Bayesian Approaches to Racial Disparities in HIV Risk Estimation Among Men Who Have Sex with Men

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Background: Men who have sex with men (MSM) continue to be overrepresented for new HIV infections compared with non-MSM. This disparity becomes even more alarming when considering racial groups. We describe the race-specific effects in HIV prevalence among MSM relative to non-MSM and explore the causes of disagreement among estimates.

Methods: We used data from the National Epidemiologic Survey on Alcohol and Related Conditions, a nationally representative longitudinal survey conducted in the US Bayesian learning corrected for potential misclassification of MSM status and adjusted for residual confounding, hypothesized to explain the MSM racial disparity in HIV. We articulated the structure and strength of the latent confounders that would make race-specific risk gradients equivalent.

Results: Compared with non-MSM, the adjusted prevalence odds ratio (POR) and 95% credible interval for black MSM having self-reported HIV infection was 5.8 (2.0, 16), while the POR for white MSM was 12 (4.2, 31). For all MSM, the POR for HIV infection was 9.3 (3.6, 23) with black men having 2.6 times the odds of prevalent infection compared with white men.

Conclusions: The observed race-specific associations in MSM are likely not due to misclassification alone, but represent a constellation of factors that differ between racial groups. We recommend specific risk factors in surveys needed to further identify the behavioral characteristics that lead to the observed differences when the estimates are stratified by race.

(*Epidemiology* 2017;28: 215–220)

Men who have sex with men (MSM) in the United States continue to be at a disproportionally higher risk for being infected with the human immunodeficiency virus (HIV) compared with non-MSM. As of 2014, the Centers for Disease Control and Prevention (CDC) estimated that over 1.2 million persons nationally ages 13 years and older were infected with HIV, with approximately 50,000 new cases occurring annually.[1] Within these incident cases, for 2010, 29,800 (60%) were among MSM[1] despite national estimates of MSM representing only about 2% of the US population ages 15 to 44 years.[2] Compared with non-MSM, this translates to a 38- to 75-fold greater risk for HIV infection among MSM.[3] Notwithstanding well-known prophylaxis against transmission of the virus, the infection remains epidemic in MSM due to both risk-taking behavior (condomless anal intercourse) and social sexual networks (greater exposure to risk).[4]

The Racial Disparity Conundrum

When examined across racial groups, the HIV disparity for MSM becomes even more alarming. According to the 2014 CDC data, while the annual incidence of HIV in black and white MSM was about 11,000 new cases in the United States for each race,[1] in the general population white men outnumbered black men approximately five to one.[5] A population-based study of MSM and HIV conducted in the southern United States found that black MSM were nearly five times as likely to be living with HIV compared with white MSM, and while one in 22 white MSM was infected with HIV, the ratio was one in five for black MSM.[6] In addition, due to greater stigma of self-identification of MSM in the black community,[7] the number of black MSM is likely underestimated, and therefore the degree of disparity may not have been accurately captured by use of self-identification in the risk assessment.

Understanding this disparity requires identification of the factors that affect HIV seropositivity (i.e., being infected) and risk-taking behavior (i.e., the propensity to be infected), as well as assessment of whether the factors differ across MSM racial groups. In two separate meta-analyses of HIV risk factors among black and non-black MSM, Millett et al.[8,9] identified notable, and perhaps paradoxical, racial differences. While black MSM, compared with non-black MSM, were more likely to report HIV infection (OR: 3.0, 95% CI: 2.1, 4.4), they were also more likely to practice certain HIV-protective behaviors (OR: 1.4, 95% CI: 1.2, 1.6), including fewer sex partners (OR: 0.6, 95% CI: 0.5, 0.9), lower substance use (OR: 0.7, 95% CI: 0.5, 1.0), and less anal intercourse without

Editor's Note: A commentary on this article appears on p. 221.
Submitted 10 December 2015; accepted 19 October 2016.
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Financially supported by DHHS Office of Minority Health (BAAMHS study).
This paper was selected for the Tyroler Student Prize Paper Award for the 2016 Epidemiology Congress of the Americas.
The authors report no conflicts of interest.
SDC Supplemental digital content is available through direct URL citations in the HTML and PDF versions of this article (www.epidem.com).
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ISSN: 1044-3983/17/2802-0215
DOI: 10.1097/EDE.0000000000000582

a condom (OR: 0.6, 95% CI: 0.5, 0.7). Yet there were notable characteristics positively associated with HIV infection, such as more sexually transmitted infections (OR: 6.0, 95% CI: 5.4, 6.7), and lower use of anti-retroviral therapy when HIV positive (OR: 0.4, 95% CI: 0.3, 0.6). In addition, while black MSM disclosed fewer male sex partners, homosexual or gay identity, HIV status, and substance use, this may be a manifestation of social stigma.[7,9]

Sociodemographic (older age, lower income, being in a relationship, unknown partner HIV status) and psychosocial (distress, social support) correlates of HIV infection are also differently distributed in black compared with non-black MSM.[10,11] Although a biologic basis has been hypothesized,[10] research has not substantiated this.[9,11] "Poorly conceived and operationalized" measures of socioeconomic status are believed to be one of the primary drivers of the reported racial disparity rather than a genetic component.[12] In summary, myriad factors may contribute to the observed racial disparity. Given the biology of infection is likely the same, the difference in risk estimates across racial groups may be due to some unmeasured or poorly measured confounder, after controlling for the established risk factors previously enumerated.

Bayesian Approaches for Correcting Misclassification and Residual Confounding

Goldstein et al.[13] described bias that arises in estimate of risk of self-reported HIV when sexual behavior is assessed through proxy variables, and applied Bayesian techniques to correct for such misclassification. This work was limited to black and African American men. Building on this work, we sought to apply Bayesian techniques in a large US-based survey for correcting MSM status as a risk associated with HIV infection, stratified by racial group: black or African American and white. We specifically evaluated associations of MSM behavior with HIV infection status (compared with non-MSM behavior) in each racial subgroup to attain a more nuanced understanding of heterogeneity within a broad sample. Our treatment of race as an effect modifier is based on the intersectionality framework[14] and multiple minority stresses due to stigma and discrimination.[15] Historically, surveillance programs studied these groups independently or considered race as a categorical covariate for analysis.

In addition to correcting for purported misclassification of MSM in the survey, we use Bayesian hierarchical priors to adjust for residual confounding[16] suspected to be induced by suboptimal assessment of MSM behavior and other HIV risk factors. The overarching goal of this work was to obtain improved prevalence estimates for race-specific MSM effects for informing interventions and their evaluation.

METHODS

Source and Study Population Selection

Data were retrieved from the Black and African American Men's Health Study (BAAMHS) and the second wave of the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC-2) as has been detailed previously,[13] and will only be briefly described here. We derived validation data from BAAMHS (n = 622) corresponding to the sensitivity (Sn) and specificity (Sp) of using male partner gender to identify MSM behavior (reported anal intercourse), and to determine the prevalence odds ratio (POR) for the association between MSM behavior and self-reported HIV infection. The NESARC-2 data included all non-Hispanic male respondents stratified by racial group as black or African American and white.

Variable Selection

Our proxy of interest was self-reported MSM behavior (available in both BAAMHS and NESARC-2), classified by having a history of male sexual partners, and the primary outcome was self-reported HIV infection. Race was self-reported and classified as black or white. Potential confounders of the MSM and HIV relationship in both BAAMHS and NESARC-2 included being in a recent relationship, having a history of sexually transmitted diseases, having been sexually abused, and recent drug use including narcotics, stimulants, depressants, and hallucinogens.

Bayesian Correction for Misclassification of MSM Behavior

Misclassification of MSM status is adjusted through use of three models: (1) an *outcome* model that represents the adjusted log odds of self-reported HIV infection given true MSM behavior, and adjusted for potential confounding; (2) an *exposure* model that represents the log odds of true MSM behavior; and (3) a *measurement* model that relates the observed MSM behavior to the conditional probability of true MSM behavior by the Sn and Sp of the exposure predictor (allowing for differential misclassification). To clarify, Sn is the proportion of MSM who identify as such in the survey; conversely, Sp is the proportion of non-MSM who identify as such. Details of these and all ensuing models and prior distributions can be found in the eAppendix (http://links.lww.com/EDE/B129).

Bayesian Correction of Residual Confounding

Residual confounding was incorporated into the exposure and outcome models by incorporating an unmeasured (latent) covariate, U.[17] Didactically, this variable represents the spectrum of unmeasured and unknown confounders that influence self-reported HIV positivity. Operationally, we let U_i have differing prevalences among black ($i = 1$) and white ($i = 2$) men, corresponding to the two stratified models. Given the expectation the biology of infection is the same, U_i accounted for the HIV prevalence difference between the race-specific MSM effects. The prevalence and effects of the unmeasured latent confounder(s) were estimated via a sensitivity analysis.[19] Parameters were informed from the Bayesian analyses and candidate values were selected as detailed in the eAppendix (http://links.lww.com/EDE/B129).

Implementing the Statistical Analysis

Bayesian analysis was implemented in R using Markov chain Monte Carlo simulation via Just Another Gibbs Sampler, running 10,000 iterations over two chains, and discarding the first 1,000 observations for burn-in. Model convergence was ascertained via parameter sampling history and Gelman and Rubin's convergence diagnostic. Annotated R code and the convergence plots are available in the eAppendix (http://links.lww.com/EDE/B129).

To detect possible effect modification by race, we conducted separate (stratified) analyses for black and white men in both the frequentist and Bayesian analyses. The Wald test of heterogeneity was used on the stratum-specific adjusted estimates for the MSM log odds in the frequentist analysis.[18]

RESULTS

Description of Study Population

NESARC-2 included 14,564 men of which 2,301 (10%) identified as non-Hispanic black and 8,775 (71%) identified as non-Hispanic white, yielding a final study population of 11,076 black or white men. There were 503 (4%) men who reported male sexual partners and were therefore classified as MSM: 97 (19%) were black and 406 (81%) were white. Overall prevalence of self-reported HIV infection was <1% (n = 57), with three times the proportion in black men (1%, n = 24) compared with white men (0.3%, n = 33). Among MSM, HIV prevalence was 5% (n = 6) in black men compared with 4% (n = 16) in white men. Additional characteristics are provided in Table 1.

Naïve Modeling (Uncorrected Frequentist Analysis)

Table 2 presents analyses that assume perfect measures of covariates without residual confounding. For all men in the study population, the adjusted POR of HIV infection for MSM compared with non-MSM was 10 (95% CI: 5.5, 18). Black men had a three-fold increase in prevalent HIV infection (95% CI: 1.7, 5.2) compared with white men. The stratified adjusted POR of HIV infection for MSM compared with non-MSM for black men was 4.5 (95% CI: 1.4, 12), and for white men was 16 (95% CI: 7.4, 34), an almost four times greater association (Wald test P value = 0.06).

Modeling Through Bayesian Learning (Corrected Analysis)

Table 3 presents the mean and 95% credible intervals (CrI) from the posterior distributions for the Bayesian analysis, corrected for misclassification of MSM behavior and residual confounding. For all men, the adjusted POR of HIV infection for MSM compared with non-MSM was 9.3 (95% CrI: 3.6, 23). Black men had 2.6 times the odds of prevalent HIV infection (95% CrI: 1.5, 4.7) compared with white men. The stratified adjusted POR of HIV infection for MSM compared with non-MSM for black men was 5.8 (95% CrI: 2.0, 16), and for white men was 12 (95% CrI: 4.2, 31). Compared with the naïve analysis (factor of four), the difference between estimates reduced to a factor of two, although there was substantial overlap in the credible intervals.

TABLE 1. Comparison of Key Characteristics of Non-Hispanic Men in NESARC by MSM Status and Stratified by Racial Group, 2004–2005

	Black or African American Men			White Men		
Variable	**MSM**	**Not MSM**	**All**	**MSM**	**Not MSM**	**All**
No. (%[a])	97 (4)	2,204 (96)	2,301 (10)	406 (4)	8,369 (96)	8,775 (71)
HIV/AIDS, n (%)						
No	91 (95)	2,186 (99)	2,302 (99)	390 (97)	8,354 (>99)	8,820 (>99)
Yes	6 (5)	18 (1)	24 (1)	16 (4)	15 (<1)	33 (<1)
In a recent relationship, n (%)						
No	38 (40)	502 (19)	545 (20)	129 (26)	1,418 (13)	1,565 (13)
Yes	59 (59)	1,700 (81)	1,768 (80)	277 (74)	6,945 (87)	7,260 (87)
History of STDs, n (%)						
No	94 (96)	2,197 (>99)	2,301 (>99)	401 (99)	8,324 (>99)	8,762 (>99)
Yes	3 (4)	5 (<1)	8 (<1)	5 (1)	39 (<1)	44 (<1)
History of sexual abuse, n (%)						
No	82 (83)	2,163 (98)	2,253 (97)	353 (87)	8,137 (98)	8,531 (97)
Yes	15 (17)	39 (2)	54 (3)	52 (13)	215 (2)	270 (3)
Recent drug use[b], n (%)						
No	75 (80)	1,997 (89)	2,096 (89)	318 (79)	7,454 (89)	7,844 (89)
Yes	22 (20)	207 (11)	230 (11)	88 (21)	915 (11)	1,009 (11)

[a]Proportions take into account NESARC's multistage sampling, with stratification, clustering, and weighting of the study population.
[b]Including narcotics, stimulants, depressants, and hallucinogens.
STD indicates sexually transmitted disease.

TABLE 2. Adjusted Estimates of Self-reported HIV Infection Associated with MSM Behavior Compared with Non-MSM Among Non-Hispanic Men in NESARC, Uncorrected for Misclassification and Residual Confounding (Frequentist Analysis)

Analysis	POR[a] (95% CI)
All men	
MSM (ref: non-MSM)	10 (5.5, 18)
Black or African American (ref: White)	3.0 (1.7, 5.2)
Stratified by race[b]	
Black or African American MSM (ref: non-MSM)	4.5 (1.4, 12)
White MSM (ref: non-MSM)	16 (7.4, 34)

[a]Adjusted for being in a recent relationship, having a history of sexually transmitted diseases, having been sexually abused, and recent drug use including narcotics, stimulants, depressants, and hallucinogens.
[b]Wald test of heterogeneity of effects P value = 0.06.
CI indicates confidence interval.

TABLE 3. Posterior Mean Adjusted Odds Ratios and 95% Credible Intervals for Self-reported HIV Infection Associated with MSM Behavior Compared with Non-MSM Among Non-Hispanic Men in NESARC, Corrected for Misclassification of MSM and Latent Confounding Using Hierarchical Priors (Bayesian Analysis)

Analysis	Posterior POR[a] (95% CrI)
All men	
MSM (ref: non-MSM)	9.3 (3.6, 23)
Black or African American (ref: white)	2.6 (1.5, 4.)7
Stratified by race	
Black or African American MSM (ref: non-MSM)	5.8 (2.0, 16)
White MSM (ref: non-MSM)	12 (4.2, 31)

[a]Adjusted for being in a recent relationship, having a history of sexually transmitted diseases, having been sexually abused, and recent drug use including narcotics, stimulants, depressants, and hallucinogens.
POR indicates mean of posterior of the distribution of the prevalence odds ratio.

Characteristics of the Latent Confounders

We observed a large discrepancy in POR of MSM and HIV positivity compared with non-MSM when stratified by race. While we considered misclassification of MSM status and residual confounding as possible explanations of this phenomenon, the data argue that these explanations are insufficient. Therefore, the difference in the race-specific POR may be due to extrinsic factors and the sensitivity analysis was a way to quantify the discrepancy in terms of some unknown constellation of factors, U, that are not measured or mismeasured in our analysis. We approached this sensitivity analysis by asking the question: "What sort of factor(s) would make risk in black MSM relative to black non-MSM the same as in white MSM relative to white non-MSM?"

From the Bayesian corrected models in Table 3, we noted that the stratified adjusted POR of HIV infection for black and white men began to converge, although not completely, and may appear to some to be effect modification (Figure, broken lines). However, assuming the biology of infection is the same across racial groups, there should be a single true POR of HIV infection for MSM behavior, irrespective of race and other known risk factors (Figure, solid line). Consequently, we conducted the sensitivity analysis to determine for each race what would change the corresponding POR from the observed value of the mean of the posterior distribution to the hypothesized true POR centered around 9.0 (extrapolated from where the two PORs would overlap in the Figure).

For black men, the unmeasured confounder U_1 would require a strongly positive association with HIV positivity, and greater prevalence in non-MSM versus MSM (Table 4), to move POR to ~9.0. Depending on the strength of the latent confounder, the difference between prevalence of U_1 in non-MSM versus MSM ranged from 4% to 38%. For white men, U_2 would also require a strongly positive association with HIV positivity, with greater prevalence in MSM versus non-MSM (Table 5). The difference between prevalence of U_2 in MSM versus non-MSM ranged from 4% to 54%. Therefore, in black men, U_1 represents negative confounding that attenuated the true relationship between MSM behavior and HIV infection, and in white men U_2 represents positive confounding that exaggerated the true relationship.

DISCUSSION

In this analysis of prevalence of self-reported HIV infection correlated with MSM behavior, we sought to articulate the characteristics leading to divergent race-specific effects, and further correct the corresponding estimates. We found that relative to non-MSM, white MSM have an apparent higher POR for HIV infection compared with black MSM. In addition, this difference persisted even after accounting for misclassification and confounding to the extent the data permit.

Compared with white men, the apparent weaker association of HIV infection due to MSM behavior in black men may appear counterintuitive. However, it can partially be attributed to higher rates of HIV infection in the non-MSM

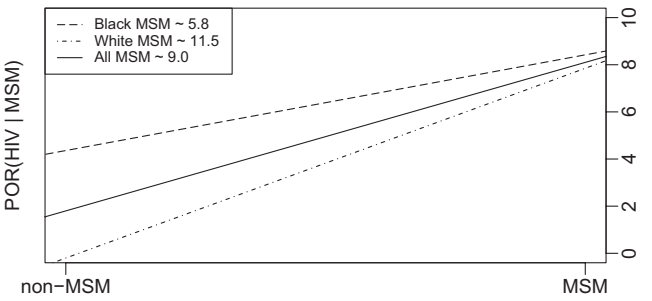

FIGURE. Graphical representation of race-stratified adjusted prevalence odds ratios for HIV positivity comparing MSM with non-MSM, and hypothesized true prevalence odds ratio irrespective of race ("all MSM").

TABLE 4. Sensitivity Analysis of Latent Confounding in MSM and Non-MSM by Black Race in NESARC-2: Strength and Prevalence of Risk Factors to Mitigate Risk Difference of MSM Behavior Predicting HIV Infection Between Non-Hispanic Black and White Men

	Prev(U_1) in MSM					
OR(U_1)[a]	0.0	0.02	0.04	0.06	0.08	0.10
5	0.26[b]	0.30	0.34	0.38	0.44	0.48
10	0.12	0.16	0.20	0.24	0.28	0.32
15	0.08	0.12	0.16	0.20	0.24	0.28
20	0.06	0.10	0.14	0.18	0.22	0.26
25	0.04	0.08	0.12	0.16	0.20	0.26
30	0.04	0.08	0.12	0.16	0.20	0.24
35	0.04	0.08	0.12	0.16	0.20	0.24

[a]OR of the latent confounder predicting HIV infection, adjusted for MSM behavior, "being in a recent relationship," "having a history of sexually transmitted diseases," "having been sexually abused," and "recent drug use."

[b]Prevalence of the latent confounder, U_1, in non-MSM necessary with corresponding strength of U_1 (row) and prevalence of U_1 in MSM (column) to move risk estimate of MSM behavior predicting HIV infection to OR ~9.3.

TABLE 5. Sensitivity Analysis of Latent Confounding in MSM and Non-MSM by White Race in NESARC-2: Strength and Prevalence of Risk Factors to Mitigate Risk Difference of MSM Behavior Predicting HIV Infection Between Non-Hispanic Black and White Men

	Prev(U_2) in Non-MSM					
OR(U_2)[a]	0.0	0.02	0.04	0.06	0.08	0.10
2.5	0.48[b]	0.50	0.52	0.58	0.60	0.64
5	0.18	0.22	0.24	0.28	0.32	0.34
10	0.08	0.12	0.14	0.18	0.22	0.24
15	0.06	0.08	0.12	0.16	0.18	0.22
20	0.04	0.08	0.10	0.14	0.18	0.20
25	0.04	0.06	0.10	0.14	0.16	0.20

[a]OR of the latent confounder predicting HIV infection, adjusted for MSM behavior, "being in a recent relationship," "having a history of sexually transmitted diseases," "having been sexually abused," and "recent drug use."

[b]Prevalence of the latent confounder, U_2, in MSM necessary with corresponding strength of U_2 (row) and prevalence of U_2 in non-MSM (column) to move risk estimate of MSM behavior predicting HIV infection to OR ~9.3.

referent groups. CDC surveillance data indicated 16% of new HIV infections in black men occurred from heterosexual contact compared with 5% in white men, a three times greater incidence.[20] Other contributing factors to the discrepancy are likely behavioral. Millet et al.[8,9] identified specific relevant factors that differed by MSM racial groups. Our analysis builds upon this work by articulating the magnitude and prevalence of such behaviors quantitatively, thereby allowing for a more directed search for the root causes, and adjustment of estimates when these factors cannot be measured.

The evidence for the conjecture of both negative and positive confounding underscores the importance of modeling via a stratified approach. Namely, in black men, non-MSM may be more likely to engage in HIV risk-taking behaviors compared with MSM, while in white men, MSM may have an additional set of risk factors conferring excess HIV risk compared with non-MSM.

In searching for possible causes of the negative confounding, we speculate that racial differences of injection heroin use, a correlate of HIV infection, may be a suitable candidate.[21] In addition to black injection drug users having greater risk of HIV infection compared with white injection drug users,[22] prevalence of heroin in NESARC-2 was greater among black non-MSM versus MSM,[23] both of which, when not taken into account or when mismeasured in the statistical analysis, could attenuate the association of black MSM with HIV positivity. As observed, controlling for the latent confounding strengthened the POR, although not completely setting it equivalent to white MSM, indicating excessive residual confounding.

As an example of positive confounding, previous research has suggested that white MSM are more likely to use stimulants such as methamphetamines,[24] and postuse of these to have condomless receptive anal intercourse, increasing risk for HIV infection.[25] Amphetamine use in NESARC-2 was two times more prevalent in white MSM versus non-MSM, and white MSM reported over seven times greater use compared with black MSM.[23] This risk factor when unaccounted or mismeasured would tend to exaggerate the prevalence ratio comparing white MSM to non-MSM, that is, positive confounding. When we accounted for the latent confounder in the analysis, we saw the POR attenuate, yet again not completely setting it equivalent to black MSM.

Our approach to correcting residual confounding rests on the assumption that observed confounders in the MSM and HIV relationship capture some salient features of unobserved confounders—this is inherent in the hierarchical Bayesian method applied. As none of the measured confounders were strong enough (in their association with HIV infection or race) to produce the effects hypothesized to exist if race is not an independent cause of HIV positivity, we ruled out that the observed racial disparity is due to misclassification or measurement error. Instead, the race-specific MSM effects must have arisen due to a constellation of factors that differ between racial groups, possibly related to chronic minority stresses resulting in internalized and externalized differences. Consequently, the HIV racial disparity among MSM cannot be satisfactorily modeled in the data available to us, and further insight will require new data that accurately capture differences in relevant risk factors by race. Surveys must specifically address behavioral differences that can lead to HIV infection for MSM. In addition, when effect modification is expected a priori, as noted by others, relying on the statistical result of a hypothesis test may be subject to Type II error: a false negative.[18] Rather the researcher needs to consider the magnitude of the stratified estimates and be more fluid in interpretation of hypothesis testing.[18,26]

There are several limitations to this study. First, data from NESARC-2 were self-reported and recorded with an interviewer present, potentially resulting in under-reporting of stigmatizing behaviors and conditions,[27] such as being MSM and HIV positive. While our analysis accounted for misclassification of MSM, the outcome is self-reported HIV infection rather than true serostatus. Second, our modeling of the unmeasured confounder as a single variable limits the ability to infer its individual characteristics rather than the net sum, akin to latent class analysis. Third, we adjusted for misclassification of white MSM behavior using priors derived from a black population. There may be greater stigma for self-identifying as MSM in the black community (thus more false negative responses to the male partner question);[9] however, when we increased the prior on sensitivity, the prevalence PORs did not meaningfully shift. Finally, NESARC-2 employed a complex survey methodology that was not factored into the Bayesian analysis, as we are not aware of applicable methods. Results need to be interpreted within the context of the study sample rather than generalized to the US population.

In summary, the racial disparity conundrum persisted despite our efforts to statistically account for uncertainty from misclassification and residual confounding. Exposure to the virus clearly differs based on behavioral differences, likely due to missing information in our data about racial disparities. We have demonstrated that partially accounting for these behavioral differences begins to mitigate the race-specific MSM effects, yet better data are needed to fully understand the apparent difference.

ACKNOWLEDGMENTS

We thank John L. Peterson, Department of Psychology, Georgia State University, for a helpful review of a draft version of this manuscript.

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