Sources of racial disparities in HIV prevalence in men who have sex with men in Atlanta, GA, USA: a modelling study

Steven M Goodreau, Eli S Rosenberg, Samuel M Jenness, Nicole Luisi, Sarah E Stansfield, Gregorio A Millett, Patrick S Sullivan

Summary

Background **In the USA, men who have sex men (MSM) are at high risk for HIV, and black MSM have a substantially higher prevalence of infection than white MSM. We created a simulation model to assess the strength of existing hypotheses and data that account for these disparities.**

Methods **We built a dynamic, stochastic, agent-based network model of black and white MSM aged 18–39 years in Atlanta, GA, USA, that incorporated race-specific individual and dyadic-level prevention and risk behaviours, network attributes, and care patterns. We estimated parameters from two Atlanta-based studies in this population (n=1117), supplemented by other published work. We modelled the ability for racial assortativity to generate or sustain disparities in the prevalence of HIV infection, alone or in conjunction with scenarios of observed racial patterns in behavioural, care, and susceptibility parameters.**

Findings **Race-assortative mixing alone could not sustain a pre-existing disparity in prevalence of HIV between black and white MSM. Differences in care cascade, stigma-related behaviours, and *CCR5* genotype each contributed substantially to the disparity (explaining 10·0%, 12·7%, and 19·1% of the disparity, respectively), but nearly half (44·5%) could not be explained by the factors investigated. A scenario assessing race-specific reporting differences in risk behaviour was the only one to yield a prevalence in black MSM (44·1%) similar to that observed (43·4%).**

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 particularly. Further work is urgently needed to identify other sources of, and pathways for, this disparity, to integrate concomitant epidemics into models, and to understand reasons for racial differences in behavioural reporting.**

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Department of Anthropology, University of Washington, Seattle, WA, USA (S M Goodreau PhD, S E Stansfield MA); **Department of Epidemiology, Emory University Rollins School of Public Health, Atlanta, GA, USA** (E S Rosenberg PhD, S M Jenness PhD, N Luisi MPH, Prof P S Sullivan PhD); **and amfAR, the Foundation for AIDS Research, New York, NY, USA** (G A Millett MPH)

Correspondence to:
Dr Steven M Goodreau, Department of Anthropology, Campus Box 353100, University of Washington, Seattle, WA 91895, USA
goodreau@uw.edu

Introduction

Men who have sex with men (MSM) account for most new HIV diagnoses in the USA.[1] Concentrations of HIV infections among MSM are highest in southern USA, and Georgia is the only state that ranks in the top five for both percentage of MSM and absolute number of MSM living with a diagnosis of HIV.[2] The HIV epidemic in MSM is characterised by marked, long-standing racial disparities between black and white populations: in a study in Atlanta, GA, the estimated prevalence was 43% in black MSM and 13% in white MSM, a 3·3-fold disparity.[3,4]

Several explanations for the disproportionate effect of HIV among black MSM have been offered and thoroughly reviewed,[5,6] including distal structural factors such as poverty, stigma, and institutionalised racism. However, the proximal causal pathways through which these factors enact disparate epidemics have proven challenging to elucidate. For structural factors to cause differentials in incidence of HIV infection, they must mediate one or both of two more proximal causes: the frequency with which HIV-negative individuals are potentially exposed to HIV, and the probability of transmission if exposed, which comprises factors associated with either the HIV-negative partner (eg, circumcision status) or the HIV-positive partner (eg, viral suppression).

Much work about racial disparities in HIV infection among MSM focuses on self-reported individual risk behaviours (eg, number of sex partners, substance use), and thus has limited explanatory power, because most behaviours are not more common in black MSM than white MSM.[5,6] Inadequate HIV suppression among HIV-positive partners of HIV-negative MSM places them at increased risk of acquisition, with racial disparities in the care continuum probably contributing to racial disparates in incidence.[7] Few studies of susceptibility differences have been done, although the *CCR5Δ32* mutation, which is more prevalent in populations of European ancestry than of African ancestry, is protective against infection.[8] Differences in sexual-network properties are another potential set of explanations, although the primary evidence for meaningful differences by race is mixed.[4,6,9,10]

Stigma related to sexuality and HIV can also affect the health of black MSM and influence their apparent and real HIV risks by shortening partnership durations and suppressing discussion of HIV status before sex (ie, serodiscussion). Stigma and mistrust of research might

Research in Context

Evidence before this study

Disparities in HIV infection between black and white populations have been a hallmark of the US HIV epidemic for decades, but to account for them fully has proved challenging. We searched PubMed with combinations of the keywords "HIV", "AIDS", "MSM", "men who have sex with men", "disparity", "disparities", "race", "racial", "ethnic", "ethnicity", "black", "African American", "white", and "Caucasian" for articles published in English on or before June 5, 2016 (the date of our final search). We focused on studies that discussed either the empirical evidence for, or sources and causal mechanisms of, disparities in incidence and prevalence of HIV infection between black and white men who have sex with men (MSM) in the USA. We found many empirical studies over decades that show the existence of these disparities, and several critical literature reviews and meta-analyses that show the existence and magnitude of many potential sources, including racial assortativity, proximal differences in the care cascade, sexual networking, and biological cofactors, and distal factors such as stigma and poverty. However, quantification of the magnitude of disparity that the many proximal sources could generate or sustain over time necessitates dynamic modelling, which the authors of many of the papers acknowledge and call for. One group of investigators developed a data-driven network model to assess disparities in very young (age 16·0–21·8 years) MSM during a 15 year timeframe, but they did not attempt to partition the detected disparities attributed to each of the proposed sources, or to estimate the proportion unexplained. Additionally, the authors of a series of theoretical modelling papers laid out the expected relations between the generation and maintenance of disparities, although these relations have not been verified in the specific context of racial disparities in HIV infection in US MSM.

Added value of this study

Our study is the first to show in a dynamic model that a combination of many proposed proximal sources of racial disparities in HIV among US MSM generate a reverse disparity. We quantified the amount that each component, in combination with race assortativity, contributes to observed or reverse disparities, with the care cascade, biological cofactors, HIV serostatus disclosure, and some aspects of partnership dynamics each contributing a substantial amount to the observed disparity. We showed that misclassification within other behavioural components would be sufficient to generate the observed prevalence of HIV among black MSM. We examined the power of these causes to generate and sustain disparities, and showed that the ability to sustain pre-existing disparities on their own is short-lived.

Implications of all the available evidence

We provide the most thorough assessment so far of the ability for proposed sources to either generate or sustain observed racial disparities in HIV among US MSM in the long term. High incidence of HIV infection among black MSM is one of the most pressing public health concerns, and reduction of 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 show how much these sources cannot explain, highlighting the areas where more evidence is crucially needed.

cause under-reporting of risk-enhancing behaviours by black MSM: evidence suggests that correction for under-reporting leads to equivalent, rather than lower, risks for black MSM compared with white MSM.[11]

Race-assortative mixing (the tendency to select same-race partners) can enable disparities arising from other factors to remain concentrated within 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 probably cannot sustain disparities arising from other sources. Modelling theory, including work on HIV and other sexually transmitted infections (STIs), predicts that a given epidemic typically heads towards a fixed equilibrium, irrespective of its current state.[14] For each subpopulation, a specific prevalence represents the point at which incident infections are balanced by deaths among people with HIV. We have built a web tool that shows this concept to build further intuition in the context of a simpler model (appendix). The theory suggests that, if race-assortative mixing alone cannot generate a disparity then it also cannot sustain a disparity indefinitely or 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 proximal factors, to generate racial disparities in HIV in MSM or maintain disparities over the long timeframes that they have been reported, has not been assessed. A study in which data-driven models were used to examine this question for young MSM over 15 years showed that racial differences in incidence of HIV infection narrowed over the course of the simulated epidemic.[16]

Dynamic network models are well positioned to assess multiple proposed mechanisms for the ability to generate or sustain disparities, and have been called for.[7] We implemented a model parameterised by data from young MSM in Atlanta to answer two questions. First, assuming that disparities emerged from some unmeasured differences early in the epidemic, how long could they be sustained under reported race-assortative mixing, with or without other recorded racial differences? Second, how much of the 3·3-fold racial disparity in prevalence of

For the **web tool** see https://prism.shinyapps.io/mixing

See Online for appendix

Panel: Notes on model transitions

Arrivals
Constant rate, equal number of black and white men who have sex with men

Departures by ageing out of model population
Occurs deterministically at age 40

Departures due to background mortality
Occurs with age-specific 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 partly suppressed

Main and casual partnership formation
Modelled with exponential random graph models, 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
Modelled with exponential random graph models, occur in patterns that maintain the race-specific partnership-type-specific relational durations

Anal intercourse 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 anal intercourse 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
Modelled at the level of the relational pair, function of the races of the pair, relational type, and whether diagnosis occurs before or after the start of relationship

Condom use
Determined separately for each act, function of races of pair, relation type, and diagnosis and disclosure status of partner with HIV

Sexual role selection
Function of individual propensities, which vary by race; bi-directional anal intercourse allowed

Transmission
Probabilities depend on condom use; infection stage, viral load, and sexual position of the partner with HIV; and the HIV-negative partner's circumcision status (if insertive) and *CCR5Δ32* 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 reinitiation
Occur at race-specific rates to maintain reported prevalence of treatment and durable treatment by race

Suppression
Can occur at partial or full levels, at race-specific frequencies

HIV infection could be generated by specific measured proximal factors, alone or in combination? We also further explore our findings for the second question to consider the potential effect of behaviour misclassification caused by societal factors.

Methods

Model design

We used dynamic, stochastic network models that extend previous work.[17] We began with 10 000 MSM, each of whom possessed fixed (eg, race, circumcision status, *CCR5Δ32* status) and dynamic (eg, age, infection status) attributes. Men with HIV had additional dynamic attributes (eg, diagnosis status, treatment status, stage, viral load). We simulated multiple transitions for each man by week, concurrently with relational dynamics (panel). Models simulated three contact networks for anal intercourse: main partnerships, casual partnerships with repeat contacts, and one-time contacts. These simulations employed separable-temporal exponential random graph models,[18] implemented in the R package suite Statnet and the R package EpiModel. These methods allow fine control over several relational structures, including dependencies among having partners of each type. Tables 1 and 2 include modelled relational structures. Behavioural, demographic, and care-continuum parameters varied by race, or for counterfactual analyses were averaged across races. We modelled each scenario 16 times, with race-specific outcomes tracked until reaching equilibrium prevalence (appendix).

For **Statnet** see https://statnet.org/trac/wik

For **EpiModel** see www.epimodel.org

Data sources

We obtained race-specific risk and prevention behaviours and network attributes from two studies of HIV disparities between black and white MSM and in Atlanta that were done in 2010–14 (tables 1, 2).[4,13,19] Involvement was a prospective HIV incidence cohort (n=803); the MAN Project was a cross-sectional chain-referral sexual

	Black MSM value	White MSM value	Determinant group
Proportion of men never testing for HIV	7·7%	5·2%	Care continuum
Mean inter-test interval for men who test	43 weeks	45 weeks	Care continuum
Proportion linked to care within 3 months of diagnosis	71·6%	82·9%	Care continuum
Proportion of diagnosed men who are on antiretroviral therapy	22·3%	39·2%	Care continuum
Proportion of men with current viral suppression who are durably suppressed for 1 year	58·8%	69·1%	Care continuum
Proportion of men on antiretroviral therapy with viral suppression	62·4%	67·7%	Care continuum
CCR5Δ32 genotype frequencies	..	..	*CCR5Δ32*
Δ32 homozygote	0·0%	2·1%	..
Heterozygote	3·4%	17·6%	..
Wildtype homozygote	96·6%	80·3%	..
Probability of disclosure of HIV-positive status to new main partner at outset of relationship	68·5%	88·9%	Stigma-associated behaviours
Probability of disclosure of HIV-positive status to new casual partner at outset of relationship	52·7%	82·8%	Stigma-associated behaviours
Probability of disclosure of HIV-positive status to one-time contact	44·5%	69·1%	Stigma-associated behaviours
Momentary degree distributions (ie, proportion of men with a given number of main and casual relationships at a point in time)	..	..	Majority of sexual behaviours
No main or casual	50·6%	43·5%	..
No main, one casual	15·1%	18·4%	..
No main, two casual	5·3%	9·5%	..
One main, no casual	20·7%	23·3%	..
One main, one casual	6·1%	3·3%	..
One main, two casual	2·2%	2·0%	..
Mean number of one-time anal intercourse events per week, for men with a given momentary degree distribution	..	..	Majority of sexual behaviours
No main or casual	0·073	0·057	..
No main, one casual	0·091	0·084	..
No main, two casual	0·080	0·091	..
One main, no casual	0·055	0·057	..
One main, one casual	0·052	0·058	..
One main, two casual	0·052	0·058	..
Quintiles for mean number of one-time anal intercourse events per week	..	..	Majority of sexual behaviours
1	0·000	0·000	..
2	0·010	0·003	..
3	0·039	0·036	..
4	0·074	0·068	..
5	0·212	0·231	..
Proportion exclusively insertive	24·2%	22·8%	Majority of sexual behaviours
Proportion exclusively receptive	32·1%	22·8%	Majority of sexual behaviours
Circumcision prevalence	87·4%	91·8%	Residual determinants
Mortality rates (per year)	..	..	Residual determinants
Aged 18–24 years	0·00159	0·00103	..
Aged 25–34 years	0·00225	0·00133	..
Aged 35–39 years	0·00348	0·00214	..

The appendix contains sources and derivations. MSM=men who have sex with men.

***Table 1*: Individual-level parameters related to HIV infection that vary by race**

networks study (n=314).[4,13,19] Venue-time-space sampling was used for both (in the MAN Project, it was used for network-seed-level respondents). We included MSM from the baseline visit in Involvement and seeds from the MAN Project. Because these disparity-focused studies addressed MSM aged 18–39 years, the main timeframe during which HIV disparities appear, our model does so also.

Participants completed self-administered computer-based questionnaires assessing demographics and prevention and risk behaviours for themselves and their most recent sexual partners (Involvement, up to five in the previous 6 months; MAN Project, up to ten in 12 months).[4] We measured dyadic behaviours for pre-sexual, ongoing relationship, and last-sex periods. Analyses included only dyads in which anal intercourse occurred at least once (n=2626 dyads). We obtained treatment and efficacy parameters from published literature (appendix). We adopted susceptibility estimate for *CCR5Δ32* heterozygotes from the only MSM-specific study,[8] which showed a stronger effect than reported in studies of heterosexual people, because our model focused on MSM and our goal was to estimate maximum potential explanatory power for hypothesised factors.

Black MSM reported lower levels of sexual risk behaviours than did white MSM, consistent with published work and reports from the two source studies.[3,4,13,19] However, the mean duration of black MSM's relationships was consistently shorter than that of white MSM's relationships, and serodiscussion was less common in black than in white MSM, possibly reflecting differences resulting from stigma.[20] We thus combined sexual behaviours into two groups: stigma-associated behaviours that generally favour greater transmission for black MSM, and most other sexual behaviours, which are reported more commonly by white MSM. Table 3 outlines scenarios, all of which included race-assortative mixing at reported levels (~90% of relationships within-race across all partner types).

We did not aim to project the future or to recreate past temporal trajectories (which would require highly detailed historical data that are not available). Rather, our aims are framed within a context in which disparities in HIV burden have long existed at high levels, and in which disparities in many determinants have been examined in isolation across various study designs. For our first question (ie, how long could racial disparities in HIV infection be sustained under reported race-assortative mixing?) simulation duration has an explicit meaning. For our second question (ie, how much of the racial disparity in prevalence could be generated by specific measured proximal factors?) we focus on equilibrium, which represents the maximum disparity a scenario can generate or sustain. Intervention models often include a calibration step in which model parameters are varied to obtain historical prevalence before beginning intervention rollout. Instead, by design, we estimated all parameters

	Black–black dyad value	Black–white dyad value	White–white dyad value	Determinant group
Mean main partnership duration	348 days	372 days	555 days	Stigma-associated behaviours
Mean casual partnership duration	131 days	286 days	144 days	Stigma-associated behaviours
Mean anal intercourse acts per week in main partnership	1·19	1·79	1·56	Majority of sexual behaviours
Mean anal intercourse acts per week in casual partnership	0·75	1·13	0·98	Majority of sexual behaviours
Base probability of condom use during anal intercourse, main partnership	0·38	0·10	0·15	Majority of sexual behaviours
Base probability of condom use during anal intercourse, casual partnership	0·39	0·11	0·16	Majority of sexual behaviours
Base probability of condom use during anal intercourse, one-time contact	0·49	0·15	0·22	Majority of sexual behaviours
Probability of intra-event role-versatility among two role-versatile men who have sex with men	0·42	0·56	0·49	Majority of sexual behaviours
Mean difference in square root of ages, main partnerships	0·42	0·45	0·52	Majority of sexual behaviours
Mean difference in square root of ages, casual partnerships	0·50	0·63	0·63	Majority of sexual behaviours
Mean difference in square root of ages, one-time contacts	0·46	0·59	0·59	Majority of sexual behaviours

The appendix contains sources and derivations.

***Table 2*: Dyad-level parameters related to HIV infection that vary by race**

	HIV care continuum	*CCR5Δ32*	Stigma-associated behaviours	Majority of sexual behaviours	Residual determinants
Null (all factors set to between-race mean)	–	–	–	–	–
As observed (all factors race-specific)	✓	✓	✓	✓	✓
Factor groups in isolation					
Care continuum	✓	–	–	–	–
CCR5Δ32	–	✓	–	–	–
Stigma-associated behaviours (relationship duration, HIV serodiscussion)	–	–	✓	–	–
Majority of sexual behaviours	–	–	–	✓	–
Residual determinants (background mortality, circumcision rates)	–	–	–	–	✓
Combined					
Biomedical determinants	✓	✓	–	–	–
Care and disclosure*	✓	–	✓	–	–
All sexual behaviours	–	–	✓	✓	–
All risk factors in black men who have sex with men	✓	✓	✓	–†	✓
Misclassification of risk behaviours	✓	✓	✓	–	✓

Dashes show factors set to between-race mean values. Ticks show factors set to observed race-specific values. All scenarios included observed race-assortative mixing.
*For this scenario, HIV serodiscussion was the only stigma-associated factor set to observed race-specific values. †Black men who have sex with men assigned values for white men who have sex with men.

***Table 3*: Modelled scenarios**

from data sources, without calibration, because the ability of observed factors to generate observed prevalence by race and the disparity between them is precisely the objective.

For our first question, we specified networks in which the prevalence of HIV is 43% in black MSM and 13% in white MSM, matching that in Involvement.[4] We set all behaviours for black and white MSM to between-race mean values (ie, a null scenario), with reported levels of race assortativity. We repeated the simulations with all parameters set to observed race-specific values (ie, as observed).

For our second question, we created initial networks in which the prevalence of HIV infection in both black and white MSM was 5%, and did the simulation under all scenarios in table 3. Every scenario included race assortativity. By grouping the modelled factors and assigning either observed race-specific values or mean values averaged across races, we probed the disparity generated by all hypothesised factors together (ie, as observed), each factor group in isolation, and combinations of factor groups. The final scenario set sexual behaviours in black MSM as equivalent to those in

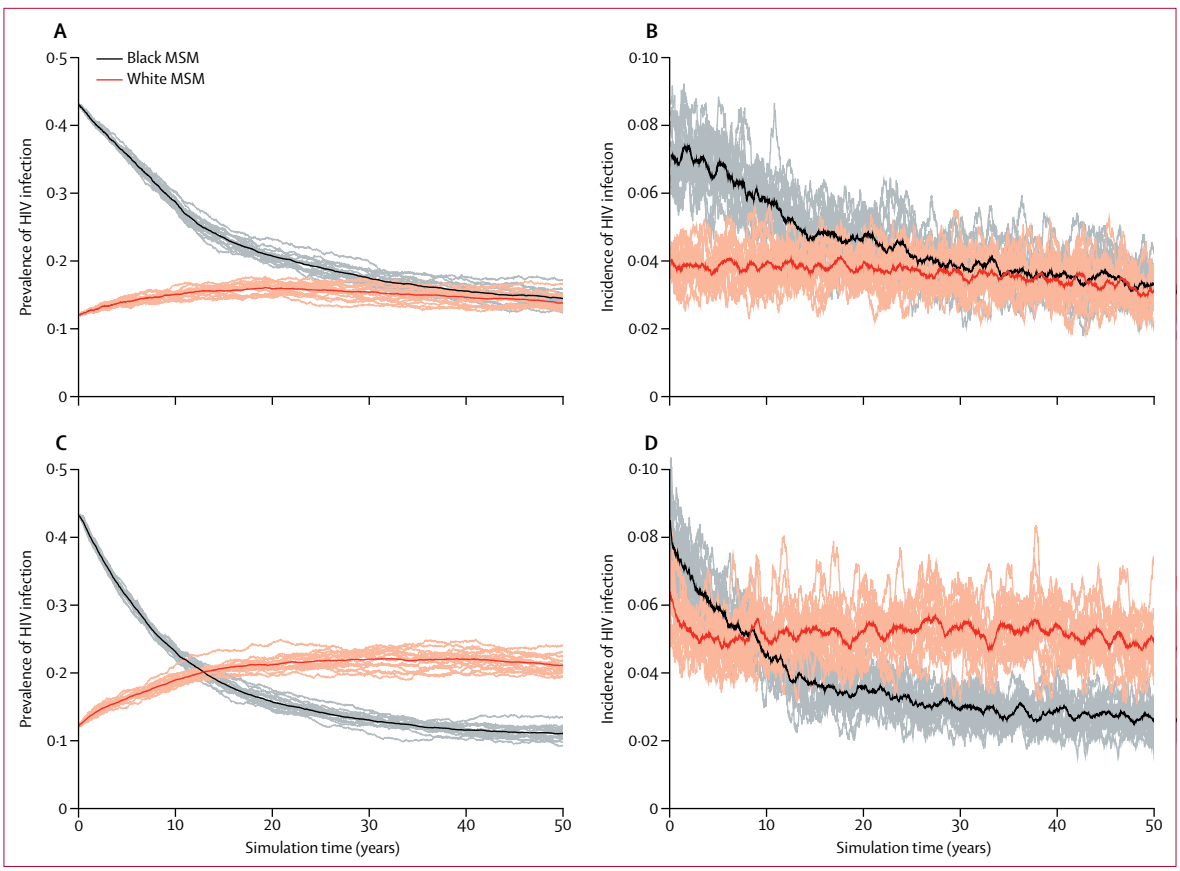

***Figure 1*: Changes in race-specific prevalence (A) and incidence (B) of HIV infection in the null model; and in race-specific prevalence (C) and incidence (D) of HIV infection in the as-observed scenario**

These scenarios probe the ability for pre-existing disparities that could have been generated early in the epidemic to be sustained, either by assortative mixing alone (ie, the null model) or by the full set of race-specific behavioural, biological, demographic, and clinical conditions as drawn from our studies and published work (ie, the as-observed scenario). Initial prevalence by race is set to that observed in our source data. Individual lines represent each of 16 simulations; thick lines represent means. MSM=men who have sex with men.

white MSM, to probe the potential effects of misreporting due to increased social desirability bias in 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 specific runs, starting with observed race-specific prevalence, to confirm that the final disparity was insensitive to initial prevalence, as predicted by modelling theory (appendix). The model code is available online.

For **model code** see https://github.com/statnet/MSMRaceDisparities_LancetHIV2017

Role of the funding source

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

Results

For our null model beginning with observed race-specific prevalence, incidence was initially higher in black MSM because of the difference in prevalence in partners. Subsequently, incidence and prevalence converged, with prevalence disparity falling to half the initial disparity in 6·7 years, and by over 90% in 22 years (figure 1A, 1B). When all parameters were set to observed race-specific values (ie, as observed), disparities disappeared even more quickly (the disparity in prevalence fell by 50% and 90% in 3·3 years and 9·7 years, respectively), and equilibrium prevalence was higher for white than for black MSM (figure 1C, 1D).

Table 4 shows results for how much of the 3·3-fold racial disparity in prevalence could be generated by specific measured proximal factors, alone or in combination. Figure 2 shows the race-specific prevalences for the same models, relative to observed results in the Involvement trial. Leaving all parameters as observed by race yielded mean prevalences of HIV infection in black and white MSM identical to those obtained with as-observed parameters seeded with observed prevalences. The same was true for the null scenario. This finding supports the extension of basic modelling theory to our more complex case: the set of race-specific equilibrium prevalences is

	Prevalence of HIV infection in black MSM	Prevalence of HIV infection in white MSM	Ratio of prevalence of HIV infection in black MSM to prevalence of HIV infection in white MSM	% of disparity explained
Observed (Involvement cohort)	197/454 (43·4%, 38·9 to 48·0)	46/349 (13·2%, 9·9 to 17·0)	3·30 (2·47 to 4·40)	N/A
Null	544/5578 (9·8%, 9·4 to 10·0)	556/5605 (9·9%, 9·2 to 10·3)	0·99 (0·92 to 1·04)	−0·4% (−3·5 to 1·8)
As observed	558/5540 (10·1%, 9·5 to 10·3)	1078/5568 (19·4%, 19·1 to 20·0)	0·52 (0·50 to 0·54)	N/A
Factor groups in isolation				
Care continuum	609/5557 (11·0%, 10·6 to 11·7)	505/5630 (9·0%, 8·4 to 9·4)	1·23 (1·13 to 1·31)	10·0% (5·7 to 13·7)
CCR5Δ32	694/5523 (12·6%, 12·0 to 13·5)	490/5593 (8·8%, 8·2 to 9·1)	1·44 (1·35 to 1·54)	19·1% (15·4 to 23·5)
Stigma-associated behaviours	691/5552 (12·4%, 11·3 to 13·6)	542/5561 (9·7%, 9·1 to 10·3)	1·29 (1·17 to 1·41)	12·7% (7·2 to 17·8)
Majority of sexual behaviours	703/5591 (12·6%, 12·0 to 13·0)	1708/5381 (31·7%, 31·1 to 32·4)	0·40 (0·37 to 0·41)	N/A
Residual factors	612/5591 (10·9%, 10·6 to 11·4)	561/5596 (10·0%, 9·7 to 10·7)	1·10 (1·02 to 1·16)	4·3% (1·0 to 6·8)
Combined factor groups				
Biomedical determinants	844/5519 (15·3%, 14·4 to 16·2)	451/5630 (8·0%, 7·6 to 8·7)	1·93 (1·77 to 2·05)	40·5% (33·7 to 45·5)
Care and disclosure	696/5528 (12·6%, 12·1 to 13·2)	512/5601 (9·1%, 8·8 to 9·7)	1·38 (1·31 to 1·46)	16·4% (13·6 to 20·0)
All sexual behaviours	638/5609 (11·4%, 11·0 to 11·8)	1572/5387 (29·2%, 28·6 to 29·5)	0·39 (0·38 to 0·40)	N/A
All risk factors in black MSM	1113/5423 (20·5%, 19·4 to 21·3)	508/5600 (9·1%, 8·4 to 9·4)	2·28 (2·18 to 2·36)	55·5% (51·3 to 59·1)
Misclassification of risk behaviours	2255/5116 (44·1%, 43·9 to 44·0)	1635/5483 (29·8%, 29·2 to 30·7)	1·48 (1·45 to 1·53)	20·8% (19·7 to 22·9)

Data are n/N, unless otherwise specified. For observed values (ie, the first row), we report 95% CIs; all other data are reported with the IQR. For simulated prevalence numbers, values represent the mean (IQR) observed at the end of the 16 simulations, with initial population size of 10 000. Simulated prevalence ratios are calculated separately at the end of each of the 16 runs and are reported as mean (IQR). For percentage of disparity explained, we compared the mean (IQR) of the 16 simulated prevalence ratios with the observed point estimate, using the formula $(PR_{sim}-1)/(PR_{obs}-1)$, where PR_{sim} is the simulated prevalence ratio and PR_{obs} is the observed prevalence ratio. For scenarios in which all simulations yielded a larger prevalence of HIV infection for white than for black MSM, we report the percentage disparity as N/A. The percentage disparity explained for the null model should be centered on 0, with stochasticity, by design. MSM=men who have sex with men. N/A=not applicable.

***Table 4*: Racial disparity in prevalence of HIV infection generated by specific measured proximal factors, alone or in combination**

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 appendix.

We next considered the power of each factor group in explaining the observed disparity. Differences in the care continuum, *CCR5Δ32* status, and stigma-associated behaviours each explained a substantial disparity. However, the majority-of-sexual-behaviours scenario did the opposite, yielding substantially lower prevalences in black than in white MSM. Racial differences in residual determinants (eg, circumcision, background mortality) yielded only a slight disparity. Most scenarios yielded a prevalence of HIV in white MSM close to observed prevalence, but the majority-of-sexual-behaviours scenarios overestimated it. Prevalence in black MSM was underestimated by all models.

When factor sets were combined, nearly half the disparity was explainable by biomedical determinants, and about a sixth by care and disclosure factors. The scenario varying all sexual behaviours by race yielded the same magnitude reverse disparity as most sexual behaviours alone (0·39 and 0·40, respectively, with overlapping IQRs). The all-risk-factors-in-black MSM scenario, which assessed a priori race-specific factor values that would favour higher prevalence in black than in white MSM, yielded a large disparity, and was the only scenario to include individual runs with a disparity within the observed data's confidence interval. The various factor sets generally yielded prevalences in white MSM close to observed prevalence, except for the all sexual behaviours scenario. Some factor sets yielded prevalences in black MSM closer to observed prevalence than factor groups individually; all were still underestimates, however.

In a scenario probing the misclassification of risk behaviours, in which all values were as observed by race but black MSM were assigned values reported by white MSM for most sexual behaviours, mean prevalence in black MSM closely matched the observed 43·4% prevalence. However, prevalence in white MSM was higher than observed, generating a smaller-than-expected disparity.

Discussion

We used data from Atlanta-based studies focused on racial disparities in HIV prevalence and a previously described model structure[17] to assess whether the hypotheses that have been put forward in scientific literature (average differences in networks of sexual relationships, sexual behaviours within relationships, the care cascade, disclosure, and *CCR5* genotype frequencies) are sufficient to explain the observed disparities. We found that these hypothesised explanatory factors accounted for, at most, 55·5% of observed disparities (table 4).

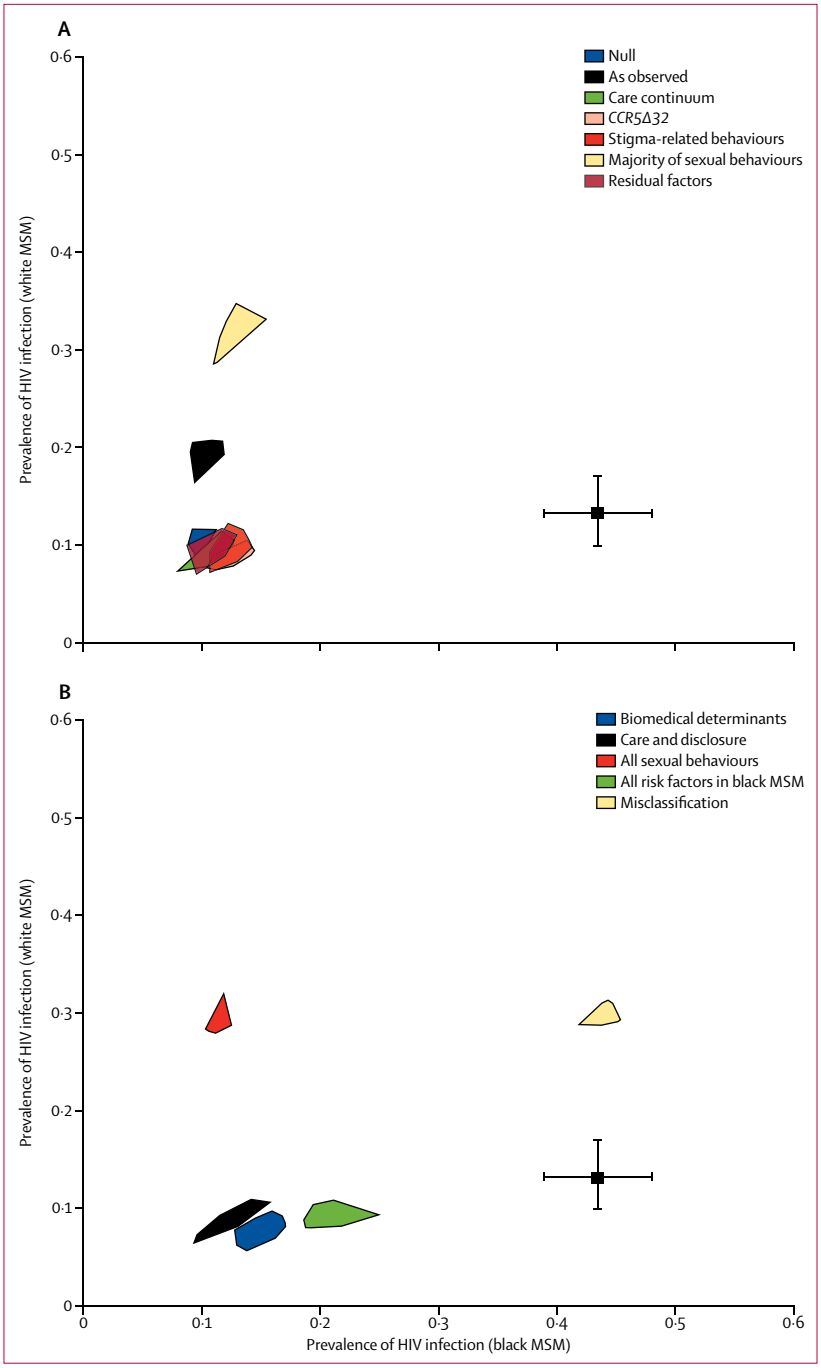

***Figure 2*: Prevalence of HIV in black *vs* white MSM in the first seven secnarios (A), and final five scenarios (B)**
This figure provides visual insight into whether the challenges in explaining observed disparities in HIV prevalence between black and white MSM are due to systematic underestimation of prevalence in black populations, overestimation in white populations, or both. Observed point estimates are shown with 95% CIs. Parts (A) and (B) are divided for reasons of legibility only. Similarly, for the sake of legibility, individual runs are not shown; shapes represent the convex hull of 16 points reflecting individual runs. Scenario explanations are listed in table 3. Many scenarios approximate prevalence in white MSM; however, the first 11 scenarios depicted all strongly underestimate prevalence of HIV in black MSM. This finding supports consideration of a scenario incorporating misclassification of some risk behaviours by black MSM as a result of potential stigma or mistrust of research. This final scenario captured prevalence in black MSM, but overestimated prevalence in white MSM. MSM=men who have sex with men.

All else being equal, race-assortative mixing patterns in MSM cannot generate or sustain a racial disparity, let alone promote increased disparities over time. Our findings suggest that race-assortativity, along with pre-existing prevalence disparities, are an insufficient explanation for population-level race disparities in incidence of HIV infection. However, the lack of population-level explanatory ability is different from the explanatory ability of prevalence among partner populations to predict short-term race-specific individual-level risk. In incidence studies,[13] prevalence of HIV infection in specific partner pools (eg, black partners,[13] older partners[12]) remains a strong individual predictor of HIV acquisition. We urge that such associations not be used to stigmatise groups with higher prevalences but rather to reinforce the urgent need for approaches that improve the health of everyone living with, or at risk of, HIV, while respecting the dignity of all affected communities.[4,13,23]

Low engagement in HIV care engagement and low attainment of viral suppression in black MSM explained a meaningful proportion of disparities in prevalence. Other researchers 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 of achieving the US National HIV/AIDS Strategy goal of increasing access to care, which would help to achieve the goals of reducing new infections and HIV-related disparities.[24] Prospective studies are needed to understand more fully the 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 *CCR5Δ32* mutation had a large potential role in disparities in prevalence between black and white MSM. This role hinged on the inclusion of a partly protective effect for the heterozygous genotype. A meta-analysis of predominantly small, diverse studies[26] did not show a consistent protective effect for heterozygosity, but importantly did not include the only race-focused study of US MSM that informed our parameter.[8] The immutable nature of genetic differences presents a programmatic and messaging challenge. Because other groups with lower prevalence (eg, Asian Americans) also lack deletion genotypes, the ability for genetic differences alone to produce HIV disparities is probably limited, but we have shown their potential to operate in concert with other factors to yield substantial disparities.[6]

Relatedly, we did not consider STIs to be biological cofactors, given the additional model complexity and overlap with causal and associative mechanisms for HIV that would result. Results from Involvement showed high and similar racially disparate incidences of gonorrhoea,

chlamydia, and syphilis, and statistical modelling suggested that rectal STIs explained 15% of subsequent HIV infection.[4,27] In that work, increased HIV transmission from co-infected MSM was not considered. Models incorporating STIs might be expected to yield greater racial disparities in prevalence of HIV, and are in development.

Our analysis provides several new insights into the role of risk behaviours in epidemics in MSM. Modelling only the set of risk behaviours at observed levels, which are generally reported less commonly in black than in white MSM, produced an inverse disparity. That this disparity was nearly identical to the scenario with all factors as observed shows the strong leverage for risk behaviours in determination of disparities, which masks the role of biomedical factors in creation of disparities. When modelling equal, rather than lower, behavioural risks for black and white MSM, the direction of disparity was reversed and population-observed prevalence in black MSM was obtained, suggesting that under-reporting could potentially help to explain disparities. Racial differences in reporting have been discounted because of the qualitative consistency of reports across time and geography, but validation studies have shown under-reporting.[6,11,21,22] Further understanding of the social determinants of under-reporting and development of risk biomarkers in studies in MSM are needed.[28] Two behaviours that are postulated to be influenced by stigma (reduced HIV serodiscussion and shorter partnership duration) worked differently from other behaviours. When considered separately, they yielded a modest disparity, showing a mechanism through which stigma can shape HIV risk in black MSM.

Our model did not explain the full magnitude of disparities reported in surveillance and research studies, and we suggest several possible sources not accounted for in our model, beyond STIs. We incorporated the only published, nationally representative estimates of care engagement among MSM by race, derived from US Centers for Disease Control and Prevention data sources, because relevant Atlanta-specific data were unavailable. Such Atlanta-specific data might show greater racial differences in engagement and viral suppression. This lack of data emphasises the need for improved care continuum surveillance for all racial groups of MSM at all jurisdictional levels. We modelled race-assortative mixing probabilities as uniform within race; individual heterogeneity in the propensity for interracial partnerships, attributable to residential or cultural segregation, could 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, for which 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 with populations of 5000 and 20 000 (data not shown), and the results were consistent with our findings.

Because we focused on MSM aged younger than 40 years, disparity convergences were faster than would be observed in reality. We did not model HIV pre-exposure prophylaxis, an effective biomedical prevention modality, because observed disparities pre-date regulatory approval. Elsewhere we extend our framework to consider pre-exposure prophylaxis,[30] with subsequent work addressing its effect on racial disparities.

High incidence of HIV infection among black MSM is one of the most pressing public health concerns in the USA and a national priority.[24] Our model shows the roles of access to effective HIV care, biological susceptibility, and behaviours in influencing this risk. We show that some residual sources of disparity remain unexplained and require further exploration if health equity is to be achieved. Programmes and policies must emphasise expanded access to antiretroviral therapies for HIV treatment and prophylaxis, while seeking to alleviate underlying social determinants and stigmas that shape black MSM'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.

Contributors

SMG led the design and coding of the model, did some of the simulations, led the analysis of the simulations, and co-led writing. ESR contributed to the design of the model, led the analysis of the source data to parametrise the model, and co-led 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 parametrise the model. SES did some of the simulations and contributed to analysis of the results. GAM contributed to the conceptualisation of the question, analysis, and writing. PSS contributed to the design of the model, analysis of the source data and simulations, and writing.

Declaration of interests

SMG, ESR, SMJ, NL, SES, and PSS report grants from the National Institutes of Health during the study. ESR received personal fees from Medidata and Cengage Learning outside the submitted work. PSS also received grants and personal fees from the Centers for Disease Control and Prevention, grants from MAC AIDS Fund, and grants from Gilead outside the submitted work. GAM declares no competing interests.

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