Master's in Public Health Capstone

Reproducing a Study on Racial and Ethnic Disparities in HIV and STIs in the United States

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Honor Pledge

I, Anjana Renganathan, have neither given nor received unauthorized assistance (as detailed in the Brown School student handbook) in the completion of this work. I certify that this work is authentically my own.

Abstract

Rates of sexually transmitted infections have grown over the past decade. Racial and ethnic disparities, alongside social determinants of health, result in higher disease burdens for racial and ethnic minorities. This is costly financially, emotionally and socially for the infected individuals, their communities and the government.

In this paper, we attempt to reproduce a study utilizing NHANES data from 1999 to 2012 to examine racial and ethnic disparities in STI and HIV rates. Reproducibility and replicability have become increasingly important in all fields of academic research and should be applied consistently and thoroughly to public health research as well. As such, while reproducing the original study, we aim to measure our success while pursuing best practices.

R Studio was used to calculate weighted prevalence and multivariate logistic regressions stratified by gender and race/ethnicity. Statistically significant differences in prevalence and odds ratios of STIs between racial/ethnic groups were recreated. Prevalences were successfully reproduced, but the multivariate logistic regressions differed in some elements. When examining markers of reproducibility, the mean percent difference for the weighted prevalence was -0.4% ± 6.0%, while the odds ratios had a higher percent difference of 1.0% ± 33.5%. 5 odds ratios flipped in statistical significance, but the overall precision of the multivariate logistic regression increased with the reproduction.

This study roughly reproduced the disparities in sexual health found in the original study, further emphasizing the need for culturally competent care, efficient programming and comprehensive sexual education to address this unmet need.

Keywords: social determinants of health, STIs, HIV, racial disparities, reproducibility, public health

Introduction

A report from the United States Center for Disease Control shows that sexually transmitted infections (STIs) have increased for the fifth uninterrupted year to an all-time high in 2018 (HHS et al., 2018). STIs can be caused by viral, bacterial, or parasitic pathogens, and may or may not be treatable depending on the infection. The cost, consequences, and stigma associated with the infection differ as well, from cheap, common antibiotics easily taken and hidden from friends and family to expensive chronic care of a stigmatized disease. Common STIs include chlamydia, gonorrhea, syphilis, human immunodeficiency virus (HIV), herpes simplex virus, genital warts, and hepatitis C infections, each of which are explained further in Appendix E.

While the overall increase of STIs in the past few years is alarming, it should also be noted that certain subpopulations are highly impacted by it. When compared to the white population, African Americans, American/Indians, and Hispanic populations tend to have up to five times higher rates of most STIs (HHS et al., 2018). This undue burden of disease is often a notable presentation of the social determinants of health. After all, sex, while private, does not occur in a vacuum. Alongside bodies, sexual partners bring socialized norms and practices to their bed, from opinions on condoms to number of past sexual partners to past sexual and health education. These norms and practices can be traced back to larger social determinants of public health that overlay not only STIs, but the overwhelming majority of diseases. Social determinants of health are economic, physical, cultural, social, structural, and organizational variables that can impact a person's health and health outcomes and include factors like health insurance status, healthcare access, language barriers, income level, social norms, housing security, rurality, and discrimination (Dean & Fenton, 2008).

However, even when controlling for variables measuring social determinants of health, race and ethnicity remain as factors that significantly affect the prevalence of an STI. This is not due to genetic or biologic factors but is rather due to the society and history of the United States and how it shapes the treatment of people today. Racism, and its impact on the health of people who experience it, can exacerbate previously mentioned social determinants of health. This includes reduced access to employment, housing and education, increased exposure to adverse risk factors, adverse cognitive and emotional effects, increased allostatic load, further weathering and lower engagement in healthy behaviors, as well as increased participation in unhealthy

coping behaviors(Brondolo, Rieppi, Kelly, & Gerin, 2003; German & Latkin, 2012; Nazroo, 2003; *No Title*, n.d.; Paradies et al., 2015; Sanders-Phillips, 2009). This can and does cause pain, suffering, shame and embarrassment, as well as higher risk of STIs and HIV, implying an easier pathway to complications from STIs, including infertility, chronic pain, miscarriage or premature delivery (Brooke Steele C, Richmond-Reese V, 2006; HHS, CDC, Oid, NCHHSTP, & DSTDP, 2018). All this, in populations that may already fear and distrust health care institutions due to historical mistreatment and exploitation, as in the Tuskegee Syphilis experiments carried out by the CDC (Scharf et al., 2010). Additional barriers to treatment include lack of sexual education, lack of transportation, loss of pay from taking time off, provider bias, and a significant difference in quality of care for minority patients (Brooke Steele C, Richmond-Reese V, 2006).

The effects of infection, diagnosis and treatment can ripple out into lost hours of productivity, lost hours of salary, and a preventable burden on health care institutions and the United States government. The total estimated lifetime medical cost of the 19.7 million cases of STIs occurring in 2009 was to the tune of 15.6 billion dollars (range 11-20.6 billion)(Owusu-Edusei et al., 2013). These preventable diseases, occurring in our most vulnerable populations and racking up billions in cost to individuals and the government, should be more strongly examined and unraveled to better understand who, how and when to take preventative measures and treatment to cut the chain of infection. In order to better serve these communities, both public health practitioners, health care institutions and policy makers should be updated regularly and reliably on the prevalence of these preventable, but easily life altering diseases.

A study by Operario et al. published in 2015 examined the racial and ethnic disparities in HIV and STIs in the United States, utilizing the National Health and Nutrition Examination Survey from 1999 to 2012(Operario, Lee, Kuo, & Zaller, 2015). It has since been cited 9 times solely within the scope of Google Scholar and is an excellent example of the utility of epidemiological data as provided by the NHANES survey to examine not only national health, but the shifting scene of heavily prevalent diseases like STIs within it.

However, as with all academic research, we should always be careful and critical when accepting research conclusions. Reproducibility and replicability of research have surged to the forefront of academic minds since the early 2000s. The “reproducibility crisis” can roughly be said to have kicked off with the publication of a methodologically sound and rationally insane study with strong evidence that all humans have some level of extrasensory perception, or ability

to see into the future by a researcher at Cornell (Bem, 2011). Since then, research replication and reproducibility has been tested in a variety of fields from psychology to economy to cancer biology research, with varying reproducibility rates of 36% to 61% to 11% respectively (Begley & Ellis, 2012; Camerer et al., 2016; Open Science Collaboration, 2015). One survey by Nature found that more than 70% of researchers across a variety of fields tried and failed to reproduce someone else's research, and that more than 50% had failed to replicate their own(Baker & Penny, 2016). Nonetheless, trust in published literature remains high, at around 73%, of researchers believing that existing literature is reliable. This is especially concerning when combined with a publication bias that sees a little less than double the journal acceptance rates for successful reproduction studies compared to failed ones(Baker & Penny, 2016).

The idea of reproducibility and replicability in public health services and systems research is even more recent, only spanning across the past half-decade. In a study examining 6 public health studies with clear descriptions of data management, analyses and statistical methods, the researchers found that most reproductions were consistent with their original studies, but that there were inconsistencies in transcription of results and methodology details omitted (Harris, Wondmeneh, Zhao, & Leider, 2019). Another study based on a survey of American Public Health Association members found that only 14% of respondents shared their statistical coding, datasets or both (Harris, Johnson, Carothers, Combs, Luke, et al., 2018). The same study reported that, if the researcher relocated to another institution, their colleagues would struggle to find their data (33%) or code (43%)(Harris, Johnson, Carothers, Combs, Luke, et al., 2018). These two studies eventually cumulated in the production of five modules, named the Reproducible Research Toolkit, for the coding2share project (Harris, Johnson, Carothers, Combs, Wang, et al., 2018).

This toolkit is an invaluable resource for researchers and the public health academic community alike, as utilizing these measures can shorten time required for scientific research, facilitate sharing and synergy of ideas and reduce research waste (Harris, Johnson, et al., 2019). Improving reproducibility can also result in fewer errors, and more citations(McKiernan et al., 2016; Piwowar, Day, & Fridsma, 2007).

The objective of this capstone is to complete a reproduction of a study on racial and ethnic disparities in HIV and STI rates in the United States that uses publicly available NHANES data from 1999 to 2012. The tables of weighted prevalence and a multivariable logistic

regression analysis will be completed with the available information in the paper. In doing all this, I hope to not only examine the relationship between racial and ethnic disparities in HIV and STI rates in the United States, but to explore the practice and importance of reproducibility in public health studies.

Methods

The methodology and statistical analysis of the original paper was mimicked as much as feasibly possible. Publicly available, deidentified data from the National Health and Nutrition Examination Survey (NHANES) was collected from the years 1999 to 2012. Due to its public availability, IRB approval was not required. NHANES is a nationally representative survey of the civilian population of the United States undertaken by the Centers for Disease Control and Prevention. It has a complex survey design involving oversampling, survey nonresponse and post-stratification that is described thoroughly in CDC documentation, with additional modules on analysis displayed on the NHANES website (Centers for Disease Control and Prevention & National Center for Health Statistics, 2019; Johnson et al., n.d.). The survey includes both a questionnaire component and a smaller clinical examination phase where biospecimens like urine and blood were collected and later analyzed.

In total, seven survey cycles spanning two years each were utilized in this study. The variables included self-reported sociodemographic data from the questionnaire component like race/ethnicity, age, education level, family income, marital status, place of birth, possession of health insurance, health care access and occupation. Sexual health variables from the questionnaire component collected were self-reported chlamydia, gonorrhea, herpes, genital warts, and HIV testing. Finally, a new variable named ‘ever had STI’ was created, where participants who self-reported any STI were grouped together. Finally, variables from the clinical examination phase included HIV antibody, urine chlamydia, HSV-2, and hepatitis C antibody. STIs like HPV and syphilis, which are included in the NHANES survey, were excluded from the study because their data collection did not span across the entire timeframe. The sample included only the participants gave data for all the covariates and completed both the questionnaire and clinical examination components. As such, there was an incidental age restriction to adults between 20-49 years. The final survey sample included 19,510 participants.

The original research team was contacted prior to the statistical analysis for further details on the data management, statistical software and packages used, as well as any other

unknown processing that may have occurred. However, the statistician for the project was unavailable and uncontactable, due to international travel and the five years that had passed since the original study.

Data management included 'passing' over NA or missing variables, recoding family income into a binary (>20k, <20k), recoding place of birth into a binary based on survey cycle 2007-2009 (Born in States or DC, Others), recoding health care access into a binary (Yes, No) and vertically merging directly comparable variables between years like health insurance status and occupation. A new weighting variable spanning 14 years was calculated based off 'WTMEC4YR' for the 1999-2000 and 2001-2002 cycles and 'WTMEC2YR' for the remaining cycles. This was due to differences in length of weighting variable years before and after 2002, and is described more thoroughly in NHANES modules (Centers for Disease Control and Prevention & National Center for Health Statistics, 2019).

Statistical analysis was done utilizing R Studio (version 1.1.463), with significant use of the packages RNHANES, survey, dplyr, and jtools (Jacob & Long, 2020; Lumley, 2020; Package, 2020; RStudio Team, 2016; True, 2016). The analyses were separated by gender. A table of weighted prevalence of each STI, self-reported or laboratory tested was completed. A multivariable logistic regression analysis was then performed separately on males and females, adjusted for race/ethnicity, age, education, family income, marital status, place of birth, possession of health insurance, health care access and occupation. The reference group was non-Hispanic, white, a high school graduate or equivalent, married, born in the United States or DC, had health insurance, had health care access, worked at a job or business and had a family income over $20,000.

Methodology for testing the reproducibility of the paper was taken from a paper by Harris et al in 2018(Harris, Wondmeneh, et al., 2019). For descriptive statistics, with or without a bivariate analysis, the mean percent difference between the original and reproduced values was calculated. For multivariate inferential statistics, the original and reproduced regressions were compared on effect size, statistical significance and precision, or width of the confidence interval. Effect size was also determined as the mean percent different between reproduced and original values. Assessing statistical significance involved determining if any statistical tests flipped in significance between the original and the reproduction. Precision was assessed as the width of the confidence interval produced by the logistic regression. Finally, the original study

was examined for what details could have possibly improved the reproducibility of the paper. The Reproducibility Toolkit was fully utilized for this paper as well (Harris, Johnson, Carothers, Combs, Wang, et al., 2018). The statistical analysis and codebook are made available on the author's Github, as seen in Appendix D. The core public health and biostatistics/epidemiology competencies are addressed in Appendix B, while the project timeline is available in Appendix C.

Significant differences in methodology between the original study and the reproduction include the use of R Studio and the 'survey' package, instead of STATA and 'svy', as well as absence of the 'regular health care provider' variable which was not found in any NHANES datasets. Other possible differences include how the variables were coded or recoded.

Results

The weighted prevalence estimates by racial/ethnic group for HIV and STI rates were reproduced successfully in Tables 1a and 1b. When examining markers of reproducibility, the mean percent difference across both tables was -0.4% ± 6.0%. As in the original paper, for both males and female, the difference in prevalence of self-reported and lab tested STIs and HIV was statistically significant when comparing by racial/ethnic groups, with the exclusion of self-reported herpes for men and hepatitis C antibodies in women. Males across all racial/ethnic groups had higher prevalence for laboratory tested evidence of HIV, chlamydia, and Hep C when compared to females. The only notable exception was HSV-2 which was half the size of the prevalence in women. This trend reversed when looking at self-reported variables, with women reporting higher prevalence for all lifetime STIs, and lifetime HIV testing.

When looking at the US male population alone, African American and White men had the highest prevalence of self-reported lifetime STIs, at 7.2% and 7.3% respectively. African American men had the highest prevalence of HIV antibodies, urine chlamydia, and HSV-2, as well as self-reported lifetime gonorrhea, chlamydia, genital warts and HIV testing across all racial/ethnic groups. When comparing Hispanic and White men, the latter had a higher prevalence for HIV antibodies, urine chlamydia, and HSV-2, as well as self-reported lifetime gonorrhea and chlamydia. Finally, the Mixed and Other racial/ethnic group for men had a much smaller sample size, but even so had a higher prevalence of self-reported lifetime chlamydia than White males. White males had the highest prevalence of self-reported lifetime genital warts

across all racial/ethnic groups. Self-reported lifetime herpes did not differ in a statistically significant way across racial/ethnic groups in males.

For the US female population, African American females had the highest prevalence of self-reported lifetime STIs at 16.7%. African American women also had the highest prevalence for HIV antibodies, urine chlamydia, and HSV-2, as well as self-reported lifetime gonorrhea, chlamydia, herpes, and HIV testing across all racial/ethnic groups. When comparing Hispanic and White women, the latter had a higher prevalence of urine chlamydia and HSV-2, as well as self-reported lifetime gonorrhea and chlamydia. Compared to White females, Mixed and Other females had a higher prevalence of HIV antibodies and urine chlamydia, as well as self-reported lifetime gonorrhea and chlamydia. White females had the highest prevalence of self-reported lifetime genital warts across all racial/ethnic groups. Finally, Hepatitis C did not differ in a statistically significant way across racial/ethnic groups in females.

The male and female adjusted multivariate logistic regressions examining racial/ethnic disparities for HIV and STI rates were not reproduced exactly from the original study, as seen in Tables 2a and 2b. However, the effect size, effect direction and statistical significance was mostly preserved between the original regression and reproduction. The logistic regressions all met the assumptions of independent observations and linearity when it came to age. However, there was strong evidence of multicollinearity, especially for race/ethnicity, education and marital status across many of the models.

In the reproduced regression, African American males had 4.63 times the odds of testing positive for HIV antibodies when compared to white males (OR 4.63, 95% CI: 2.07-10.40). African American males were also more likely to have higher odds of testing positive than white males for urine chlamydia (OR 3.85, 95% CI: 2.31-6.42) and HSV-2 (OR 4.69, 95% CI: 3.87-5.68). Hispanic males had higher odds than white males of testing positive for HIV antibodies (OR 2.80, 95% CI: 1.23-6.37) and HSV-2 (OR 1.50, 95% CI: 1.16-1.94). Mixed males had a fourth of the odds of white males for testing positive for urine chlamydia (OR 0.25, 95% CI: 0.07-0.99). For self-reported lifetime STIs, African American males had higher odds than white male of reporting gonorrhea (OR 6.45, 95% CI:2.22-18.79), chlamydia (OR 2.52, 95% CI: 1.25-5.10), and lifetime testing for HIV (OR 2.06, 95% CI: 1.81-2.35). They had half the odds of white men for self-reporting genital warts (OR 0.48, 95% CI: 0.32-0.71). Hispanic men also had lower odds than white men for self-reporting genital warts (OR 0.59, 95% CI: 0.36-0.96).

Finally, mixed males had lower odds than white males for self-reported lifetime HIV testing (OR 0.72, 95% CI: 0.57-0.90).

African American females, when compared to white females, had 50.98 times the odds of testing positive for HIV antibodies (OR 50.98, 95% CI: 6.53-397.78). African American females also had higher odds than white females of testing positive for urine chlamydia (OR 4.57, 95% CI 2.55-8.18) and HSV-2 (OR 5.61, 95% CI 4.81-6.51). Hispanic females had higher odds than white females for testing positive for HSV-2 (OR 1.35, 95% CI 1.11-1.64). Finally, mixed women had nearly triple the odds of testing positive for urine chlamydia compared to white women (OR 2.97, 95% CI 1.33-6.62). African American women had higher odds than white women of self-reporting any lifetime STI (OR 1.30, 95% CI: 1.08-1.57), gonorrhea (OR 3.99, 95% CI: 1.28-12.37), chlamydia (OR 3.55, 95% CI 2.03-6.21), herpes (OR 1.51, 95% CI: 1.17-1.96), and HIV testing (OR 2.33, 95% CI 1.98-2.74). Hispanic women had about half the odds of white women of self-reporting lifetime genital warts (OR 0.51, 95% CI: 0.34-0.77). Finally, mixed women had lower odds than white women of self-reporting lifetime HIV testing (OR 0.75, 95% CI 0.58-0.96).

The reproducibility of the multivariate logistic regression was examined with effect size, significance and precision. The effect size was measured via the mean percent difference in the odds ratios, calculated to be 1.0% ± 33.5%. The significance was compared across the original and reproduction and we found that it changed for five groups. The odds ratios of Hispanic males for urine chlamydia and of mixed females for self-reported lifetime chlamydia changed from statistically significant to statistically insignificant. The odds ratios of Hispanic males for self-reported lifetime genital warts, of mixed males for urine chlamydia, and of mixed females for self-reported lifetime HIV testing changed from statistically insignificant to statistically significant. Finally, the precision of the reproduction was measured via the mean width of the confidence intervals. The original mean width was 13.34, while the reproduced mean width was 12.29.

When examining the paper for details that would have facilitated the reproduction of its analysis, we found that having more information on the management and recoding of controlled variables in the multivariate logistic regression would have been the most useful. The paper also did not include the name or construction of the weighting variable or how missing values were handled. These details were then extrapolated from the NHANES modules instead.

Discussion

This study reproduced the original study's results showing stark racial disparities in STIs and HIV infection in NHANES data from 1999 to 2012, even after controlling for sociodemographic, health access and income variables. In general, African American and Hispanic populations had both higher prevalence and odds of acquiring or currently having any of the examined STIs. For example, African American men had nearly 5 times the odds of white men of testing positive for HIV antibodies. Racial disparities also tended to stack with gender disparities, with African American women having 51 times the odds of testing positive for HIV antibodies when compared to white women. The significance of the findings was extremely strong in many cases.

Measuring reproducibility with percent difference in reported outcomes showed that the reproduction was a definite success for Table 1. The mean percent difference for the weighted prevalence of different diseases across racial groups was merely -0.4% ± 6.0%. Most differences for the weighted prevalence values were minor changes that affected paired positive and negative percentages or were a difference of a tenth most likely caused by rounding differences. However, the weighted prevalence of testing negative for HIV antibodies in African American males in the original study was 99.7%, which when added to the percent testing positive exceeds 100%. I attribute this error to a possible typo, as the original's weighted prevalence of positive HIV antibody tests in African American males is exactly the same at the reproduced value.

The multivariate logistic regression was not reproduced as perfectly, with more than double the mean percent difference and a much larger standard deviation at 1.0% ± 33.5%. There were also five estimates that flipped in significance, even though the overall precision, measured with mean confidence interval width, went down between the original and the reproduction. This is most likely due to differences in data management and categorization of the variables controlled for in the logistic regression, such as employment, income and education. Many of these variables that flipped in significance also have either small sample sizes or were borderline significant or insignificant in the original study, and so small changes in data handling resulted in large differences in possible interpretation. The increased precision, or smaller confidence intervals, is most likely also due to the data handling. Many of the choices made in recategorizing variables into binaries or fewer categories were done to improve the significance of the logistic regression estimates, even if they were not reported in the original. Other data

management choices were simply a function of how NHANES data collection was restructured over the span of 14 years.

The strengths of the original study and this reproduction include a large samples size that stretches across a lengthy time period. This boosts the power of the study and produces more comprehensive results on how STIs and HIV present themselves in a large population. The stratification by gender and race/ethnicity also allows for patterns in data and disparities to be more easily seen. Reporting both HIV antibody results and self-reported HIV testing also adds a more nuanced view of possible problems. Clear, descriptive information on the data collection process, analysis methodology and best methods when handling it, available via the original study or the NHANES documentation, was also immensely valuable. Finally, adjusting for multiple socioeconomic variables in the logistic regression allows a clear view of the racial/ethnic disparities in sexual health, without synergistic influence.

As an example, a study by Zuvekas and Taliaferro found that health insurance status can explain up to 30% of the disparity between Hispanics and whites and 40% of the disparity between African Americans and whites in having a regular source of care. They also found that income and education level are equally or more important when explaining racial disparities in having a regular source of healthcare, the family's perception of access and use of ambulatory (outpatient) care (Zuvekas & Taliaferro, 2003). By controlling for factors like health insurance status, the study can better and more clearly examine disparities exclusively related to race and ethnicity.

The results are therefore consistent with existing literature on racial disparities in sexually transmitted diseases. In a national report by Health and Human Services, it was found that when comparing rate of reported cases of chlamydia to that in White population, African Americans had 5.6 times the rate found in White populations (HHS et al., 2018). Similarly, American Indian/Alaskan Native (AI/AN) populations had a rate 3.7 times greater, while Hispanic populations had a rate 1.9 times greater (HHS et al., 2018). This pattern held across multiple STIs, just as it did in this reproduction, with African Americans having 7.7 times the White rate for gonorrhea, with AI/AN and Hispanic populations having 4.6 and 1.6 times the rate respectively. (HHS et al., 2018). Similar odds ratios were present in both the original paper and this reproduction, emphasizing that even with minor changes to data management, the overall

trend is clear. Racial health disparities manifest across multiple national databases and many different studies on health outcomes, even outside of sexual and reproductive health.

Additionally, the resulting emotional, financial and medical cost of STIs on the population is no small thing. Of the 19.7 million cases occurring in 2009 alone, the total estimated lifetime medical cost was around 15.6 billion dollars (range 11-20.6 billion) (Owusu et al., 2013). 95% of this was due to viral infections, like HIV, HSV-2, and HPV, which have no cure, but lifelong treatment options to minimize symptoms. Chlamydia cost an estimated 516.7 million dollars, while gonorrhea cost an estimated 162.1 million dollars (Owusu-Edusei et al., 2013). This further emphasizes the influence STIs, compounded with racial disparities, can wield over population health. These studies can hopefully be leveraged to push education, programming and policy to further mitigate the influence race or ethnicity can have on sexual health outcomes and illnesses, especially for African American, Hispanic, and female populations. Special emphasis should be paid to HIV testing and antibodies, as this viral STI has the starkest racial disparities while also being the costliest to treat.

Possible solutions include cultural competency training for providers, such as translator services, racially and ethnically diverse recruitment and retention practices, training and education, coordinating with trusted, local community health workers and ensuring care is culturally competent and sensitive (Brach & Fraserirector, 2000). The Affordable Care Act (ACA) provides one opportunity to try and address health care access and insurance, both social determinants of health, while also reducing the prevalence of STIs and HIV in minority populations. However, it's important to keep in mind that while health care access improved substantially for racial/ethnic minorities with the implementation of the ACA, it's benefits may not translate equally for all racial/ethnic groups in terms of health indicators, especially when looking at people under the age of 26 (Chen, Vargas-Bustamante, Mortensen, & Ortega, 2016; Scott et al., 2015).

Limitations of this study include the fact that the reproduction was not perfect and that there are possible differences in data management and recoding when compared to the original. This reproduction also used R Studio and the 'survey' package in place of STATA and 'svy', which have minor differences in calculation programming. One variable used in the original multivariate logistic regression, 'regular healthcare provider', was not found in any NHANES survey cycles and was therefore not included. Finally, all of the self-reported STIs as well as

HIV testing are not only subject to poor recall and memory, but reporting and social desirability biases.

Future studies should aim to more clearly examine the ‘Mixed, Other’ racial/ethnic group, as the small sample size, even with the combination of multiple survey cycles, was prohibitive to some statistical tests. Additional research should also pursue what impact geographic, cultural, and urban-rural factors have on racial/ethnic disparities in HIV and STI rates.

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Appendices

Appendix A. Tables

Table 1a. Weighted Prevalence of HIV and STIs in US Adult Males by Race/Ethnicity (NHANES, 1999-2012)

	African American	Hispanic	White	Mixed, Other	p-value
HIV antibody					<0.0001
Yes	2.6 (2.0-3.6)	0.9 (0.5-1.4)	0.3 (0.2-0.7)	0.00	
No	97.4 (96.4-98.0)	99.2 (98.6-99.5)	99.7 (99.4-99.8)	100.00	
Urine Chlamydia**					<0.0001
Yes	4.6 (3.4-6.1)	1.9 (1.3-2.7)	1.0 (0.7-1.4)	0.4 (0.1-1.0)	
No	95.4 (93.9-96.6)	98.1 (97.3-98.7)	99.0 (98.6-99.3)	99.7 (99.0-99.9)	
HSV-2					<0.0001
Yes	34.2 (31.6-36.9)	12.8 (11.0-14.7)	10.1 (9.0-11.3)	10.1 (6.9-14.6)	
No	65.8 (63.1-68.4)	87.3 (85.3-89.0)	89.9 (88.7-91.0)	89.9 (85.5-93.1)	
Hep C					0.018
Yes	3.0 (2.3-4.1)	1.6 (1.2-2.3)	2.4 (1.9-3.0)	0.9 (0.3-2.5)	
No	97.0 (95.9-97.8)	98.4 (97.7-98.8)	97.6 (97.0-98.1)	99.1 (97.5-99.7)	
Ever had STI*					<0.001
Yes	7.2 (6.0-8.6)	4.1 (3.2-5.3)	7.3 (6.2-8.5)	4.5 (2.7-7.4)	
No	92.8 (91.4-94.0)	95.9 (94.7-96.8)	92.7 (91.5-93.8)	95.5 (92.6-97.3)	
Ever had gonorrhea*					<0.0001
Yes	1.4 (1.0 – 2.0)	0.4 (0.2-0.8)	0.2 (0.1-0.4)	0.2 (<0.01-0.8)	
No	98.6 (98.0-99.0)	99.6 (99.2-99.8)	99.9 (99.6-99.9)	99.8 (99.2-100.0)	
Ever had chlamydia*					0.0013
Yes	1.9 (1.3-2.7)	0.7 (0.4-1.1)	0.5 (0.3-0.8)	0.7 (0.1-4.6)	
No	98.1 (97.3-98.7)	99.3 (98.9-99.6)	99.5 (99.2-99.7)	99.4 (95.44-99.91)	
Ever had herpes					0.385
Yes	2.8 (2.2-3.7)	1.8 (1.2-2.8)	2.4 (1.9-3.1)	1.7 (0.8-3.7)	
No	97.2 (9.6-97.8)	98.2 (97.2-98.8)	97.6 (96.9-98.2)	98.3 (96.3-99.2)	
Ever had genital warts*					<0.0001
Yes	2.3 (1.7-3.2)	1.8 (1.3-2.5)	5.0 (4.1-6.2)	2.5 (1.2-5.1)	
No	97.7 (96.9-98.3)	98.2 (97.5-98.7)	95.0 (93.9-95.9)	97.5 (94.9-98.8)	
Ever tested for HIV*					<0.0001
Yes	58.6 (56.0-61.2)	32.9 (30.4-35.5)	42.2 (40.4-44.1)	33.0 (28.3-38.2)	
No	41.4 (38.8-44.0)	67.1 (64.5-69.6)	57.8 (55.9-59.7)	67.0 (61.8-71.7)	

*self-reported. ** is between the ages of 20-39 only

Table 1b. Weighted Prevalence of HIV and STIs in US Adult Females by Race/Ethnicity (NHANES, 1999-2012)

	African American	Hispanic	White	Mixed, Other	p-value
HIV antibody					<0.0001
Yes	1.3 (0.9-2.0)	<0.01 (<0.01- 0.1)	<0.01 (<0.01-0.1)	0.3 (<0.01-2.0)	
No	98.7 (98.0-99.1)	100.0 (99.9-100.0)	100.0 (99.9-100.0)	99.7 (98.0-100.0)	
Urine Chlamydia**					<0.0001
Yes	4.4 (3.4-5.7)	2.0 (1.3-2.9)	0.9 (0.6-1.4)	3.0 (1.4-6.2)	
No	95.6 (94.3-96.6)	98.0 (97.1-98.7)	99.1 (98.6-99.4)	97.0 (93.8-98.6)	
HSV-2					<0.0001
Yes	58.3 (55.9-60.6)	23.7 (21.4-26.3)	19.8 (18.4-21.2)	18.3 (14.6-22.6)	
No	41.7 (39.4-44.1)	76.3 (73.7-78.7)	80.2 (78.8-81.6)	81.7 (77.4-85.4)	
Hep C					0.1514
Yes	2.0 (1.4-2.7)	0.9 (0.5-1.7)	1.5 (1.1-2.0)	0.9 (0.3-2.4)	
No	98.0 (97.3-98.6)	99.1 (98.3-99.5)	98.5 (98.0-98.9)	99.1 (97.6-99.7)	
Ever had STI*					<0.0001
Yes	16.7 (14.8-18.7)	8.5 (7.0-10.4)	13.5 (12.2-14.8)	10.9 (7.7-15.4)	
No	83.3 (81.3-85.2)	91.5 (89.6-93.0)	86.5 (85.2-87.8)	89.1 (84.6-92.3)	
Ever had gonorrhea*					<0.0001
Yes	1.3 (0.9-2.0)	0.2 (0.1-0.7)	0.2 (0.1-0.4)	1.1 (0.3-3.8)	
No	98.7 (98.0-99.1)	99.8 (99.3-99.9)	99.8 (99.6-99.9)	99.0 (96.3-99.7)	
Ever had chlamydia*					<0.0001
Yes	2.9 (2.2-4.0)	1.5 (0.9-2.3)	0.5 (0.3-0.9)	1.5 (0.7-3.0)	
No	97.1 (96.0-97.9)	98.5 (97.7-99.1)	99.5 (99.1-99.7)	98.6 (97.0-99.3)	
Ever had herpes*					<0.001
Yes	8.6 (7.2-10.2)	4.0 (2.9-5.5)	6.1 (5.2-7.1)	4.7 (2.8-7.7)	
No	91.4 (89.8-92.8)	96.0 (94.5-97.1)	93.9 (92.9-94.7)	95.3 (92.3-97.2)	
Ever had genital warts*					<0.0001
Yes	6.2 (5.2-7.5)	3.6 (2.8-4.8)	8.4 (7.5-9.5)	5.3 (2.9-9.4)	
No	93.8 (92.6-94.8)	96.4 (95.3-97.2)	91.6 (90.5-92.5)	94.7 (90.6-97.1)	
Ever tested for HIV*					<0.0001
Yes	67.9 (64.9-70.8)	48.5 (45.7-51.4)	51.3 (49.3-53.3)	40.2 (34.7-45.9)	
No	32.1 (29.2-35.1)	51.5 (48.6-54.4)	48.7 (46.7-50.7)	59.8 (54.1-65.3)	

* self-reported **between the ages of 20-39 only.

Table 2a. Adjusted Multivariate Logistic Regressions Examining Disparities in Race/Ethnicity for HIV and STI rates in US Adult Males (NHANES, 1999-2012)

	African American Males		Hispanic Males		Mixed, other Males		White Males (ref.)
	OR (95% CI)	p	OR (95% CI)	p	OR (95% CI)	p	
Lab Testing Results							
HIV antibody	4.6 (2.07-10.40)	***	2.80 (1.23-6.37)	*	N/A	N/A	1.00
Urine chlamydia (age 20-39)	3.85 (2.31-6.42)	****	1.72 (0.76-3.88)	0.20	0.25 (0.07-0.99)	*	1.00
HSV-2	4.69 (3.87-5.68)	****	1.50 (1.16-1.94)	**	1.28 (0.80-2.05)	0.30	1.00
Hep C antibody	0.63 (0.40-1.01)	0.06	1.10 (0.60-2.02)	0.77	0.52 (0.18-1.44)	0.21	1.00
Self-Reported in Questionnaire							
Ever had STI	1.07 (0.81-1.40)	0.65	0.82 (0.57-1.20)	0.33	0.72 (0.39-1.33)	0.30	1.00
Ever had gonorrhea	6.45 (2.22-18.79)	***	1.64 (0.33-8.23)	0.55	1.06 (0.15-7.59)	0.95	1.00
Ever had chlamydia	2.52 (1.25-5.10)	*	0.90 (0.36-2.23)	0.82	1.23 (0.18-8.24)	0.83	1.00
Ever had herpes	1.36 (0.93-1.99)	0.11	1.25 (0.70-2.24)	0.46	0.92 (0.38-2.24)	0.85	1.00
Ever had genital warts	0.48 (0.32-0.71)	***	0.59 (0.36-0.96)	*	0.62 (0.27-1.38)	0.24	1.00
Ever tested for HIV*	2.06 (1.81-2.35)	****	0.95 (0.08-1.12)	0.55	0.72 (0.57-0.90)	**	1.00

<0.05=* <0.01=** <0.001=*** <0.0001=****

Table 2. Adjusted Multivariate Logistic Regressions Examining Disparities in Race/Ethnicity for HIV and STI rates in US Adult Females (NHANES, 1999-2012)

	African American Females		Hispanic Females		Mixed, other Females		White Females (ref.)
	OR (95% CI)	p	OR (95% CI)	p	OR (95% CI)	p	
Lab Testing Results							
HIV antibody	50.98 (6.53-397.78)	***	0.64 (0.05-8.03)	0.73	10.59 (0.79-142.02)	0.08	1.00
Urine chlamydia (age 20-39)	4.57 (2.55-8.18)	****	2.01 (0.92-4.38)	0.08	2.97 (1.33-6.62)	**	1.00
HSV-2	5.61 (4.81-6.54)	****	1.35 (1.11-1.64)	**	1.18 (0.86-1.63)	0.31	1.00
Hep C antibody	0.79 (0.50-1.25)	0.32	0.96 (0.41-2.28)	0.93	0.98 (0.30-3.20)	0.98	1.00
Self-Reported in Questionnaire							
Ever had STI	1.30 (1.08-1.57)	**	0.80 (0.61-1.06)	0.13	0.99 (0.64-1.53)	0.96	1.00
Ever had gonorrhea	3.99 (1.28-12.37)	*	0.90 (0.27-3.02)	0.86	4.74 (0.57-39.49)	0.15	1.00
Ever had chlamydia	3.55 (2.03-6.21)	****	1.92 (0.92-3.98)	0.08	2.46 (0.92-3.98)	0.08	1.00
Ever had herpes	1.51 (1.17-1.96)	**	0.96 (0.63-1.47)	0.86	0.89 (0.48-1.65)	0.72	1.00
Ever had genital warts	0.80 (0.62-1.03)	0.08	0.51 (0.34-0.77)	**	0.76 (0.37-1.53)	0.44	1.00
Ever tested for HIV*	2.33 (1.98-2.74)	****	1.02 (0.86-1.21)	0.82	0.75 (0.58-0.96)	*	1.00

<0.05=* <0.01=** <0.001=*** <0.0001=****

Appendix B. Core Competencies

- **Explaining the role of quantitative and qualitative methods and sciences in describing and assessing a population's health.**
    - Researching epidemiological surveys and analysis, such as the use of NHANES data in Operario et al. study, and how they can be used to describe and assess population health
- **Explaining social, political and economic determinants of health and how they contribute to population health and health inequities.**
    - Examining racial and ethnic disparities in HIV and STI rates in the United States
- **Analyze quantitative and qualitative data using biostatistics, informatics, computer-based programming and software as appropriate.**
    - Using quantitative methods (multivariable logistic regression, weighted prevalence) to describe and assess population health in regard to HIV and STIs
- **Interpret results of data analysis for public health research, policy or practice.**
    - Interpreting the results of the multivariate logistic regression assessing HIV and STI rates.
- **Describe the basic biology and epidemiology of major infectious diseases and health conditions in global health.**
    - The analysis has a focus on sexually transmitted diseases and human immunodeficiency virus infections, both of which are major infectious diseases and a heavy concern for not only the United States but the greater international community.
- **Apply and interpret common statistical methods for inference found in public health studies.**
    - Interpreting the results of the multivariate logistic regression assessing HIV and STI rates.
- **Demonstrate an understanding of the components of reproducible research.**
    - Note and display best practices of reproducible research, while also reproducing a study.
- **Demonstrate an understanding of systematic biases (selection and information biases) that affect observational, quasi-experimental and experimental studies.**
    - Examine the systemic biases that affect observational studies like NHANES, as well as the biases present in the form of racial and ethnic disparities and the lack of solid structures, support and advocacy of reproductions and replications of research studies.

Appendix C. Timeline of capstone activities, goals and due dates from November 10th to April 5th, separated out by weeks.

Activity	Wk 1 (Nov 10th)	Wk 2 (Nov 17th)	Wk 3 (Nov 24th)	Wk 4 (Dec 1st)	WINTER BREAK	Wk 5 (Jan 12th)	Wk 6 (Jan 19th)	Wk 7 (Jan 26th)	Wk 8 (Feb 2nd)	Wk 9 (Feb 9th)	Wk 10 (Feb 16th)	Wk 11 (Feb 23rd)	Wk 12 (Mar 1st)	SPRING BREAK	Wk 14 (Mar 15th)	Wk 15 (Mar 22nd)	Wk 16 (Mar 29th)	Wk 17 (Apr 5th)
Literature Review & Notes in Excel - HIV STIs - Racial Ethnic Disparities - Reproducibility	X																	
Write Introduction		X													X			
Contact Researcher	X																	
Collect Data from NHANES	X																	
Consult Dr. Harris on STATA v R Studio	X																	
Book Writing Center Appointments	X																	
Research Multivariate Logistic Regressions in Program of Choice		X					X	X										
Research Weighting Process/Data Collection in NHANES		X				X												
Write Methods			X												X			
Write Competencies			X												X			
Writing Center Appointment 1				X														
Check in with Dr. Harris				X														
Compile Bibliography				X									X		X			
Submit Project Proposal				Dec 6th														
Data Management/Cleaning/Etc						X												
Multivariate Logistic Regression + Interpretation							X	X							X			
Check in with Dr. Harris								X										
Heavy Edits Intro/Methods									X									
Results Section									X	X					X			
Writing Center Appointment 2										X								
Discussion Section											X	X			X			
Conclusion													X		X			
Writing Center Appointment 3													X					
Final Meeting with Dr. Harris													X					
Review															X of above			
Buffer																X	X	X

Appendix D.
Permalink to Author's Github: https://github.com/arenganathan28/MPH_Capstone

Appendix E:

Chlamydia is a bacterial disease caused by *Chlamydia trachomatis*, and can infect both men and women via oral, vaginal or anal sex with someone who has an infection. It can also be passed to infants during childbirth. Chlamydia can be treated with antibiotics, but they cannot cure any permanent damage caused by the disease, like pelvic inflammatory disease (PID), chronic pelvic pain, infertility and increased risk of ectopic pregnancies. During 2018, chlamydia had a rate of 539.9 cases per 100,000 people in the population, an increase of 2.9% since the previous year. Between 2011 and 2018, the rate of chlamydia cases in the United States increased 19.1% (HHS et al., 2018).

Gonorrhea is a bacterial disease caused by *Neisseria gonorrhoeae*, and can infect both men and women via any type of sex, including contact via mouth, throat, eyes, urethra, vagina, penis, or anus. Severe cases of gonorrhea in pregnant mothers can result in the infection being passed to the infant. Gonorrhea can be treated with antibiotics but is rapidly evolving into strains resistant to available antibiotics, like cefixime and ceftriaxone. People infected with gonorrhea for long periods of time can have scarring in their fallopian tubes or urethra, as well as PID, pelvic pain, infertility and ectopic pregnancies in women. During 2018, rates of gonorrhea increased 5% to 179.1 cases per 100,000 population. Since its historic low in 2009, the rate of gonorrhea cases in the United States has increased 82.6% (HHS et al., 2018).

Syphilis is a bacterial disease caused by *Treponema pallidum* and can infect both men and women by contact of skin or mucous membrane with sores on an infected person. The primary stage is a sore that develops where the bacteria entered the body. Secondary syphilis is a rash covering the entire body, with wart-like sores on the mouth and genitals. Latent syphilis has no symptoms, and the previous signs and symptoms may never occur again, or the disease can progress to the tertiary stage where severe complications can affect the brain, nerves, eyes, heart, blood vessels, liver, bones and joins. Finally, babies born to infected women can become infected with congenital syphilis via the placenta or during birth. During 2018, primary and secondary syphilis had a rate of 10.8 cases per 100,000 population, an increase of 14.9% from the previous year, and a 71.4% increase from 2014(HHS et al., 2018).

Herpes Simplex Virus (HSV) is one of the most prevalent STIs, and is transmitted by a virus, with two types present, Type 1 (HSV-1 or oral herpes) and Type 2 (HSV-2 or genital herpes). Type 1 causes sores around the mouth and lips and can cause type 2. However, most cases of genital herpes are caused by HSV-2, where sores occur around the genitals and rectum. Most infections are subclinical. There is no cure, but there are treatments to relieve symptoms. NHANES data from 2015-2016 showed an age-adjusted seroprevalence of 12.1%, with seroprevalence increasing with age, and a general trend of decreasing or plateauing prevalence (McQuillan, Kruszon-Moran, Flagg, & Paulose-Ram, 2018).

Genital warts, also known as condylomata acuminate, and are caused by HPV types 6 and 11 about 90% of the time. Of the population of 18 to 59 year olds, 5.6% have ever been diagnosed with genital warts. The estimated prevalence of genital warts is 1000 cases per 100,000 in the 18 to 45-year-old population (Dinh, Sternberg, Dunne, & Markowitz, 2008). There are nearly two dozen strains of HPV and some of the more virulent strains are linked to the overwhelming majority of genital cancers, from 99.7% of cervical cancer to 63% of penile cancers (Walboomers et al, 1999).

Hepatitis C is a viral disease that causes inflammation of the liver. It can be transmitted via oral, vaginal or anal sex with an infected person, even without symptoms. Most people do not require treatment, but those with chronic infections might. Complications of HepC include increased risk of HIV, possible infection of an unborn baby in infected mothers, cirrhosis, and liver cancer when left untreated. The prevalence of chronic HepC in the United States between 2009 and 2016 is about 0.80% (Peery et al., 2019).

Human Immunodeficiency Virus (HIV) is a virus that destroys CD4 cells, a type of white blood cells. AIDS, or acquired immunodeficiency syndrome, results from being infected with HIV and occurs in its final stages. It severely weakens the immune system and leaving the body vulnerable to otherwise minor illnesses and infections. In the 1980s, prior to public and clinician understanding or a treatment, the life expectancy following an AIDs diagnosis was one year. Transmission of the virus occurs through unprotected anal, vaginal or oral sex, sharing needles, or in an infant when an infected mother gives birth. There is no cure for HIV, but antiretroviral therapy (ART) can reduce the body's viral load, reducing both the risk of transmitting the disease to other people and giving

the immune system a chance to recover. Treatment can reduce the difference in lifespan between infected and uninfected populations to just 3 years (Marcus et al, 2019). In 2016, the estimated incidence was 14.3 cases per 100,000 people (HHS et al., 2018; Linley et al., 2019).