5605 = 9.9% [9.2% - 10.3%]	0.99 [0.92 - 1.04]	-0.4% [-3.5% - 1.8%]
As-observed	558/5540 = 10.1% [9.5% - 10.3%]	1078/5568 = 19.4% [19.1% - 0%]	0.52 [0.50 - 0.54]	N/A
Factor groups in isolation				
Care continuum	609/5557 = 11.0% [10.6% - 11.7%]	505/5630 = 9.0% [8.4% - 9.4%]	1.23 [1.13 - 1.31]	10.0% [5.7% - 13.7%]
CCR5 32	694/5523 = 12.6% [12.0% - 13.5%]	490/5593 = 8.8% [8.2% - 9.1%]	1.44 [1.35 - 1.54]	19.1% [15.4% - 23.5%]
Stigma-associated behaviors	691/5552 = 12.4% [11.3% - 13.6%]	542/5561 = 9.7% [9.1% - 10.3%]	1.29 [1.17 - 1.41]	12.7% [7.2% - 17.8%]
Majority of sexual behaviors	703/5591 = 12.6% [12.0% - 13.0%]	1708/5381 = 31.7% [31.1% - 32.4%]	0.40 [0.37 - 0.41]	N/A
Residual factors	612/5591 = 10.9% [10.6% - 11.4%]	561/5596 = 10.0% [9.7% - 10.7%]	1.10 [1.02 - 1.16]	4.3% [1.0% - 6.8%]
Combined factor groups				
Biomedical determinants	844/5519 = 15.3% [14.4% - 16.2%]	451/5630 = 8.0% [7.6% - 8.7%]	1.93 [1.77 - 2.05]	40.5% [33.7% - 45.5%]
Care and disclosure	696/5528 = 12.6% [12.1% - 13.2%]	512/5601 = 9.1% [8.8% - 9.7%]	1.38 [1.31 - 1.46]	16.4% [13.6% - 20.0%]
All sexual behaviors	638/5609 = 11.4% [11.0% - 11.8%]	1572/5387 = 29.2% [28.6% - 29.5%]	0.39 [0.38 - 0.40]	N/A
All BMSM risk factors	1113/5423 = 20.5% [19.4% - 21.3%]	508/5600 = 9.1% [8.4% - 9.4%]	2.28 [2.18 - 2.36]	55.5% [51.3% - 59.1%]
Misclassification of risk behaviors	2255/5116 = 44.1% [43.9% - 44.0%]	1635/5483 = 29.8% [29.2% - 30.7%]	1.48 [1.45 - 1.53]	20.8% [19.7% - 22.9%]

— For observed values we report the mean and 95% confidence intervals.

— For simulated prevalence numbers, values represent the mean and interquartile range observed at the end of the 16 simulations, with initial population size of 10,000.

— Simulated prevalence ratios (PRs) are calculated separately at the end of each of the 16 runs; we report the mean and interquartile range of these 16 values.

— For the percent of disparity explained, we compare the mean and interquartile range of the 16 simulated prevalence ratios to the observed point estimate, using the formula $(PR_{sim}-1)/(PR_{obs}-1)$. For scenarios in which all simulations yielded a larger HIV prevalence for White than Black men, we report the percent of disparity explained as not applicable (N/A). The percent of disparity explained for the Null model should be centered on 0, with stochasticity, by design.