THESIS

INDIVIDUAL AND STRUCTURAL PREDICTORS OF HUMAN PAPILLOMAVIRUS: RACE AS AN INTERACTION EFFECT AND THE CONSTRUCTION OF RACIALIZED SEXUALITIES

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ABSTRACT

INDIVIDUAL AND STRUCTURAL PREDICTORS OF HUMAN PAPILLOMAVIRUS: RACE AS AN INTERACTION EFFECT AND THE CONSTRUCTION OF RACIALIZED SEXUALITIES

Human Papillomavirus (HPV) is the most common sexually transmitted infection in the United States, and has different prevalence rates among different gender, racial, ethnic, and class groups. Many studies have identified number of sex partners as the most predictive variable for HPV status which implies individual behavior is responsible for differences in HPV rates between social groups. The purpose of this thesis is to evaluate the extent to which individual and structural factors correlate with HPV status, and whether those correlations vary by race.

This study uses public-use data from the National Health and Nutrition Examination Survey from years 2011-2014. Logistic regression models which included individual risk behaviors, structural resources, and interactions with black and white race showed that number of sex partners has a different effect on HPV risk for black and white women. These findings suggest that citing number of sex partners as the primary predictor of HPV risk may falsely universalize whiteness, and pathologize black sexuality.

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CHAPTER 1: INTRODUCTION

Human Papillomavirus (HPV) is the most common sexually transmitted infection (STI) in the United States (Satterwhite et al. 2013). While most HPV infections are harmless, high-risk strains are found in virtually every incidence of pre-cancerous cervical lesions and Invasive Cervical Cancer (ICC) (Grulich et al. 2010). Medical researchers examine which variables are associated with HPV in order to develop effective intervention strategies for its prevention and management.

Several HPV studies have used National Health and Nutrition Examination Survey (NHANES) data and found that the only variables consistently independently correlated with HPV status were age, gender, and number of sex partners (Chaturvedi et al. 2015; D'Souza et al. 2014; Dunne et al. 2007; Markowitz et al. 2009). Because gender has biological associations with HPV transmission, number of sex partners is the strongest predictor which is also eligible for intervention strategies. Health intervention strategies are varied but we can broadly categorize them as targeting individual behaviors (such as hand-washing, wearing seat belts) and/or structural elements (such as easy access to vaccines, health care).

Targeting a behavior such as number of sex partners seems like a reasonable strategy, however, I was skeptical of this finding because HPV is distributed differentially throughout the population by race and class. Assuming number of partners is the primary cause, this implies that marginalized racial and ethnic groups and low-income people have greater HPV prevalence of because they have a greater number of sex partners than white, affluent people.

This implication seems to play into racist and classist narratives of sexuality. I argue that researchers should reexamine findings which appear to support status quo power relations within

their social context to ensure a valid interpretation. I also questioned these findings because research on other STIs has shown that black women in particular are at increased risk of STIs even without engaging in risk behaviors, and after controlling for access to resources (Hallfors et al. 2007; Halpern et al. 2004; Harawa et al. 2003; Santelli et al. 2000, Ellen, Aral and Madger 1998).

The purpose of this thesis is to examine the causality of individual behaviors and structural factors in determining racial disparities in rates of HPV, and to evaluate the implications the respective causes have for how sexualities are racialized.

I used NHANES data from the 2011-2014 cycles and created logistic regression models which control for socioeconomic indicators and risk behaviors, and then evaluated the effect of race and number of sex partners, as well as all possible interactions between model variables and race. I focused only on black women and white women to simplify a relational analysis, and examined both oral and vaginal HPV.

Drawing on Foucault's theory of knowledge/power, and Hammond's advice to feminist sexuality researchers to examine how racialized sexual constructions relate to each other and broader power relations, I put my findings into the context of historical power relations between black and white women, and the knowledge produced about their sexualities.

Ultimately, I use a structural theory of health inequalities by Link and Phelan (1995) to explain my results. I adapt their theory to include not just material inequalities, but also unequal power relations in knowledge production, and specifically the relational narratives created about black and white women's sexualities. I conclude that number of partners is a significant predictor of oral and vaginal HPV risk, but it fails to explain differences by race.

In the next chapter I review literature on health care research and policy; connections between knowledge, power, race, and sexuality; and contemporary HPV research.

CHAPTER 2: LITERATURE REVIEW

Introduction

My research examines the causality of individual behaviors and structural factors in determining racial disparities in rates of human papillomavirus (HPV), and the implications the respective causes have for the construction of racialized sexuality. I first describe the disciplinary and theoretical approach to my research question, and then present an outline of the three sections of the literature review: 1) a mapping of trends in health care research, policy, and public perception which led to the debate between individual and structural causes of health inequalities; 2) a review of the theoretical literature which informs my critique of contemporary HPV research; and 3) an overview of the health effects of HPV, research which posits causal mechanisms of HPV transmission, and the arguments which lead me to my specific research question. I begin by situating my own research within sociology and, more specifically, medical sociology.

I situate my research most broadly in the discipline of sociology. I draw upon C. Wright Mills' concept of the sociological imagination (2000) to examine disparities in health outcomes. Mills distinguishes between personal troubles, which "occur within the character of the individual and within the range of his or her immediate relations with others," and public issues, which "have to do with the organization of many such milieu into the institutions of an historical society as a whole, with the ways in which various milieu overlap and interpenetrate to form the larger structure of social and historical life" (2000:4). Throughout this research I take the view that the disparate outcomes in rates of HPV constitute a public issue and therefore merit intervention strategies targeted at the population level; this perspective runs contrary to research

which identifies individual behaviors as the causal mechanism of HPV disparities, implying they are personal troubles (Chaturvedi et al. 2015; D'Souza et al. 2014; Gillison et al. 2012). This distinction is not just a rhetorical one as the decision to analyze HPV disparities as either public issues or personal troubles determines the type of intervention strategy selected to lessen disparities, and also has implications for how scientific literature constructs the groups experiencing higher or lower rates of HPV.

More specifically, my research is situated within the sub-discipline of medical sociology (also called sociology of medicine, and sociology of health and illness). Medical sociology examines sociological concerns in medical research and population health, and may take a critical turn in existing medical health discourse and strategy. This approach is distinct from sociology *for* medicine, which uses sociology in the service of medicine and adopts the norms and values of the medical system (Straus 1957). Critiques of medical sociology include comments that medical sociology, like sociology for medicine, is entrenched in "medical bias" (Gold 1977:165), and that it is an applied social science with less theoretical rigor and more influence from medical research agencies than the sociological community (Turner 1995). However, despite these characterizations, Turner's history of medical sociology connects the origins of sociology to nineteenth-century medical practices, such as medical surveys, citing Foucault's assertion that "sociology and medicine are inextricably linked together" (1995:6).

Throughout his text on medical sociology Turner draws on Foucault's analysis of medical issues to bolster medical sociology's theoretical grounding as Foucault's perspective aids sociologists in seeing "the body of individuals and the body of populations as products of power and knowledge" (Turner 1995:17). As I develop later, my research takes seriously Foucault's analytics of knowledge/power, as well as his description of how power shapes sexual subjects.

This aspect is critical to my discussion of how the type of knowledge researchers produce concerning the cause of HPV disparities has consequences for how sexualities are constructed.

In the next section I review three types of literature to explain the background of my research question: First I review several historical trends in health research which led to the debate between individual and structural explanations of health disparities. I discuss the rise of medicalization in the second half of the 20th century and the critiques it received from citizens and industry. Then I discuss how the growing use of health services and awareness of environmental causes of disease prompted industry leaders to emphasize personal responsibility over ones' health, which both the government and critics of medicalization supported. Medical sociologists have criticized the emphasis on personal responsibility over ones' health pointing to its ineffectiveness at lessening health disparities by race and class, and invocation of a victim-blaming ideology. I outline the various theories which seek to explain health disparities by race and class and identify the approach that critical medical sociologists favor.

Next I review literature on the role of power in knowledge production and the construction of sexualities: I introduce Foucault's theory of knowledge/power and explain how sexuality research is both embedded within and reinforces the power relations of the social context within it is produced. I draw on women of color feminists Higginbotham, Hammonds, and Clark Hine to explain the concept of *racialized* sexuality and the relational construction of white and black women's sexuality. Following Hammonds' advice to researchers I focus on HPV disparities between white women and black women and the power relations that contribute to unequal health outcomes.

Lastly, I review contemporary research examining causes of HPV specifically, and my empirical and theoretical critiques, which guide me to my specific research question. I outline trends in research on the causal mechanisms of HPV and their related intervention strategies, which I argue inadequately attend to access to resources, and inadvertently produce knowledge with racial implications which support the ideology of patriarchal white supremacy.

Trends in health care

Medicalization, as developed by sociologist Irving Zola, is one of the most successful and enduring concepts from the field of medical sociology (Conrad, Bird and Fremont 2000). Zola developed the term to describe how the medical profession was expanding into ever wider spheres of life, especially deviant behaviors, resulting in medical professionals replacing religious or legal actors as agents of social control (1972). Medical professionals and the larger community previously understood problems such as alcoholism, child abuse, opiate addiction, and sexual functioning as moral issues, but the rise of medicalization reframed those issues in medical terms of illness or health (Crawford 1980).

The trend towards medialization increased the amount of money the federal government and industry spent on health care. For example, throughout the 1950s-1970s more people had access to health benefits through their employers, and the government subsidized medical research and training. As more people sought out expert medical advice and utilized health services, government and industry noticed the growing costs. In terms of overall cost, health expenditures nearly doubled in the U.S. from 4.1% of the gross national product in 1950 to 8.1% in 1976. In government, the Council on Wage and Price Stability noted the expanding portion of the federal budget devoted to healthcare which went from 8.9% in 1969 to 11.3% in 1975. In

industry, General Motors claimed that in 1975 it spent more money on health insurance than it did with its principle metal supplier, U.S. Steel; and Standard Oil said in 1976 that its employee health costs had tripled in the last seven years (Crawford 1977).

As workers used more health services they became increasingly aware of the social and environmental causes of disease and injury. For example, Americans increasingly understood cancer as a disease caused by exposure to carcinogens in the environment. The environmental health movement and environmental sociologists pointed out the amount of toxic waste and chemicals produced by industry (Brown 2007; Brown and Mikkelsen 1997; Corburn 2005; Szasz 1994), and lack of regulation and research on household chemicals which endangered community health (Brodeur 1985).

During this time of increased medicalization researchers and activists were also more aware of injury and disease caused by the workplace and lack of safety regulation (Rosner and Markowitz 1987). Unions argued for better occupational health and safety, which prompted the government to create the Occupational Safety and Health Administration who debated appropriate limits and regulations to promote workplace safety. These environmental and workplace safety regulations affected industry who then warned of the impending unemployment, inflation, and economic downturn should regulations be too restrictive (Crawford 1977).

While government and industry worried about the increasing costs of health care and demands for safer environments and work spaces, researchers and citizens questioned the efficacy of the increased reliance on modern medicine. Publications such as “On the limitations of modern medicine” in *Science, Medicine and Man* (Powles 1973), *The End of Medicine*

(Carlson 1975), and *Medical Nemesis: The Expropriation of Health* (Illich 1975) pointed to the limitations of modern medicine to curb morbidity and mortality rates and began to popularize the idea that professional Western medicine was ineffective and oversold. McKeown (1979) argued that changes in living conditions, hygiene, and nutrition contributed more to longer life expectancies than medical technology.

At the same time, while medicine was advancing into additional spheres of life, those in the women's movement criticized the power and authority the medical community had to pathologize the female body (Morgen 2002). As a result, many in the women's movement sought alternatives to conventional medicine and turned to personalized health care from holistic health movements, self-care, and self-help movements. These movements popularized therapies such as meditation, biofeedback, and homeopathy, and diffused the idea that true health and wellbeing comes from preventative efforts and lifestyle changes (Crawford 1980).

Out of these debates over cost of treatment, effectiveness of treatment, cause of disease, and limits of medical expert authority arose a new trend in medical care and popular conceptions of health which emphasized personal responsibility over health and behavioral change to avoid illness. Sociologist Robert Crawford describes the new health consciousness with the term *healthism,* defined as:

> The preoccupation with personal health as a primary—often the primary—focus for the definition and achievement of well-being; a goal which is to be attained primarily through the modification of life styles, with or without therapeutic help. . . Healthists will acknowledge. . . that health problems may originate outside the individual, e.g. in the American diet, but since these problems are also behavioral, solutions are seen to lie within the realm of individual choice. . . For the healthist, solution rests within the individual's determination to resist culture, advertising, institutional and environment constraints, disease agents, or, simply, lazy or poor personal habits (1980:368).

As part of this movement, the government encouraged expanding health *education* rather than health *services* (Somers 1971). Government and industry encouraged the view that health is a social duty and personal responsibility, which allowed critics of expanding health costs to question citizens' *right* to health care. Leon Kass, an opponent of national health care wrote:

> All the proposals for National Health Insurance embrace, without qualification, the no-fault principle. They therefore choose to ignore, or treat as irrelevant, the importance of personal responsibility for the state of one's health. As a result, they pass up an opportunity to build both positive and negative inducements into the insurance payment plan, by measures such as refusing or reducing benefits for chronic respiratory disease care to persons who continue to smoke (1975:41).

The National Consumer Health Information and Health Promotion Act of 1976 emphasized "appropriate" use of health care, and "prevention or moderation of illness or accidents that appear controllable through individual knowledge and behavior" (1976:841-42). Corporations embraced this shift in popular and political discourse because they could place the burden of cost and responsibility back on the individual.

Crawford noted that it was mainly the white, middle class who increasingly consumed heath magazines, vitamins, and exercise equipment; as well as health-related topics in news and media. This segment of the population already enjoyed better health (Crawford 1980). While healthist discourse fails to highlight health disparities experienced by different socioeconomic classes and racial and ethnic minorities, according to its logic, the explanation for any health disparity lies in the behavior or culture of the individual or group.

A sociological perspective offers a fundamental critique of healthism. Instead of focusing on individual behaviors, sociologists examine how social institutions or beliefs affect different people in different ways. For example, a sociological perspective may examine how differential health outcomes are distributed by socioeconomic status, race, and gender (Blaxter 1991;

McCartney, Collins and Mackenzie 2013). There is ample evidence of this differential distribution of health outcomes: Women may live longer than men, but experience greater morbidity and illness throughout life (Annandale and Hunt 2000). Those of a higher socioeconomic status tend to live longer than those of lower status (Black et al. 1980; Blaxter 1991). Like gender and class, race affects health outcomes.

Alegria and colleagues (2011:364) define racial and ethnic disparities as "differences in access, health care quality or health care outcomes that are not due to clinical needs or the appropriateness of treatment." In other words, if clinical needs are the same across racial groups for a given medical issue, and the health outcomes are unequal, that would constitute a racial health disparity. For example, black Americans have higher death rates for chronic diseases such as heart disease, cancer, and diabetes even after accounting for differences in socioeconomic status (NCHS 2007). Factors such as "stress produced by racism, residential segregation, and inferior quality of residential spaces" may explain some of the health differences (Freund, McGuire and Podhurst 2003:38).

Researchers have critiqued cultural and behavioral explanations for differences in health outcomes, such as healthism implies, as sexist (Daykin and Naidoo 1995; Kenner 1985; Lees 1986; Oakley 1989; Wilton 1994), and racist (Donovan 1984; Douglas 2013; Pearson 1989) on the grounds that they ignore the material realities of peoples' lives, and obscure the way unequal power relations and access to resources are institutionalized. While the term healthism emerged in the 1980s, its use has continued into the twenty-first century, with current research noting its usage in obesity and body image research (Lee and Macdonald 2010; Roy 2008; Wright, O'Flynn and Macdonald 2006), health and self-surveillance (Lupton 2013), and health ideologies with undertones of religious zeal (Loefler 2003; Pelters and Wijma 2016).

Theories of differential health outcomes

There are many theories beyond healthism for why health disparities exist. Early research on the existence of systematic health inequalities and a typology of explanations came from the Black Report in 1980 (Black et al. 1980). The typology the report developed summarizes the common theories in an understandable and empirically supported way. Researchers in sociology and public health have and continue to cite it when discussing social health inequalities (Blaxter 1991; Freund, McGuire and Podhurst 2003; McCartney, Collins and Mackenzie 2013).

The British Secretary of State appointed a team of U.K. researchers in 1977 to explore differences in health by social status and analyze the results to find the causal mechanisms. They examined morbidity and mortality rates between English classes, operationalized primarily by occupational status, and found the trend of better health outcomes for those of higher class status for a variety of illnesses. The research team summarized four different types of explanations for these health inequalities and explained which theory they found most convincing. They discuss 1) artefact explanations, 2) theories of natural or social selection, 3) cultural/behavioral explanations, and 4) material/structural explanations.

The artefact explanation suggests that while health outcomes and social status may be correlated, this correlation alone fails to prove causality. Continuities of this pattern may be due more to the changing occupation structure where partly-skilled and unskilled manual labor positions are disappearing, and increasingly filled with older people whose health would already be in decline. The report critiques this perspective noting that the change in occupation is often perceived to be greater than it actually is, and occupation change alone would fail to account for the prevalence of such stratified health outcomes.

Theories of natural and social selection suggest that health determines social position, rather than the other way around, and that those of poor health enter lower social strata because they are unable to complete the training and demands of professional careers. The report summarizes this theory with "Those men and women who by virtue of innate physical characteristics are destined to live the shortest lives also reap the most meagre rewards" (Black et al. 1980:6.7). The Black Report critiques social selection theories by pointing out that high levels of stamina and strength may be more necessary in manual labor jobs which are correlated with poorer health.

The cultural/behavioral explanations align most closely with healthism and suggest that health disparities arise out of different health habits among classes. Behavioral proponents point to evidence such as poor diet, with lower-income groups eating more white bread, sugar, and potatoes and fewer fruits than more affluent groups. They also point out higher rates of smoking among those in manual labor occupations than professionals, and more active/athletic leisure activities among professionals. The Black Report notes the argument's similarities to Oscar Lewis' culture of poverty thesis (1966), and argues that neither adequately attend to structural inequalities.

Materialist and structuralist explanations emphasize "the role of economic and associated socio-structural factors in distribution of health and well-being" (Black et al. 1980:6.9). The explanations vary according to the degree of causality given to economic factors rather than other indicators of social status such as race, gender, or education. Despite the criticism of being a purely Marxist account, the Black Report researchers found that the structuralist theory explained the data better than the other theories they explored.

Different health disciplines prefer different modes of intervention to lessen health inequalities, although the disciplines of medical sociology, public health, and epidemiology all agree that health inequalities exist between social groups, and few would currently argue for the artifactual or selection theories for health inequalities. The general consensus among health researchers is that both behavior and environment/structure influence health outcomes, but each discipline emphasizes behavior or structure to different degrees.

For example, the field of epidemiology uses the metaphor of a "web of causation" to describe the interaction of multiple intertwining aspects: agent (source of disease), host (where or how the agent creates or transmits disease, such as contaminated water) and environment (the setting which allows the host to survive) (Freund, McGuire and Podhurst 2003). The strength of this framework is that it acknowledges multiple causes; however it is more useful for widespread infectious disease than noninfectious disease, such as cancer or heart disease. While epidemiologists are aware of multiple causes of disease, their tendency to use separate quantitative data and statistical correlation between a few variables divorces the data from its context. The result leads to explanations centered more on individual behavior than social context. While epidemiology generally pays less attention to social context, *social* epidemiologists highlight the importance of the eco-social environment (Krieger 1994; Krieger 2001; Link and Phelan 1995).

Like epidemiologists' web of causation, public health researchers rely on a multifaceted approach to health intervention strategies. There are several models of causation and intervention within the field of public health. The traditional model of potential impact of health care intervention is a four-tiered pyramid, with the base of the pyramid representing the most impactful strategies, those which target the whole population, and the ascending levels

representing primary, secondary, and tertiary care with diminishing impact (Gold and Teutsch 1994). Public health researchers have proposed alternative models, such as Frieden's five-tier health impact pyramid, where the base of the pyramid is socioeconomic factors (2010). Frieden notes that although health interventions aimed at ameliorating socioeconomic inequalities would be the most effective, they are also the most controversial as they would often require "fundamental social change," which is generally viewed as outside the purview of public health programs (2010:592). While public health (and epidemiological) researchers may see the importance of social conditions, conventional and politically non-threatening interventions often limit their fields.

Medical sociologists attentive to differences in power and resources generally prefer structural explanations of health inequalities. The "most prominent and sustained" theory linking socioeconomic status and health comes from Bruce Link and Jo Phelan who have backgrounds in social epidemiology and medical sociology (Freese and Lutfey 2011). Link and Phelan's work on fundamental causality began with their article "Social Conditions as Fundamental Causes of Disease" in 1995 which focused on socioeconomic status, and their work continues today with their most recent publication: "Is racism a fundamental cause of inequalities in health?" in 2015.

In their early work Link and Phelan (1995) followed a structuralist approach and developed an argument for social conditions as *fundamental causes* of disease, insisting the social conditions must change first in order for any individualized approach to be effective. They noted that while health promotion research may acknowledge a connection between social factors such as socioeconomic status, race, gender, and health outcomes, this literature often treats social factors as merely a proxy for more specific risk factors, and often controlled social conditions out of research. Rather than the initial causal variable, the focus is on mediating

variables such as diet, cholesterol, hypertension or lack of exercise; risk factors over which researchers perceive individuals have some control.

Link and Phelan criticized this approach and advised researchers to contextualize risk factors and examine how they came to be in the environment in the first place. They emphasize analyzing the role resources such as money, knowledge, power, prestige, social support and social networks have in helping people avoid risk or cope with disease. By focusing on access to resources instead of specific risk mechanisms their theory of fundamental cause explains how health disparities continue to exist throughout time even if the specific risk factor ameliorates, or a specific disease becomes less stratified.

For example, consider Kadushin's prediction in the 1960s that socioeconomic health disparities were quickly disappearing (1964). He noted that the risk factors plaguing the working class in the first half of the century such as dire housing conditions, poor sanitation, dangerous work conditions, and poor vaccine access, had gotten much better. Rates of diphtheria, measles, typhoid fever, tuberculosis, and syphilis declined dramatically. However, Link and Phelan claim that Kadushin's prediction of equal health across classes was premature because different risk factors emerged which disproportionately affected the poor and working classes. For example, people of all classes smoked in the beginning of the twentieth century, but then smoking became more common among the lower class. Greater education and access to nutritious food was also stratified, as was strenuous work conditions and opportunity to exercise. Now heart health, hypotension, and obesity are stratified health outcomes. The specific risk mechanisms and health outcomes changed, but the overall health inequalities remained.

To account for these changes in mechanisms but not of inequalities over time, Link and Phelan developed an explanation of social conditions as fundamental causes of disease. Their theory has four essential elements: 1) the social condition influences multiple disease outcomes, 2) it affects disease outcomes through multiple risk factors, 3) it involves access to resources to minimize or cope with risk, and 4) it is reproduced over time via replacement of intervening mechanisms (Phelan, Link and Tehranifar 2010). Essentially their argument focuses on social and economic resources as fundamental causes because they are responsible for a variety of health disparities over time. Rather than targeting one specific risk factor and one health issue at a time targeting inequalities in resources has a lasting effect on multiple health inequalities.

Link and Phelan have tested their fundamental cause hypothesis against various mortality rates data and found it supported (Phelan et al. 2004; Phelan, Link and Tehranifar 2010; Tehranifar et al. 2009). Other researchers have tested their hypothesis using data on statins and cholesterol (Chang and Lauderdale 2009) and diabetes (Lutfey and Freese 2005). While Lutfey and Freese later critiqued the fundamental cause theory for assuming that people will actually utilize resources they have access to, they agree with the core of the theory: that unequal access to resources results in continued, varied, health disparities (2011). Several other studies found evidence for the various components of their theory without testing it explicitly, such as research illustrating the association of multiple risk factors which are differentially distributed by race and class (Dahl, Hofoss and Elstad 2007; House and Williams 2000; Kunst et al. 1998).

Link and Phelan explain the implications their theory has for future health research and offer two pieces of advice: First, that researchers studying a health outcome where social conditions may be a fundamental cause should be cautious to imply direct causality via intervening variables in a path or regression model. They urge medical sociologists and social

epidemiologists to offer more context than just the most proximate causes of disease. Second, they recommend researchers examine the mechanisms that create the inequality of resources in the first place and include those mechanisms in intervention strategies.

Link and Phelan's theory of fundamental cause is an example of a critical medical sociology approach because they focus on power relations and critique conventional, individualized health interventions. They began by researching socioeconomic status and extended their argument to power relations in race. While their work does not explicitly discuss knowledge production, their attention to underlying material power relations connects to Foucault's emphasis on the role of power in constructing medical subjects.

The following section examines Foucault's articulation of knowledge/power, and explains the connection between power, knowledge production, and the way power constitutes sexual subjects. Sexuality research especially contributes to the construction of racialized sexualities, which is essential for understanding how explanations of HPV disparities have racialized implications.

Power, knowledge, race, and sexuality

This section provides a theoretical overview of 1) Foucault's definition of power and its relation to knowledge, 2) the politics involved in sexuality research, and 3) the necessity studying white and black women's sexuality as relational constructs. This theoretical background provides the basis of my criticism of HPV research which fails to explicitly engage with race, but nonetheless produces knowledge about racialized sexualities. I begin by explaining Foucault's definition of power and the way power is deployed in the creation of knowledge about sexuality. I then discuss trends in the knowledge production of black and white sexualities and why

researchers must study them relationally. I conclude by explaining how studying sexuality without explicitly attending to race and gender implicitly centers whiteness and contributes to knowledge which pathologizes black female sexuality.

I begin with Foucault's definition of power because of its connection to medical sociology and relevance to the politics of knowledge production (Turner 1995). I first differentiate it from more conventional understandings of power. For example, Max Weber's definition of power, defined in relation to the state, is "a human community that (successfully) claims the monopoly of the legitimate use of physical force within a given territory" (Weber, Mills and Gerth 1963:78). According to Weber, traditional, charismatic, or legal authority legitimize the power of the state. This definition of power assumes physical force is a means of enforcing the power relation, and also that power is something a person or institution *has* which can be wielded *over* others.

Foucault's definition of power explicitly departs from these assertions and instead articulates power as based in relation to *knowledge* rather than *force*. In *The History of Sexuality, Volume 1*, Foucault states that "power is not something that is acquired, seized, or shared". . . but rather power is "exercised from innumerable points" (1978:94). Since power is unlike a substance that can be seized, shared, or hoarded, "there is no binary and all-encompassing opposition between rulers and ruled" (1978:94). Power always exists with resistance, and "this resistance is never in a position of exteriority in relation to power. . . These points of resistance are present everywhere in the power network" (1978:95). To this end it is better to conceive of power not as a thing to be wielded over another, but as a "moving substrate of force relations," (1978:93) which leads to Foucault's famous statement that "power is everywhere; not because it embraces everything, but because it comes from everywhere" (1978:93).

If power comes from everywhere, what enables or hinders its movement? Foucault's answer is discourse, which "transmits and produces power; it reinforces it, but also undermines and exposes it, renders it fragile and makes it possible to thwart it" (1978:101) . . "Indeed, it is in discourse that power and knowledge are joined together" (1978:100). Foucault draws the connection between power, knowledge, and discourse to question the "repressive hypothesis" of sexuality; namely that contrary to the 17th century when sexual practices were discussed frankly and without secrecy, the 19th century ushered in a time of Victorian bourgeoisie sexuality. The only decent form of sexuality became the marital bed for purposes of procreation, and talk of sexuality was prohibited and censored. Foucault counters this hypothesis by noting that the 19th century also saw a "steady proliferation of discourses concerned with sex" (1978:18). He does not mean that people used more sexually graphic or profane language, but rather that there were more *institutional incitements* to speak about it.

Take for example the Catholic sacrament of confession. In the 19th century the church encouraged confessors to divulge their every thought, dream, desire, and transgression regarding sexuality. With the rise of medicalization doctors and psychiatrists increasingly took the place of the priest, while the patient took the place of confessor in the production of "truth." Sex therefore, rather than being prohibited and repressed "was taken charge of, tracked down as it were, by a discourse that aimed to allow it no obscurity, no respite" (1978:20). Medical professionals specified, classified, and incorporated aberrant sexualities into discourse of health and pathology. They classified heterosexual, monogamous, marital, and procreative sex as healthy while deeming all other forms of sexuality pathological. Foucault summarized these varying positions. He writes that "the essential point is that sex was not only a matter of sensation and pleasure, of law and taboo, but also of truth and falsehood, that the truth of sex

became something fundamental, useful, or dangerous, precious or formidable: in short, that sex was constituted as a problem of truth" (1978:56).

This explication of Foucault's definition of power and its relation to knowledge and discourse is necessary for understanding how power operates within sexuality research. Following Foucault and feminist standpoint theory I believe that knowledge is always produced within of relations of power, never from a universally objective perspective. I argue that rather than claiming the knowledge we produce is Truth, we must examine our social positions and ontological assumptions, and communicate them with our research. Feminist Philosopher Harding refers to this practice as "strong objectivity," which is opposed to the "weak objectivity" of those who claim a completely objective position consistent with a positivist approach to research (2006:85).

While Foucault's definition of power is useful for seeing how power operates in sexual health research, his work has limitations with regard to racialized sexuality. Critical scholars JanMohamed and Stoler (1995; 1995) pointed out Foucault's lack of engagement with race and colonialism even though they draw on his deconstructive tools for those topics. In light of this limitation of Foucault's work, I bring in theories from feminist researchers Higgenbotham and Hammonds to explain how sexuality is always already racialized, and how researchers can integrate this understanding into their work. The role of power in knowledge production is key to Foucault's, Higgenbotham's, and Hammonds' work, and thus serves as a bridge between critical medical sociology and critical race theory.

Keeping in mind the concepts of objectivity, knowledge, and power, I now discuss the ways knowledge—as Foucault conceptualizes it-- about sexuality is racialized. I start by

introducing Higgenbotham's concept of race as a metalanguage; as the "powerful, all-encompassing effect on the construction and representation of other social power relations, namely, gender, class, and sexuality" (1992:252). By viewing race as a metalanguage it becomes impossible to detach and analyze apart from the other identities it influences. We therefore avoid discussing gender as a concept distinct from race; we can only examine gendered race, or racialized gender. This approach approximates an intersectional analysis, except that a purely intersectional approach views all categories of difference (gender, race, sexuality, age, nation, etc) as ontologically equal, no one category more important than the next (Hancock 2016), while a metalanguage of race highlights race specifically.

Higginbotham discusses the connection between race and gender by pointing to the categories of "black women" and "white ladies" in the Jim Crow South prior to the 1960s. The idea of a "black lady" was inconceivable. Likewise, the word welfare queen conjures the image of a black female-headed family, even though more people who receive aid for dependent children are white (Higgenbotham 1992). Other contemporary examples include terms like "baby mama" or "suburban housewife," which imply black mothers and middle-class white women, respectively.

Bringing in discussion of sexuality, Higginbotham cites the discursive trend in the Renaissance to construct women's sexuality as lascivious and carnal, compared to the opposite image which emerged in the Victorian era of women as the keepers of moral purity. She notes that this shift to moral purity only occurred for white women's sexuality, while black women continued to be perceived as primitive and animal-like. Higginbotham notes that this was part of a binary system of classifying black people and white people as "carnality as opposed to intellect and/or spirit; savagery as opposed to civilization; deviance as opposed to normality; promiscuity

as opposed to purity" (1992:263). These racialized constructions of sexuality rationalized the violence of both slavery and sexual exploitation.

One example of sexual exploitation in the name of science comes from a now infamous study from the Public Health Service, a federal agency: the Tuskegee Syphilis Experiment. Starting in 1932 the government began a decades-long study on the effects of syphilis on black men who were unknowingly inoculated, and denied penicillin, the cure (Higgenbotham 1992:266). The knowledge the researchers presented had the effect of "isolat[ing] blacks even further within American society- to remove them from the world of health and to lock them within a prison of sickness. Whether by accident or design, physicians had come dangerously close to depicting the syphilitic black as the representative black" (Jones 1981:28). The Tuskegee Syphilis Experiment produced knowledge which further justified stereotypical depictions of black sexuality.

The proliferation of misleading, controlling, and always negative images of black women's sexuality, which cast them as amoral and promiscuous, lead to what Darlene Clark Hines calls a culture of dissemblance: "the behavior and attitudes of black women that created the appearance of openness and disclosure, but actually shielded the truth of their inner lives and selves from their oppressors" (Guy-Sheftall 1995:380). Dissemblance involved a self-imposed invisibility of their inner lives, downplaying or deny sexual expression, and the creation of a new image of black women as supermoral women (Guy-Sheftall 1995). The newly created discourse of the supermoral black women resisted and undermined knowledge which cast black women as amoral and promiscuous in relation to white women's sexual purity, but it also kept black women's sexual subjectivity invisible.

Sexuality researchers produce knowledge of sexuality within this social and historical context in the United States. While there is prolific research on sexuality, it often excludes discussion of race, and therefore implies a white universal. Hammonds notes how "black women's sexuality is often described in metaphors of speechlessness, space, or vision, as a 'void'; or empty space that is simultaneously ever visible (exposed) and invisible and where black women's bodies are always already colonized" (1994). Furthermore, Hammonds offers two pieces of advice for sexuality researchers, which help decenter whiteness as the universal, and contribute to positive images of black female sexuality:

> It seems to me that there are two projects here that need to be worked out. White feminists must re-figure (white) female sexualities so that they are not theoretically dependent upon an absent yet-ever-present pathologized black female sexuality. I am not arguing that this figuration of (white) female sexuality must try to encompass completely the experiences of black women, but that it must include a conception of the power relations between white and black women as expressed in the representations of sexuality. . . Black feminist theorists must reclaim sexuality through the creation of a counternarrative that can reconstitute a present black female subjectivity and that includes an analysis of power relations between white and black women and among different groups of black women. In both cases I am arguing for the development of a complex, relational but not necessarily analogous, conception of racialized sexualities (1994:130).

Hammonds argues that researchers must examine power relations within and among racial groups. To white feminists she explicitly cautions against constructing female sexualities without discussing their racialized dimensions, because a colorblind approach still implies a white universal and black Other.

Following Hammonds, Clark Hine, and Higgenbotham, I view research which fails to explicitly discuss or analyze race always in danger of universalizing whiteness. Research on sexuality, particularly the overtly medicalized study of sexually transmitted infections, must meaningfully engage with the history of sexualized and institutionalized violence. A structuralist approach to health disparities is one way to highlight the power relations between black and

white women: it reveals how resources are inequitably distributed and give women different opportunities to prevent and treat illness. When we pair a structural approach with the conception of race as a metalanguage we can reveal how this differential access to power connects to constructions and narratives of female sexualities which are racialized and theoretically dependent on one another. Researchers, particularly those with race and/or gender privilege, must consciously avoid contributing to the material and ideal legacies of patriarchal white supremacy which continue to influence research and inform health policy.

In the following section I review literature on the health effects of HPV, the statistical distribution of HPV by race, and studies which use statistical mechanisms to determine the most predictive variable of HPV. These studies fail to engage with race, and their analyses point to promiscuity (greater numbers of sex partners) as a main predictor of HPV. I believe this research unintentionally contributes to constructions of white female sexuality as normative, and black female sexuality as deviant.

The current study

This section begins with a brief overview of HPV and its effects on health. Then it discusses disparities in HPV by race, and reviews literature on the cause of HPV, focusing specifically on studies which use National Health And Nutrition Examination Survey data. The final section reviews my empirical and theoretical critiques of the causal analysis of HPV rates which leads to my specific research question.

HPV is the most common sexually transmitted infection in the United States (Satterwhite et al. 2013). Researchers from the Center for Disease Control and Prevention (CDC) estimate that 80%-90% of adult men and women will have HPV at some point in their lifetime (Chesson

et al. 2014). In 2013-2014 an estimated 42% of adults 18-69 had HPV (McQuillan et al. 2017). In most HPV cases it clears within two years, often going unnoticed (cancer.gov). HPV strains are grouped into low-risk and high-risk strains, with low-risk strains responsible for genital warts, and high-risk strains associated with nearly all cervical cancer (cancer.gov); 70% of oropharyngeal cancers (Chaturvedi et al. 2011); 88% of anal cancers and anal intraepithelial lesions (Hoots et al. 2009); and 48% of penile squamous cell carcinoma (Backes et al. 2009). Strains 16 and 18 alone are responsible for 70% of all cervical cancer (Grulich et al. 2010). The prevalence of HPV among various cancers highlights the need to research prevention and intervention strategies.

There are two prevention strategies to protect against HPV-associated cancers. One is to get the HPV vaccine before becoming sexually active. Gardasil, approved in 2006, and Cervarix, approved in 2009, both protect against strains 16 and 18 (Gelman et al. 2013). There is also a quadrivalent vaccine targeting strains 6, 11, 16, and 18, and a 9-valent vaccine targeting 6, 11, 16, 18, 31, 33, 45, 52, and 58 (2015). Beginning in 2006 the CDC recommended that all boys and girls ages 10-12 receive a 2-dose vaccination, and for those previously unvaccinated, for women up to age 26 and men up to age 21 (2015).

The second preventative measure is monitoring HPV status. Cervical cancer is highly preventable with regular Papanicolaou (Pap) tests to detect early stages of precancerous squamous cells (2015) and Loop Electrosurgical Excision procedures (LEEP) to remove cancerous cells, preventing the development of invasive cervical cancer (cancer.gov). Although Pap tests have decreased the incidence and mortality of cervical cancer, cervical cancer remains the eighth most common cancer among women in the United States (Society 2013).

HPV prevalence is common among all demographics, but HPV prevalence, strain type, and associated cancers are distributed differentially throughout the population by race, gender, class, and region (Barnholtz-Sloan et al. 2009; D'Souza et al. 2014; Dunne et al. 2007; Gillison et al. 2012; Hariri et al. 2012; Liu et al. 2016; Markowitz et al. 2013; Markowitz et al. 2009; Niccolai et al. 2013; Petersen et al. 2017; Vidal et al. 2014). Tables 2.1 and 2.2 show HPV prevalence by race, gender, and strain type to illustrate some of these differences.

Table 2.1: Differences in HPV prevalence by race, women ages 18-69

HPV prevalence, women	**White**	**Black**	**Hispanic**	**Years**
Vaginal, any strain	36.5%	63.2%	38.5%	2013-2014
Vaginal, high risk strain	18.7%	28.2%	21.6%	2013-2014
Oral, any strain	2.9%	4.5%	4.1%	2011-2014
Oral, high risk strain	1.1%	1.7%	1.3%	2011-2014

NHANES (McQuillan et al. 2017)

Table 2.2: Differences in HPV prevalence by race, men ages 18-69

HPV prevalence, men	**White**	**Black**	**Hispanic**	**Years**
Penile, any strain	43.7%	65%	44.4%	2013-2014
Penile, high risk strain	24.7%	40.3%	21.8%	2013-2014
Oral, any strain	11.7%	15.8%	9.9%	2011-2014
Oral, high risk strain	7.3%	7.5%	5.4%	2011-2014

NHANES (McQuillan et al. 2017)

We can note four broad trends: First, men of every racial group, and of every type and location of HPV, have a higher prevalence of HPV than women. However, this data shows the average HPV prevalence of men and women across all ages 18-69. When the data is desegregated by age, women have higher rates of genital HPV than men from ages 14-24, but

lower rates after age 25 (Lewis et al. 2018). Second, black men and women have a higher prevalence of HPV than whites and Hispanics. Third, the difference between black and white men and women is greater than the difference between men and women of either race. Fourth, the difference in HPV between races is greater for any strain of HPV than just the high-risk strains of HPV.

Given the high prevalence of HPV and its association with various cancers, there is much research on demographic and behavioral HPV correlates (D'Souza et al. 2014; Dunne et al. 2007; Gillison et al. 2012; Markowitz et al. 2009). Understanding the variables associated with HPV can help medical sociologists, epidemiologists, and public health researchers implement the most effective prevention strategies. Most studies focus on vaccine-type strain prevalence, and/or all strains of HPV, and some examine the individual strains separately. Virtually all research on HPV separates analysis between men and women. While race is almost always included as a co-variate in regression models, it is often not the specific focus of the study, even though the differences in HPV prevalence are often greater between races than between genders. This inattention to race yields blind spots in the results and creates implications about racialized sexuality.

I now outline four different studies which use multivariate logistic regression to determine which variables best predict HPV prevalence. They all use NHANES data and begin by using bivariate analysis followed by multiple regression. Most centrally, these studies illustrate a quantitative approach to determining distal and proximate correlations with HPV. The only social context they provide is through co-variates such as race, class and gender. The only measure they use to establish relationships between variables is statistical significance. Following Link and Phelan's work and advise to researchers I argue that identifying which

variables become insignificant in multivariate models is insufficient to determine full cause; we must also seek to explain why a variable like race/ethnicity becomes insignificant, and which mechanisms cause this difference in the first place. Table 2.3 summarizes the studies.

Table 2.3: Studies which use NHANES and multivariate regression to predict HPV risk

Study	Type of HPV	Bivariate correlations	Multivariate correlations
Dunne et al. 2007, NHANES 2003-2004	Vaginal HPV, all strains, high-risk and low-risk, women	Age, poverty index, education, marital status, number of recent sex partners, lifetime number of sex partners, **race/ethnicity**	Age younger than 25, marital status, number of recent and lifetime partners
Markowitz et al. 2009, NHANES 2003-2004	Vaginal and penile HPV, strains 6/11/16/18, women and men	Poverty level, history of smoking, age at sexual debut, number of lifetime sex partners, number of sex partners in last year, history of sex with same-sex partner, history of genital warts diagnosis, **race/ethnicity**	Age, poverty*, lifetime number of sex partners *only among women
D'Souza et al. 2014, NHANES 2009-2010	Oral HPV, all strains, type 16, men and women	Gender, age-cohort, sexual behavior, **race/ethnicity**	Gender, age-cohort, sexual behavior
Chaturvedi et al. 2015, NHANES 2009-2010	Oral HPV, all strains, high-risk, and low-risk, men and women	Age, gender, serum cotinine, number of lifetime and recent sex partners, **race/ethnicity**	Gender, age, current cigarette use, number of lifetime sexual partners

These four studies include analysis of both genital and oral rates of HPV, all using the same data source, including results from the 2003-2004 and 2009-2010 cycles. Each study found that race/ethnicity, in addition to other variables such as age, gender, poverty index, and number of sex partners were correlated with a positive HPV result in bivariate regression models. In other words, differences in race (or age, or number of sex partners, etc) had statistically significant differences in HPV prevalence. However, once the researchers ran multivariate

regression models, which show the correlation of each variable after controlling for all the others, race/ethnicity was found no longer statistically significant. Only gender, age, and sexual behavior remain independently associated with HPV throughout each study.

Theoretical and empirical critiques

My research departs from Dunne et al. (2007), Markowitz et al. (2009), D'Souza et al. (2014) and Chaturvedi et al (2015) on several theoretical and empirical grounds: First, I follow Hammonds' advice to feminist researchers to develop "a complex, relational but not necessarily analogous, conception of racialized sexualities" (1994:130). To this end I focus on black women and white women, rather than women in general, which would run the risk of universalizing whiteness and creating unintended implications about black and white female sexualities. Researchers can conduct relational analyses between any two racial or ethnic groups, for *only* relating black and white populations runs the risk of reifying a black/white racial binary. We could also compare constructions of white and Native women's sexualities, or black and Asian men's sexualities. But for my research focusing on black and white women is most appropriate because black and white racial groups have the most drastically different HPV (and STI) rates, and there is an important history of sexuality research which was used as a tool to control black and white women's sexualities in different ways.

Second, I attend to power relations between and among women by looking at differential access to resources. This leads me to incorporate Link and Phelan's concept of social conditions as the fundamental cause of health inequalities. I follow Link and Phelan's advice to medical sociologists, that "if the social factor is a fundamental cause, one cannot claim to have accounted for its effects by having 'explained' its association with the inclusion of intervening variables in

a path or regression model" (1995:88); and second, to "examine the broader determinants of the resources" (1995:88). If race is a fundamental cause of different rates of STIs, researchers need to examine the context in which STIs are transmitted, and the context that may foster behavioral differences. Regardless of sexual behavior, resources such as regular Pap and HPV tests, HPV vaccination, or pre-cancerous cell removal can prevent or treat HPV.

Lastly, I question the four studies in table 2.5 for two empirical and methodological reasons which I discuss in detail below: First, the differences between white and black women's risk behaviors may not explain the full difference in HPV rates, which is suggested by trends in sexual behavior and HPV rates, and previous research which has found that differences in risk behaviors do not account for differences in other sexually transmitted infections (STIs) (Ellen, Aral and Madger 1998; Hallfors et al. 2007; Halpern et al. 2004; Harawa et al. 2003; Santelli et al. 2000). Second, there is evidence that demographic characteristics such as sex-ratios influence sexual behavior and networks, which in turn influences STI outcomes, and that racial segregation impacts STI outcomes. These features are important indicators that race could be a fundamental cause of inequality in STIs.

The following tables show trends in HPV risk behaviors comparing white women and black women. While there are differences by race, they fail to always match trends in HPV prevalence. Consider the data in Tables 2.4 and 2.5 on number of sex partners for different types of sex:

Table 2.4: Mean number of sex partners for women ages 20-59

Type of sex	White	Black
Any sex	8.3	9.3
Performed oral sex	4.5	2.5

NHANES 2009-2010 (D'Souza et al. 2014)

Table 2.5: Percentage of women ages 20-59 who reported a given number of sex partners

# of partners	Any sex, white	Any sex, black	Performed oral, white	Performed oral, black
0	1.7%	3.5%	7%	32.8%
1-2	23.6%	13.1%	43.2%	36.2%
3-5	30.9%	31.8%	27.4%	22.8%
6-10	24.7%	33%	15.3%	5.8%
10+	19%	18.6%	7.1%	2.3%

NHANES 2009-2010 (D'Souza et al. 2014)

We can see from these tables that black women have on average more sex partners when considering all types of sex, and white women have a greater number of sex partners on whom they have performed oral sex. If number of sex partners determines likelihood of HPV, we would expect black women to have a higher prevalence of vaginal HPV, and white women to have a higher prevalence of oral HPV. However NHANES data from 2011-2014 reports that black women have more vaginal and oral HPV than white women (63.2% versus 36.5%, and 4.5% versus 2.9%) (McQuillan et al. 2017).

This trend is also demonstrated in data from the National Survey of Family Growth. See Tables 2.6 and 2.7.

Table 2.6: Percentage of women ages 15-44 who reported a given number of sex partners

Number of opposite-sex sexual partners in lifetime (all types of sex)	**White**	**Black**
1	19.2%	12.3%
2	9.7%	8.3%
3-6	31.4%	40.9%
7-14	18.9%	16.7%
15+	8.9%	11.3%

NSFG 2006-2008 (Chandra et al. 2011)

Table 2.7: Percentage of women ages 15-24 to ever have engaged in the following behaviors:

Activity	White	Black
Vaginal intercourse		
Ages 15-19	42%	53.1%
Ages 20-24	80.6%	93.4%
Gave oral sex		
Ages 15-19	42.6%	22.5%
Ages 20-24	77%	66%
Received oral sex		
Ages 15-19	43%	35.4%
Ages 20-24	80.3%	81%

NSFM 2006-2008 (Chandra et al. 2011)

A similar trend exists when comparing age of sexual debut, or age at first sexual intercourse. See Table 2.8.

Table 2.8: Mean age at sexual debut for women ages 20-59

Mean age at first:	White	Black
Sex act (any)	17.6	16.7
Performed oral sex	19.6	21.9

NHANES 2009-2010 (D'Souza et al. 2014)

While black women have a younger age of sexual debut for any sex act, white women first perform oral sex at a younger age. Other risk factors for oral HPV include tobacco use and drinking alcohol. See Table 2.9 for data from the Youth Risk Behavior Survey which shows alcohol, tobacco, and sex risk behaviors for students in grades 9-12.

Table 2.9: Oral health risk behaviors for female students grades 9-12

Risk behavior	White	Black
Current cigarette use (20 out of last 30 days)	4.4%	0.8%
Current daily cigarette use	3.1%	0.4%
Any tobacco (cigarette cigar, smokeless tobacco, electronic vapor product)	29.4%	21.2%
Current alcohol use (last 30 days)	35.3%	25.9%
Consumed 5 or more drinks in a row, last 30 days	18.6%	9.9%

YBRS 2015

White women report engaging more in every category of oral health risk than black women. This would further suggest that white women would have higher rates of HPV prevalence than black women, assuming behavior causes differences in HPV rates.

Researchers have noted incongruencies between risk behaviors and STIs other than HPV. Three different studies examined the correlation between risk behaviors, race, SES, and prevalence of STIs and found that while black students engaged in somewhat more risk behaviors than their white counterparts, the difference in behavior explained only some of the difference in STIs (Ellen, Aral and Madger 1998; Harawa et al. 2003; Santelli et al. 2000). Two other studies used data from different waves of the National Longitudinal Study of Adolescent Health and categorized adolescents into a series of risk-groups according to sexual behaviors and drug and alcohol consumption, and in both cases black women were most likely to be in the lower-risk behavior groups, but still had the highest risk of STIs (Hallfors et al. 2007; Halpern et al. 2004).

In addition to studies which explore the relationship between risk behaviors and STIs, researchers have examined connections between demographic characteristics and risk behaviors,

and demographic characteristics and STI rates. There is evidence that structural factors such as skewed sex ratios and racial housing segregation for black Americans contribute to higher risk behavior rates and STIs. Pouget wrote that adult sex ratios can become unbalanced due to unbalanced sex ratios at birth, excess male mortality, military service, non-heterosexual identity, employment related migration, and mass incarceration (2017). Excess male mortality, military service, and mass incarceration disproportionately affect black Americans, an effect which is explicitly racialized, not just connected to class. The effects of such imbalance include greater number of sex partners for men, and greater mixing between peripheral and core members of sexual networks, leading to a higher incidence of HIV and STIs (Laumann and Youm 1999; Pouget 2017). The effects of housing segregation, increased military involvement, and over-policing low-income and racial minority neighborhoods are all structural factors which skew sex ratios, putting black communities at greater risk.

Evidence of greater mixing within sexual networks comes from Adimora et al. who researched the sex ratio effects on likelihood to have concurrent partners (2013). She found that 99.5% of whites, but only 7.85% of blacks live in balanced sex ratio counties (among their own race); and the odds ratios for concurrent relationship prevalence was 1.67 in counties with low sex ratios, compared to counties with balanced sex ratios (Adimora et al. 2013)

Henderson provides evidence that racial segregation can contribute to racial differences in STIs by mapping the relationship between racial segregation and chlamydia rates (2015). After controlling for region, college graduation rates, sex ratios, unemployment rates, median income, population density and income inequality, regression models found that in predominantly white counties both whites and blacks had lower levels of chlamydia, whereas in

predominantly integrated/black counties, whites still had lower rates of chlamydia, but blacks had higher rates (Henderson 2015).

There is evidence that sexual behaviors vary by race, but the differences in behavior fail to match the trends in HPV prevalence. There is also evidence that racial differences in sexual behavior do not fully account for distribution differences of other sexually transmitted infections. And there are structural factors such as sex ratios and racial segregation which put blacks at higher STI risk. All this evidence leads me to question the studies which highlight number of sex partners as the causal mechanism of HPV.

With these considerations in mind I call into question NHANES data on HPV prevalence among white women and black women, following Link and Phelan's theory of fundamental causes and their advice to researchers, and taking the social context of power relations and risk factors into consideration. I specifically consider how power relations construct white and black female sexualities and the political real-life implications such research creates.

CHAPTER 3: METHODOLOGY

The purpose of this methods chapter is to present the research questions and hypotheses I developed to examine the relationship between behavioral and structural predictors of HPV among black and white women. I first describe the history and methodology of the data source that I use: The National Health and Nutrition Examination Survey. Then I present my research questions and hypotheses. I then discuss how I prepared the dataset and variables, and the statistical analysis I used to answer my research questions. Lastly, I outline the limitations of some variables and the statistical analyses.

Background on the National Health and Nutrition Examination Survey

In 1956 the 84th congress passed the National Health Survey Act which stipulated that health information be regularly collected on the US civilian non-institutionalized population. This prompted the Center for Disease Control and Prevention to begin developing three waves of the National Health Examination Surveys, the first of which was conducted in 1960 and targeted adults. The subsequent waves focused on young children and adolescents and were released later that decade ("NHANES Tutorial").

In 1971 the CDC added a large nutritional component to the survey which was then renamed the National Health and Nutrition Examination Survey (NHANES). They administered three waves of the survey through the 1970s and 1980s which focused on different age groups; and conducted an extra study of the Latinx population in 1982, called the Hispanic HANES (HHANES). In 1999 the CDC began the continuous NHANES which they release every two years and includes non-institutionalized civilians of all ages. The fields of public health,

epidemiology, nutrition, and genetics have and continue to use NHANES data to estimate disease and health risk prevalence throughout the population ("NHANES Tutorial").

The survey process for continuous NHANES includes three components: an in-person interview, a Mobile Examination Center (MEC) examination, and a follow-up questionnaire. The in-person interview consists of the initial household screening to determine whether anyone in the household is eligible to participate in the interview and examination and is followed by an interview to collect demographic information, health and nutrition information, and household-level information. The MEC examination includes collecting blood and urine samples, physical measurements, and a dental examination. Selected participants also complete a Computer-Assisted Self Interview (CASI) questionnaire on drug use or sexual behavior. Participants who completed the MEC dietary questionnaire later complete a follow-up Computer-Assisted Telephone Interview (CATI) and answer a food frequency questionnaire (Johnson et al 2014).

NHANES uses a complex four-stage probability sampling design to produce nationally representative aggregate-level estimations. In the first stage they select approximately 15 Primary Sampling Units (PSUs) of all US counties with the probability of selection proportionate to population size. In the second stage they sample approximately 360 individual or multiple census blocks of approximately equal size. The third stage involves sampling 13,500 dwelling units (DUs) including houses, apartments, and dormitories to determine which households will be screened for participation in the survey. In the fourth stage they select 11,500 households among the DUs to be screened to determine eligible participants, of which approximately 5,000 are examined (Johnson et al. 2014).

NHANES over-samples racial and ethnic minorities, persons at or below 130 percent of the federal poverty level, and white and other persons aged 80 years and over to increase reliability and precise subpopulation estimates. They have defined the race/ethnicity categories differently throughout the years, with the current classifications described as “Hispanic persons, Non-Hispanic Black persons, Non-Hispanic Asian persons, Non-Hispanic White, and Other (NCHS 2013).” The categories of Asian, Hispanic, Non-Hispanic White and Non-Hispanic Black are defined as mutually exclusive, and the category “Other” includes all other racial and ethnic groups as well as multi-racial persons. The strict racial and ethnic categorizations, as well as homogenization of multi-racial and Native persons, is a serious limitation of the survey design.

Although each year is nationally representative, the limited number of PSUs throughout the country poses a risk of participant identification, so the data is released in two-year cycles. The analytic guidelines recommend using four years of data for the most reliable estimates.

The current study focuses on specific variables represented and measured by the survey including demographic information, access to health care, oral health, sexual behaviors, and laboratory tests of oral and vaginal HPV. Demographic and socioeconomic information is collected during the in-person interview, while risk behaviors are collected during the CASI in the MEC along with self-collected vaginal swabs and oral rinses. For vaginal HPV, technicians use the Linear Array HPV Genotyping Test to determine the number of viral genomes in the sample and their strain. The oral rinse samples use the Roche Linear Array HPV Genotyping Test. Both tests test for 37 different HPV genotypes: 6, 11, 16, 18, 26, 31, 33, 35, 39, 40, 42, 45, 51, XR (52), 53, 54, 55, 56, 58, 59, 61 62, 64, 66, 67, 68, 69, 70, 71, 72, 73 (MM9), 81, 82 (MM4), 83 (MM7), 84 (MM8), IS39, and 89 (CP6108).

Research questions and hypotheses

The purpose of my thesis is to examine the causal relationship of individual behaviors and structural factors in determining racial disparities in rates of HPV. My first questions are intended to orient myself to the descriptive data provided by the survey including the baseline prevalence of oral and vaginal HPV, risk behaviors, and SES measures. Since I am comparing my research to previous studies and survey results I need to establish how close my data is to previous NHANES years.

My first research hypothesis examines the differential effect individual risk factors may have by race. This relates to previous research which found that black women with low-risk behaviors are at similar risk for STIs as their white and male counterparts who engage in high-risk behaviors (Hallfors et al. 2007; Halpern et al. 2004; Harawa et al. 2003; Santelli et al. 2000). These studies examined STIs other than HPV, and I believe there may be the same effect with HPV.

Hypothesis 1: Sexual risk behaviors will have a greater effect on oral and vaginal HPV prevalence for white women than black women.

My second research hypothesis examines the differential effect SES measures may have by race. This relates to Link and Phelan's theory of social conditions as the fundamental cause of health inequalities. By looking at the differential effects of SES by race I can determine if HPV rates vary just by access to resources, just by race, or both. I believe it is possible that race, and the conditions and experiences related to being black or white in the United States, cause disparities in HPV beyond differences in social class.

Hypothesis 2: Socioeconomic status will have a greater effect on HPV prevalence for black women than white women.

The answers to these research questions and hypotheses help me determine the role of individual risk behaviors and access to resources, as well as their interactions with race, in predicting rates of HPV. My analysis focuses specifically on racial difference so I can see if white and black women have the same predictive variables of HPV. By looking at rates of both oral and vaginal HPV I can see how the individual and structural variables work in one context where white women engage in more risk behaviors (oral sex behavior), and another context where black women engage in more risk behaviors (vaginal sex behavior).

Preparing the dataset

For this study I use public-release survey data from 2011-2014. The public release data excludes sexual behavior, drug use, and HPV results for participants under the age of 18, but includes results for adults. For confidentiality the public release data also excludes the true Primary Sampling Units and instead supplies variables for masked variance pseudo-PSU and masked variance pseudo-stratum, which hide the true location of the participants, but allow researchers to calculate reliable variance estimates.

To create the dataset I combined data from the 2011-2012 and 2013-2014 NHANES cycles. I first viewed the available NHANES variables on the CDC National Center for Health Statistics (NCHS) website and selected variables relevant to my research question. I ultimately chose 25 variables from 7 different files containing various aspects of the survey: demographics, alcohol use, cigarette use, health insurance, HPV-oral rinse, sexual behavior, and HPV-vaginal swab. I downloaded the .XPT files from the NCHS website for both year cycles, opened them in Stata 15, kept only the variables relevant to my analysis, and saved them as .dta files.

In preparing my dataset I first appended each 2011-2012 file with its 2013-2014 equivalent, and then merged all files using *seqn* as the key variable. I then renamed the variables and created labels for easier interpretation. I recoded responses of "refused," "don't know," and "inadequate" as missing. I then generated new variables from the originals to suit my analysis; for example, I created one continuous variable *drinksperweek* from four different variables in the alcohol use file to indicate the average number of alcoholic drinks the participant imbibes each week. Table 3.1 shows each of the original files, variables, and descriptions, as well as the variables I generated from them.

Table 3.1: Original NCHS files and generated variables

Original file and variable name	**Variable rename and original level of measurement** (bi=binary, con=continuous, cat=categorical)	**Generated variables**
File: Alcohol use		
alq110 *Had at least 12 alcohol drinks/lifetime?*	**drinks12** (bi)	**drinksperweek** *avg # drinks/week* (con)
alq120q *How often drink alcohol over past 12 mos*	**alcq** (con)	
alq120u *# days drink alcohol per wk, mo, yr*	**wmd** (cat)	
alq130 *Avg # alcoholic drinks/day - past 12 mos*	**alcday** (con)	
File: smoking - cigarette use		
smq020 *Smoked at least 100 cigarettes in life*	**cigs100** (bi)	**smoker** *smoke cigarettes everyday* (bi)
smq040 *Do you now smoke cigarettes*	**nowsmoke** (bi)	
File: Demographic Variables and Sample Weights		
riagendr g*ender of the participant*	**gender** (bi)	
ridageyr a*ge in years at screening*	**age** (con)	**college** *graduated college or more* (bi)
ridreth3 *Race/Hispanic origin w/ NH Asian*	**race** (cat)	**hsgrad** *graduated high school or more* (bi)
dmdeduc2 *Education level - Adults 20+*	**educ20** (cat)	**somecollege** *attended some college or more* (bi)
wtint2yr *Full sample 2 year interview weight*	--	**wtmec4yr** *sample weights for combined years*
wtmec2yr *Full sample 2 year MEC exam weight*	--	
sdmvpsu *Masked variance pseudo-PSU*	--	
sdmvstra *Masked variance pseudo-stratum*	--	
indfmpir *Ratio of family income to poverty*	**fampovrat** (con)	
File: Health insurance		
hiq011 *Covered by health insurance*	**anyhi** (bi)	

File: Human Papillomavirus (HPV) - Oral Rinse		
orxhpv *Oral HPV Result*	**oralhpv** (bi)	
File: Sexual behavior		
sxq700 *Ever had vaginal sex with a man*	**evervag** (bi)	
sxq703 *Ever performed oral sex on a man*	**everoral** (bi)	
sxd031 *How old when first had sex*	**debut** (con)	
sxd621 *How old when first had oral sex*	**agefell** (con)	
sxq624 *# male oral sex partners/lifetime*	**oralnum** (con)	**oralnumsr** *square root of oralnum* (con)
sxq724 *# male vaginal sex partners/lifetime*	**vagnum** (con)	**vagnumsr** *square root of vagnum* (con)
sxq251 *# times had sex without condom/year*	**vcondom** (cat)	**condom** *use frequency* (bi)
File: (HPV) DNA - Vaginal Swab: Roche Linear Array		
lbdrpcr *Roche HPV linear array summary result*	**rochesumv** (bi)	

I transformed the number of oral partners and number of vaginal partners variables to the square root of number of partners for each. Previous research has found that number of sexual partners has decreasing significance in HPV prediction at higher numbers, and nearly levels out at the 95th percentile (Chaturvedi et al. 2015). For example, the increase in risk would be greater between 1 and 2 partners than between 31 and 32 partners.

Transforming the variables to square roots affects how we interpret the slope. Normally with logistic regression we interpret the slopes by a one unit change in the explanatory variable: for example, for every one person increase in number of sex partners the risk of HPV increases by 8%. When the variable is transformed to a square root, rather than increasing by one unit, the

effect reflects changes in square units. In the context of this study this means the effect for number of partners changes by increasingly large intervals: 1-3 partners, 4-8 partners, 9-15 partners, 16-24 partners, 25-35 partners, etc.

A transformed variable can be included with the original untransformed variable, or not. As you will see in the results chapter I include both versions for number of oral partners, but not for number of vaginal partners, because I found the pseudo-R2 was greater when using just the transformed variable for number of vaginal partners.

For this study I used the pseudo-R2 measure developed by McKelvey and Zavoina (1975). They originally developed their measure for ordinal-level dependent variables. I ultimately chose the McKelvey-Zavoina pseudo-R2 because researchers Veall and Zimmerman (1994) evaluated several pseudo-R2 methods for binary probit models and found that McKelvey and Zavoina's most closely resembled OLS R2.

Table 3.2 shows my final variables and how they are defined.

Table 3.2: Final variables and definitions

oralhpv (dependent)	0=no HPV types detected 1=at least one HPV type detected
rochesumv (dependent)	0=no HPV types detected 1=at least one HPV type detected
age	continuous, age upon survey administration: 20-59
black	0=white 1=black
drinksperweek	continuous, average number of alcoholic drinks consumed per week in the 12 months before the survey: 0-164
smoker	0=never smoked, or does not currently smoke 1=smokes cigarettes daily
hsgrad	0=did not graduate high school or complete Generate Education Diploma (GED) 1=graduated from high school or completed GED
fampovrat	continuous, measures percent above or below federal poverty guidelines for household: 0-5
anyhi	0=no health insurance 1=at least some type of health insurance
debut	continuous, age at sexual debut: 9-60
agefell	continuous, age at first performance of oral sex on a male: 9-56
oralnum	continuous, number of partners on whom performed oral sex: 1-1000
oralnumsr	continuous, square root of number of partners on whom performed oral sex: 1-31
nooral	0=has performed oral sex on at least one male partner 1=has never performed oral sex on a male partner
vagum	continuous, number of vaginal sex partners: 1-500
vagnumsr	continuous, square root of number of vaginal sex partners: 1-22
nosex	0=has had at least 1 vaginal sex partner 1=has had 0 vaginal sex partners
condom	0= uses condoms half of the time or less 1= uses condoms at least more than half of the time

Data analysis

I used four different techniques for statistical analysis: First I generated descriptive summary statistics, then I ran a series of bivariate logistic models to note initial correlation and significance, then I used multivariate logistic regression to measure the effect of variables before and after introducing race and number of partners, and lastly I checked for interactions between each variable and race using two different methods: first by introducing interaction terms to the multivariate model, and second, by dividing the sample by race into subsamples of just black women and just white women. I compared the results of these two methods.

To generate my descriptive results I used summarize and tabulate commands in Stata 15 to provide me with means, proportions, and percentile information on each of the variables I use in the study. I used the survey weights, variance, and stratum variables for every analysis in order to produce results that correct for over-sampling and produce nationally representative results. The CDC created an online tutorial for preparing and analyzing NHANES data which I used as a guide to accommodate survey design.

I then ran bivariate models of every variable for both oral and vaginal HPV. After determining bivariate correlation and significance I used variables relating to individual risk, SES, and age to generate initial models for vaginal and oral HPV. Next I ran a model which controlled for race, and a model which controlled for number of partners, and then a model with both race and number of partners to determine direct and indirect effects. After that I looked for interactions by both introducing interaction terms for each variable with race, and also by dividing the sample by race and comparing coefficients.

Limitations

Conceptualization of "condom use": There are often limitations to researcher conceptualization of quantitative variables and my study has one such variable which could be misinterpreted. Condom use is a literal measure of how frequently the participant uses condoms, *not* a general measure of the participants' adherence to safer sex practices. The survey instrument asked participants how frequently they did not use condoms in the past 12 months and provided broad responses of "never," "less than half of the time," "about half of the time," "not always, but more than half of the time," and "always." I used those responses to create an indicator variable: 1 for those who used condoms more than half of the time, and 0 for those who used condoms half of the time or less. While this may be an accurate measure of condom use frequency, it does not relate to other safer sex practices, or take relative STI risk into account.

Couples in long-term monogamous relationships can forgo condom use without much increased risk, and many do. This variable might more strongly indicate who is involved in more long-term relationships than indicate adherence to safer sex practices. Unfortunately, the survey only includes variables for relationship status as "married," "widowed," "divorced," "separated," "never married," and "living with partner," which excludes serious relationships without marriage or cohabitation. It also asks no questions regarding fidelity or monogamy. Lastly, HPV is spread through skin-to-skin contact, not fluid exchange, so condom use provides less protection against HPV than HIV (Human Immunodeficiency Virus), for example. In short, this variable gives information on condom use without accounting for level of STI risk. It may be a better measure for people who mostly use condoms in casual relationships, rather than safer sex in general.

Comparing logistic regression models and evaluating interactions: The statistical techniques I use in this study have considerable limitations. First of all, we cannot compare the total, direct, and indirect effects of a variable using logistic regression the same way we would using Ordinary Least Squares regression. Because logistic regression models are non-linear, and because they predict probabilities rather than an observable outcome, we cannot reliably compare logit coefficients across models (Williams 2009; Mood 2010). There is more variance between the models than just the addition or subtraction of a single variable. Second, introducing interaction terms or dividing and comparing sample coefficients is further affected by the non-linearity of logistic regression. If we compare coefficients for the same variable between two groups we again introduce more variance than we would with OLS regression. Some researchers argue that interaction terms in logistic regression models are so inaccurate that even the sign and significance levels are unreliable (Ai and Norton 2003). Other researchers have argued that there are relatively simple statistical procedures to correct for possible inaccuracies, such as assuming the groups being compared have the same level of residual variation (Allison 1999), or using probability slopes which are unaffected by residual variance (Long 2009).

Despite these limitations I chose to compare coefficients across models and use interaction terms using logistic regression for two reasons: First, because the comparison studies (Chaturvedi et al. 2015; D'Souza et al. 2014; Dunne et al. 2007; Gillison et al. 2012; Markowitz et al. 2009) compared bivariate and multivariate coefficients across models, and second, because even though the results are far less accurate than they would be with OLS regression, they can still give us a *general idea* of the relationships. And unlike OLS regression, the probabilities will never exceed 1 or fall below 0. Thus, as reflected in my findings discussion later, I assume that a large change in a variable coefficient indicates there is some kind of change in that direction, and

small changes may not actually indicate a change. Likewise, small changes may occur that the models do not reflect.

In the next chapter I provide the results of this analysis.

CHAPTER 4: RESULTS

This chapter outlines the results of my analyses as they relate to my research questions, provides interpretation, and evaluates my research hypotheses. In the first section I provide the results of my descriptive research questions: What are the differences by race in HPV prevalence, SES variables, and individual risk behaviors? In the second section I outline the results of the logistic regression models: First, I show the bivariate correlations of each variable with vaginal and oral HPV as the response variable. Then I present the results of my multivariate models with two different approaches to evaluating interactions between race and the other variables; first for predicting vaginal HPV, and then for predicting oral HPV. In the final section I discuss how well the results support my research hypotheses. I discuss the theoretical implications of the findings in the discussion chapter.

Descriptive results

My first questions are descriptive and establish the baseline prevalence of oral and vaginal HPV, risk behaviors, and SES measures by race. To find these estimates I summarized or tabulated data in Stata 15 using the survey design variables provided in the data set. The results for HPV prevalence are in Table 4.1.

Table 4.1: HPV prevalence for black and white women, ages 20-59

HPV prevalence, %	**Black women** (95% confidence interval)	**White women** (95% confidence interval)
oral	**9.65%** (8.27%-11.02%)	**7.32%** (6.21%-8.43%)
vaginal	**60.37%** *	**35.54%** (32.01%-39.06%)

* Missing standard error because of stratum with single sampling unit.

There is a substantial difference in HPV prevalence between black and white women. The results from my data set are similar to the results McQuillan et al. (2017) found for the same survey years, 2013-14, including participants 18-69 years old. McQuillan et al. (2017) found that 63.2% of black women and 36.5% of white women had vaginal HPV, while 4.5% of black women and 2.9% of white women had oral HPV. My results found that 60.37% of black women and 35.54% of white women tested positive for vaginal HPV, while 9.65% of black women and 7.32% of white women tested positive for oral HPV.

I also tried calculating results without using the study weights, masked variance pseudo-PSU, and masked variance pseudo-stratum variables provided in the demographics file. These variables take survey design into account by adjusting for over-sampling certain minority groups. Differences between the two approaches were large enough that I decided to only use results which include the survey design variables. The drawback to this approach is that once age, race, and gender have been narrowed down the information for black women comes from a stratum with only one sampling unit. This prevented me from calculating standard errors and significance for certain variables.

After finding descriptive results for overall HPV prevalence I calculated the results for the socioeconomic status indicators that I used in the logistic regression models. For the binary variables I include percent prevalence and a 95% confidence interval in parentheses. For the continuous variables I include the arithmetic mean as well as the scores at the 25th percentile, the median, and the 75th percentile, as well as the standard deviation. See Table 4.2.

Table 4.2: SES indicators for black and white women, ages 20-59

SES indicators	Black women (95% confidence interval)		White women (95% confidence interval)	
% with any kind of health insurance	**81.58%** (79.37%-83.80%)		**88.91%** (87.24%-90.58%)	
Ratio of household income to US poverty guidelines	mean: sd: median: 25th percentile: 75th percentile:	**1.99** **1.55** **1.47** **0.75** **3.06**	mean: sd: median: 25th percentile: 75th percentile: (top coded at 5)	**3.12** **1.65** **3.18** **1.55** **5**
% graduated from high school	**80.27%** (77.20%-83.34%)		**89.62%** (86.93%-92.30%)	

These results show that fewer black women report having health insurance than white women, with 81.58% and 88.91% respectively. Similarly, 80.27% of black women and 89.62% of white women reported graduating from high school or completing the GED. While differences by race exist the majority in both groups report having health insurance and graduating high school.

There are larger differences by race when looking at income. I prefer to look at the median and percentiles because income is both skewed to the right, and top-coded at 400% above the poverty line, which disguises any variance at the upper end of the income distribution. While the median income ratio for black women is 47% above the poverty line, the median income for white women is 218% above the poverty line. There are substantial differences at the 25th percentile, where black women make 25% below the poverty line and white women still make 55% above it. Likewise at the 75th percentile black women make 206% above the poverty line, while white women make *at least* 400% above the poverty line.

Overall these results show us that there is a larger difference by race for income than for high school graduation or health insurance. Unfortunately, the variable for health insurance does not include description of insurance quality, only whether any kind of health insurance exists or not.

The third category of descriptive statistics I found were for individual risk behaviors. I compared number of partners, percent who never had oral or vaginal sex, age of sexual debut, smoking, drinking, and condom use between black and white women. As before, for the binary variables I include percent prevalence and a 95% confidence interval in parentheses, and for the continuous variables I include the arithmetic mean, as well as the scores at the 25th percentile, the median, and the 75th percentile, as well as the standard deviation. See Table 4.3.

Table 4.3: Risk behaviors for black and white women, ages 20-59

Individual risk behaviors	**Black women**		**White women**	
average # of vaginal sex partners	mean: sd: median: 25th percentile: 75th percentile:	**8.74** **13.91** **5** **3** **10**	mean: sd: median: 25th percentile: 75th percentile:	**8.60** **20.24** **5** **2** **9**
average # of fellatio partners	mean: sd: median: 25th percentile: 75th percentile:	**3.28** **9.19** **2** **0** **3**	mean: sd: median: 25th percentile: 75th percentile:	**7.10** **40.62** **3** **1** **5**
average age of sexual debut	mean: sd: median: 25th percentile: 75th percentile:	**16.30** **3.24** **16** **14** **18**	mean: sd: median: 25th percentile: 75th percentile:	**17.40** **3.42** **17** **16** **19**
average age of oral debut	mean: sd: median: 25th percentile: 75th percentile:	**21.14** **6.25** **20** **17** **24**	mean: sd: median: 25th percentile: 75th percentile:	**19.16** **5.20** **18** **16** **20**

average # drinks/week	mean: sd: median: 25th percentile: 75th percentile:	**3.80** **9.10** **0.50** **0.04** **4**	mean: sd: median: 25th percentile: 75th percentile:	**4.59** **8.46** **1.25** **0.18** **6**
% smoke cigarettes daily	**19.75%** (17.74-21.75)		**17.31%** (15.23-19.38)	
% who mostly or always use condoms	**45.44%** (42.54-48.34)		**29.16%** (26.79-31.54)	
% never had vaginal sex	**4.54%***		**3.92%** (2.58-5.25)	
% never had oral sex	**30.53%***		**12.48%** (10.53-14.42)	

* Missing standard error because of stratum with single sampling unit.

First I note that the difference in number of vaginal sex partners by race is remarkably small considering the large difference in vaginal HPV prevalence. Black and white women both report a median of 5 vaginal sex partners. Number of partners varies more at the 25th and 75th percentiles with black women reporting 3 partners versus white women reporting 2 and black women reporting 10 while white women report 9, respectively. The standard deviation is greater for white women than black women, at 20.24 and 13.91 respectively.

The differences in vaginal sexual debut between black and white women are somewhat greater, with ages 14 and 16 at the 25th percentile, ages 16 and 17 at the median, and ages 18 and 19 at the 75th percentile. The percentage of black and white women who reported 0 vaginal sex partners is relatively similar, with 4.54% and 3.92% respectively.

Even though black and white women have a smaller difference in oral HPV prevalence (9.65% and 7.32%) their sexual behaviors vary more. The arithmetic means for number of oral partners is 3.28 for black women and 7.10 for white women. The median is closer by race with black and white women reporting 2 and 3 partners, respectively. This suggests that the distribution for white women is especially skewed to the right with more white women reporting

high numbers of oral partners than black women. We can see a 1 partner difference between black and white women at the 25th percentile, 0 and 1, and a 2 partner difference at the 75th percentile, 3 and 5, respectively. As with number of vaginal partners the standard deviation for number of oral partners is greater for white women than black women (40.62 and 9.19).

The average age at oral sexual debut also varies by race. The median age for black women is 20 while the median age for white women is 18. There is a 1-year age difference at the 25th percentile for black and white women, 17 and 16, and a 4-year age difference at the 75th percentile, 24 and 20. There is also a much higher percentage of black women who have never performed oral sex: 30.53% compared to 12.48%.

There is a substantial difference in behavior by race regarding condom use. More black women than white women reported using condoms more than half of the time, with 45.44% and 29.16%. Remember that while this variable reports condom use it does not account for level of STI risk; for example, a monogamous couple may never use condoms, and still practice safer sex by staying monogamous.

The last two risk factors involve alcohol and cigarette consumption. Slightly more black women report smoking cigarettes daily, with 19.75% compared to 17.31% among white women. However, white women reported a greater number of alcoholic drinks per week than black women with means of 4.59 and 3.80, and medians of 1.25 and 0.50, respectively.

Overall, the difference by race for HPV prevalence is much greater for vaginal than oral HPV. Differences in risk behaviors are relatively small with the exception of more white women performing oral sex and reporting more partners. There are roughly 10 point differences in health

insurance and high school graduation by race, and a large difference in income as a ratio of the poverty line.

The large difference in vaginal HPV prevalence appears greater than the differences in vaginal sex behaviors, while the small difference in oral HPV prevalence still shows black women have more HPV even though white women engage in more oral sex risk behaviors. In order to more closely examine the relationship between number of partners, race, access to resources, and HPV prevalence, I use logistic regression models and examine interactions between race and the other variables in the models.

Logistic regression results

Bivariate correlations

Before calculating the results of the multivariate logistic regression models I completed a series of bivariate models as the comparison studies did to find the initial significance. The following results take survey design into account and include the logit coefficient, linearized standard error, and odds ratio. I included three versions of the number of partners variables. First, the original, untransformed variables, then the transformed variables, and then both original and transformed variables in the same model. See Tables 4.4 and 4.5. (Appendix A provides more detailed results for Tables 4.4 and 4.5.)

Table 4.4: Bivariate associations with vaginal HPV

Variable	Odds Ratio	Logit Coefficient	Standard Error
black**	2.83	1.04	0.12
hsgrad*	0.63	-0.46	0.19
anyhi	0.72	-0.34	0.18
fampovrat**	0.78	-0.25	0.04
debut**	0.89	-0.11	0.02
condom**	1.51	0.41	0.11
smoker**	2.06	0.72	0.11
vagnum	1.06	0.06	0.01
vagnumsr**	1.53	0.43	0.08
vagnum	0.97	-0.03	0.01
AND vagnumsr**	2.03	0.71	0.09
nosex	0.18	-1.73	0.36

* $p<0.05$; ** $p<0.01$

Table 4.5: Bivariate associations with oral HPV

Variable	Odds Ratio	Logit Coefficient	Standard Error
black*	1.95	0.67	0.31
hsgrad*	0.35	-1.04	0.42
anyhi	0.65	-0.43	0.36
fampovrat*	0.77	-0.26	0.09
oraldebut	1.01	0.01	0.02
drinksperweek	1.01	0.01	0.01
smoker**	4.83	1.58	0.30
oralnum	1.01	0.01	0.00
oralnumsr*	1.24	0.21	0.09
oralnum AND	0.83	-0.18	0.11
oralnumsr*	4.78	1.56	0.68
nooral	0.64	-0.44	0.64

* $p<0.05$; ** $p<0.01$

According to the bivariate associations with vaginal HPV, race, education, income, age at sexual debut, condom use, smoking, and number of vaginal partners sqrt are statistically significant. According to the bivariate associations with oral HPV, race, high school graduate, income, smoking, and number of oral partners sqrt are statistically significant.

Multivariate logistic regression

For both vaginal HPV and oral HPV I began with a model that just included the SES variables, individual risk behavior variables (excluding number of partners), and age. I then introduced race and number of partners one at a time, and then I included both terms. Lastly, I included a model with an interaction term between race and number of partners. I followed this nested-model procedure to determine the direct and indirect effects of race and number of partners before introducing the interaction term.

Table 4.6 shows the models used to predict vaginal HPV risk. (Appendix B provides more detailed results for Table 4.6.) I first discuss the results for the SES variables, then the individual risk variables, and then the effects of race, number of partners, and their interaction.

Table 4.6: Risk of vaginal HPV for black and white women ages 20-59

Vaginal HPV risk Odds Ratio	**Model 1**	**Model 2**	**Model 3**	**Model 4**	**Model 5**
age	0.97**	0.97**	0.97**	0.97**	0.97**
fampovrat	0.83**	0.86**	0.82**	0.86**	0.85**
anyhi	0.89	0.91	0.89	0.90	0.92
hsgrad	1.45	1.51	1.32	1.36	1.40
condom	1.35*	1.26	1.25	1.16	1.13
debut	0.92**	0.93*	1	1	1.01
smoker	1.60**	1.75**	1.48*	1.63**	1.63**
black		2.29**		2.29**	7.06**
vagnumsr			1.64**	1.64**	1.77**
blackXvagnumsr					0.65**
intercept	9.36**	6.51**	0.90	0.63	0.52
R2	0.12	0.16	0.25	0.28	0.26
N	1,411	1,411	1,410	1,410	1,410

* $p<0.05$; ** $p<0.01$

For the socioeconomic variables, only income was statistically significant. Income remained relatively stable through each model with an odds ratio of approximately 0.84 indicating that controlling for the other variables for every 1 point increase in ratio of household income to poverty line (coded 0-5) the odds of having HPV decrease by a factor of 0.84, or 16%. Due to the variable's relative stability we can see that controlling for race and number of partners does not change the effect.

Health insurance status and education are not significant in any of the models. Health insurance remains relatively stable with an odds ratio of approximately 0.90 throughout the models indicating that controlling for the other variables, having some kind of health insurance reduces the odds of having vaginal HPV by a factor of .9, or 10%, compared to not having health insurance.

Education varies somewhat more than income and health insurance across the models but still has a range of only 1.32 to 1.51. The effect is greatest in Model 2 where I controlled for race but not number of partners, and smallest in Model 3 where I controlled for number of partners but not race. This suggests to me that part of the reason high school graduates are at greater risk for HPV is related to having more partners.

For the individual behavior variables only the practice of daily cigarette smoking remained statistically significant throughout the models. The odds ratios for smoking did not fluctuate drastically, with the highest effect in Model 2, which controls for race but not number of partners, and the smallest effect in Model 3, which controls for number of partners but not race. Again, this suggests that number of partners is partially associated with whether one smokes. Overall the models suggest that smoking cigarettes daily increases the odds of having HPV by a factor of 1.63, or 63%.

Age of sexual debut is statistically significant until I introduce number of sex partners. After introducing number of partners the effect is essentially zero. This makes sense because younger age of debut is associated with having more partners.

Condom use is statistically significant only in Model 1 and becomes insignificant once I introduced race and/or number of partners. The effect of condom use is greatest in Model 1

which does not control for race or number of partners. The odds ratio indicates that paradoxically, people who use condoms at least more than half of the time are at 35% greater risk of HPV. However, frequent condom use could indicate the person has more partners with whom they use condoms, which would increase risk HPV. It would make sense that frequent condom use is associated with more partners because when I controlled for number of sex partners the effect of condom use was smaller. It also reflects an association between race and condom use because the effect is smaller after controlling for race.

The indicator variable for race, black, is significant across the models. Contrary to the comparison studies race remains significant after introducing number of sex partners, and the effect size remains the same. Before introducing the interaction term Models 2 and 4 show that being black compared to white puts one at 129% greater risk for vaginal HPV. Likewise, number of partners remains statistically significant throughout the models, and the size of the effect is not changed by introducing race. Before introducing the interaction term the odds ratio is 1.64, which indicates that every 1-interval increase in number of partners, for example, moving from 1-3 partners to 4-8 partners, increases the odds of having HPV by a factor of 1.64, or 64%.

Once I introduce an interaction term it allows for the possibility that the effect of being black or white will be different with respect to number of partners. This is different than simply controlling for race wherein the effect for number of partners is assumed to be the same for black and white women. In Model 5 the effect of number of partners controls for race, and the effect of race controls for number partners. The interaction term, blackXvagnumsr, is negative, which does not indicate a negative effect, but instead indicates that the effect for number of sex partners is *less* for black women than for white women. The odds ratio of 0.65 means that for every 1

interval increase in number of partners the effect for black women is less steep by a factor of .65, or that the rate of increase is 35% less than the rate of increase for white women.

Once we allow for the possibility that the slope for number of partners may differ by race the effect of race and number of partners increases. The effect of being black becomes much larger and puts one at 7.06 times the risk of HPV than being white. The effect increases because the model accounts for the less steep increase in risk for black women with respect to number of partners. In other words, *even though* risk for white women increases more with each increase in partner intervals, black women are *still* at greater risk, which is why the effect of the variable black increases.

Likewise, the effect of number of partners increases somewhat because when we allow for different effects by race, and also control for race, the much greater effect for white women is reflected in the non-interaction term. *After* acknowledging that the effect of number of partners is different for black and white women a 1-interval increase in number of sex partners results in a 77% increase in HPV risk.

This finding supports my first hypothesis which is that the effect of number of partners would be greater for white women.

Logistic regression using interaction terms

I looked for interactions between race and the other variables in the model. In Table 4.7 I show the results of the other interaction terms. (Appendix C provides more detailed results for Table 4.7.) Each of the interaction terms was included in a model like Model 4, which includes every variable. I evaluate the importance of each interaction term by looking at the coefficient,

which indicates how much the effects vary by race, as well as the significance level, and the change in R2 from Model 4 with R2=0.2774.

Table 4.7: Interactions between race and every other variable, vaginal HPV

Interaction	Odds Ratio	Logit Coefficient	Standard Error	R2
fampovratXblack	1.05	0.05	0.08	0.28
anyhiXblack	1.43	0.36	0.25	0.28
hsgradXblack	0.94	-0.07	0.47	0.28
condomXblack	1.05	0.05	0.29	0.28
debutXblack	1.06	0.06	0.05	0.27
smokerXblack	0.83	-0.19	0.37	0.28
vagnumsr Xblack**	0.65	-0.43	0.12	0.26
nosexXblack*	0.15	-1.89	0.56	0.13

* $p<0.05$; ** $p<0.01$

Only the interactions with number of partners or zero partners were statistically significant and they both indicate a substantial difference in effect. The effect with respect to number of partners is 35% less for black women than the effect for white women. There is an even more drastic difference in effects with respect to zero vaginal partners: The effect for black women is 85% less than the effect for white women.

There is a moderate difference in the effect with respect to smoking: The effect for black women is about 17% less than the effect for white women. There is also a moderate difference for the insurance effect, with the effect for black women 43% greater than the effect for white women. The other variable interactions do not appear large enough to merit further examination.

Logistic regression using divided-sample interactions

There are multiple ways to find interaction effects. I just used one sample of black and white women and then created interaction terms, but we can also divide the sample into the two

racial groups and then compare the coefficients. This approach gives us the effect of every variable for white and black women separately rather than giving us the percent difference between the effects.

This technique has the same issues with logistic regression and interaction terms as the previous technique. For this data in particular it is especially limited because the data from black women comes from a stratum with a single sampling unit which means we cannot produce reliable standard errors or tests of significance. With this in mind I still provide the results because it provides a simple way to get a *rough estimate* of slope differences by race. I believe these results, in addition to the findings from Models 1-5, show us both how interaction effects may vary according to technique, which highlights their imprecise nature, and offers another perspective on the potential differences by race.

In addition to the different perspective, I included another model, Model 6, which uses the variable zero partners rather than number of sex partners. Although zero partners is technically a number of partners I created a separate variable because there is a substantive difference between *not* engaging in a behavior, and engaging to *different degrees*. Model 6 excludes the variables condom use and sexual debut because participants who have never had vaginal intercourse would not have the opportunity for condom use or sexual debut. See Table 4.8. (Appendix D provides more detailed results for Table 4.8.)

Table 4.8: Interactions between race and other variables using divided-samples, vaginal HPV

Vaginal HPV risk Odds Ratio	**Model 4: Black**	**Model 4: White**	**Model 6: Black**	**Model 6: White**
age	0.97	.96	0.98	0.96
fampovrat	0.85	0.83	0.81	0.86
anyhi	1.20	0.84	1.40	0.95
hsgrad	1.29	2.0	1.43	1.65
debut	1.05	1.02		
condom	1.13	1.23		
smoker	1.34	1.41	1.03	1.86
vagnum	0.94	0.97		
vagnumsr	2.01	2.26		
nosex			0.17	0.06
intercept	0.53	0.24	2.75	1.87
R2	0.18	0.27	0.08	0.18
N	478	797	602	962

This way of examining interaction effects has the advantage of more clearly showing when a variable has an effect which actually changes directions by race. For example, according to Model 4 having health insurance decreases HPV risk for white women by 26% but increases risk for black women by 20%. The absolute difference between the slopes is about 43% which matches the finding from the previous method which also predicted black women would be at 43% greater risk for HPV than white women.

We can see another substantial difference in effect for education which shows that a high school education increases HPV risk for black women by 29%, and by 100% for white women.

The interaction term method also indicated that risk for black women would increase less than risk for white women but by a much smaller amount of about 6%.

The interaction term method found a 17% difference in risk for the effect of condom use while the effects in the race-divided Model 4 are 8% different, but in the same direction: the effect of frequent condom use increases risk of vaginal HPV more for white women than for black women.

The difference in the effect for number of partners (sqrt) was about 35% for the first method of identifying interactions and only 11% using the divided-sample method. While the magnitude is different both techniques show that the effect with respect to number of partners is greater for white women than for black women. These findings support my first hypothesis that individual risk behaviors will have a greater effect on HPV risk for white women than black women.

In the divided-sample Model 4, the effect of having zero partners has a 186% difference by race, while the model using interaction terms identified an 85% difference in the same direction. Both models agree that even comparing women with zero vaginal sex partners, black women are at higher risk for HPV than white women.

Interestingly the divided-sample method of examining interactions identified a larger difference by race for education and zero vaginal partners, and identified a smaller difference by race for condom use and number of partners (sqrt). Despite these differences the methods show the effects vary in the same direction. Having insurance, smoking, number of partners, and zero partners showed the largest interaction effect for the interaction terms, although only number of partners and zero partners were statistically significant. Insurance, education, and number of

vaginal partners and zero partners show the largest interaction effects in the divided-sample interaction models, which do not evaluate statistical significance.

The results for my number of partners variables for both interaction methods support my first hypothesis which states that individual risk behaviors will have a greater effect on HPV risk for white women than black women. Although the difference is smaller, the variable for daily smoking also has a greater effect on HPV risk for white women than for black women. The other individual behavior variables, age at sexual debut and condom use appeared to have minimal effects in the multivariate models and for interactions. Ultimately, I feel the vaginal HPV results support my first hypothesis, because for the individual risk behaviors that matter most in predicting HPV risk, the effect is greater for white women than for black women.

The findings for vaginal HPV partially support my second hypothesis, that socioeconomic status variables will have a greater effect on HPV risk for black women than white women. Insurance and education both had different effects by race. Having insurance actually increases risk of vaginal HPV for black women, and decreases risk for white women; but having at least a high school education increases risk of vaginal HPV less for black women than for white women. Since I assumed health insurance would decrease the risk of HPV, the finding for insurance failed to support my second hypothesis: having health insurance decreases risk of HPV more for white women than for black women. But the findings for education do support my second hypothesis: having a high school education increases HPV risk less for black women than white women; in other words, it has a larger protective effect against HPV for black women.

Logistic regression, oral HPV models with interaction terms

Next I discuss the results of my oral HPV models. Replicating my approach for the vaginal HPV models, I first review the effects of the SES variables, then the effects of the individual risk variables, and finally the effects of race, number of oral partners, and their interaction. See Table 4.9. (Appendix E provides more detailed results for Table 4.9.)

Table 4.9: Risk of oral HPV for black and white women ages 20-59

Oral HPV risk Odds Ratio	**Model 7**	**Model 8**	**Model 9**	**Model 10**	**Model 11**
age	1.01	1.01	1.0	1.0	1.0
fampovrat	0.84	0.87	0.80	0.83	0.83
anyhi	0.97	1	0.97	0.99	1
hsgrad	0.85	0.87	0.76	0.79	0.78
drinksperweek	1	1	0.99	0.99	0.99
oraldebut	1.03	1.02	1.09*	1.08*	1.08*
smoker	3.58**	3.72**	3.04**	3.20**	3.16**
black		1.76		1.86	22.94
oralnum			0.78	0.78	0.69
oralnumsr			8.0*	8.14*	17.62*
blackXoralnum					1.36
blackXoralnumsr					0.14
intercept	0.02**	0.02**	0**	0**	0**
R2	0.13	0.14	0.65	0.66	0.82
N	1,357	1,357	1,357	1,357	1,357

* $p<0.05$; ** $p<0.01$

None of the SES variables are significant in any of the models, and the odds ratios remain relatively constant across the models. For income, the models indicate that controlling for the other variables, for every 1 unit increase in income the risk of oral HPV decreases by a factor of 0.83. Having health insurance has virtually no effect in any of the models with an odds ratio of approximately 0.99. The effect of having graduated high school remains relatively constant with a range from 0.76 when controlling for number of partners but not race and 0.87 when controlling for race but not number of partners, which suggests that like the models for vaginal HPV, graduating high school must be associated in part with higher number of oral partners.

For the individual risk behaviors alcohol consumption is not statistically significant, and has virtually no effect in any of the models with an odds ratio of approximately 0.99. Age at first performance of oral sex is insignificant and has a very small effect in Models 7 and 8, but becomes significant and has a larger effect once I control for number of partners. In Models 9, 10, and 11, the effect of age of debut is approximately 1.08, which indicates that for every 1 year later you begin performing oral sex the odds of having oral HPV increase by a factor of 1.08, or 8%. This is counter-intuitive to what we might expect, which would be that the later in life one starts performing oral sex, the fewer years at risk one would be at, and perhaps the more conservative number of partners one would have. I expect age at oral sex debut must be associated with another variable that increases HPV risk.

Smoking has a considerable effect on HPV risk and is significant in each model. The odds ratio is lowest in Model 9, at 3.04, which controls for number of partners but not race. The effect of smoking is largest in Model 8, at 3.72, which controls for race but not number of partners. This suggests that smoking is associated with having more oral partners as the vaginal HPV models also indicated.

The effect of race is not statistically significant in any of the models. Because race was significant in the bivariate models, but not the multivariate models which controlled for number of partners, previous researchers concluded the effect of race could be attributed to other variables. However, it is important to look at how the effect of race changes even if it is not statistically significant. In Model 8, which controls for race but not number of partners, the odds ratio is 1.76, so being black instead of white puts one at 76% greater risk for HPV. In Model 10, which includes both race and number of partners, the effect of race actually increases to an odds ratio of 1.86 indicating that even after controlling for number of partners race has an effect and it is even larger than when disregarding number of partners.

The effect of number of partners is significant in every model. There is a moderate increase in the odds ratio after controlling for race; without race, for every 1 interval increase in number of sex partners risk for HPV increases by a factor of 8, or by 700%. Once we control for number of sex partners the effect of a 1 interval increase in number of partners increases by a factor of 8.14, or 714%.

The interaction term between race and number of partners is statistically insignificant in Model 11 but it drastically changes the effects of race and number of partners and it increases the pseudo-R2 by 20%. The effect of being black goes from 1.86 to 22.94, which means when we allow for the possibility of different effects with respect to number of partners for black and white women, being black puts one at higher odds of having HPV by a factor of 22.94.

Likewise, the effect of number of partners increases from 8.14 to 17.62. Allowing for the possibility of different effects by race the effect for number of partners *controlling for* race indicates that for every 1 interval increase in number of partners the odds of having oral HPV

increase by a factor of 17.62. This reflects the increased impact of the number of partners variable once we allow for different effects by race.

The interaction term itself is 0.14, meaning that the effect with respect to number of partners for black women is less than the effect for white women by a factor of 0.14, or the effect is 86% less for black women than for white women. The difference in effects with respect to number of partners by race is greater for oral HPV than for vaginal HPV. This makes sense because black women have fewer oral partners than white women but are still at higher risk.

As with vaginal HPV I tried introducing an interaction term between race and every other variable in the model one at a time. I began with a model like Model 10 which includes every variable, and then found the effect of adding an interaction term. See Table 4.10. (Appendix G provides more detailed results for Table 4.10.)

Table 4.10: Interactions between race and every other variable, oral HPV

Interaction	Odds Ratio	Logit Coefficient	Standard Error	R2
fampovratXblack	1.17	0.16	0.21	0.66
anyhiXblack	0.70	-0.36	0.97	0.66
hsgradXblack	2.38	0.87	0.93	0.66
drinksperweekXblack	0.97	-0.03	0.04	0.66
oraldebutXblack*	0.89	-0.11	0.04	0.66
smokerXblack	0.55	-0.60	0.80	0.66
oralnumXblack	1.00	0.00	0.08	0.66
oralnumsrXblack	0.81	-0.21	0.50	0.67
oralnumXblack AND oralnumsrXblack	1.36 0.14	0.31 -1.96	0.23 1.40	 0.82
nooralXblack	1.17	0.16	0.78	0.15

* $p<0.05$; ** $p<0.01$

The only interaction which is statistically significant is between race and age at oral debut. With an odds ratio of 0.89 it indicates that the effect for oral debut is 11% less for black women than white women. The SES variables have considerable differences by race: The effect

with respect to income is 17% greater for black women than white women, and the effect of graduating high school is 138% greater for black women than white women. The larger effect indicates that income and education do not mitigate HPV risk for black women as well as for white women. The effect of having health insurance is 30% less for black women than for white women, which means black women lower their risk of HPV more by having health insurance.

The effect of smoking is 45% less predictive of HPV for black women than white women.

When using just one interaction term for number of partners (sqrt) and race the effect is less different by race than using both forms of the number of partners variable. With just #partners(sqrt)Xblack the slope of number of partners is 20% less steep for black women than for white women, but when both forms of the variable are used the slope is 86% less steep for black women. I chose to include both forms of number of partners in Model 11 because that version increased the R2 by 18%.

The effect of having zero oral partners is greater for black women than white women by 17%, indicating that even when not sexually active, black women are at higher risk of HPV than white women.

As with the data on vaginal HPV I also looked for interactions with race by separating the sample into two samples, one with just black women and one with just white women. This shows me two different slopes rather than having a term which just shows the difference between the slopes. I also introduced the variable zero oral partners in Model 12 to determine the different risk levels with no oral partners. Again, this data does not have standard errors or significance

levels and should be interpreted with caution. See Table 4.11. (Appendix G provides more detailed results for Table 4.11.)

Table 4.11: Interactions between race and other variables using divided-samples, oral HPV

Oral HPV risk Odds Ratio	**Model 10: Black**	**Model 10: White**	**Model 12: Black**	**Model 12: White**
age	1.04	0.99	1.02	1.01
fampovrat	0.94	0.81	0.89	0.88
anyhi	0.57	1.21	0.66	1.26
hsgrad	1.02	0.59	1.08	0.63
drinksperweek	0.99	0.99	0.98	1
oraldebut	0.91	1.16		
smoker	1.92	3.69	2.54	4.61
oralnum	0.99	0.62		
oralnumsr	1.22	35.62		
nooral			0.50	0.46
intercept	0.09	0	0.03	0.02
R2	0.16	0.88	0.10	0.16
N	457	900	609	991

Both methods of examining interactions show a 16% difference in effects with respect to income. Greater levels of income mitigate the risk of HPV more for white women than for black women. Income decreases risk of HPV for black women by 6%, while it decreases risk for white women by 19%.

The divided-sample method shows a substantial difference by race for the effects with respect to insurance status. We can see that having insurance decreases risk of HPV for black

women by 43%, while it increases risk of HPV for white women by 21%. This relationship was the opposite for vaginal HPV which indicated having insurance increases risk of vaginal HPV for black women but decreased risk for white women.

Both interaction methods suggest that the effect of a high school education mitigates HPV risk more for white women than for black women. For the previous method the difference between effects was an effect for black women 137% greater than the effect for white women. The divided-sample method shows effects that are 73% different and also shows that the effect of education mitigates risk of oral HPV for white women, while not changing, or slightly increasing, risk for black women.

Both interaction methods identify a difference in the effect for age at oral debut. The divided-sample method shows that for every one year older someone is when they begin having oral sex the risk of HPV decreases for black women by 9%, while it increases for white women by 16%. The difference between effects is about 25% while the difference identified by the interaction term was about 11%. The differences by race change in the same direction for both techniques.

Both ways of identifying interactions show a substantial difference in HPV risk for smoking. The divided-sample method shows that smoking daily increases HPV risk for black women by 92%, while it increases risk for white women by 269%. The percent difference between the slopes is about 48%, and the percent difference when regarding the interaction term is 45% is the same direction.

Both techniques also identify a large difference in effects with respect to number of partners. The divided-sample method indicates that for every 1 interval increase in number of

partners risk of HPV increases by 22% for black women, and by 346% for white women. The percent difference between the slopes is about 97%. The interaction term for this same interaction also indicates the risk of HPV for white women increases more than for black women, by about 86%.

Lastly, the effect of having zero oral partners mitigates the risk of HPV more for white women than for black women. The divided-sample method shows an 8% difference in effects: risk for black women decreases by 50%, while risk for white women decreases by 54%. The interaction term indicates a 17% difference in effects and again shows that HPV decreases more for white women than for black women.

Overall both techniques predicted the slopes changing in the same direction but had differences in magnitude of difference by race. They predicted the same magnitudes for income and smoking, but different ones for insurance, education, oral debut, and number of partners or no partners. The direction of difference remained the same.

Using both methods for evaluating interactions has the advantage of comparing two imprecise methods and seeing where the differences occur. These results make me confident that the direction of difference is trustworthy, while the magnitude of difference is not.

For the interaction term method of identifying interactions, income, insurance, education, oral debut, smoking, and number of partners had different effects by race, although only age at oral debut was statistically significant. For the divided-sample method of identifying interactions, income, insurance, education, oral debut, smoker, and number of partners had different effects by race. This method did not identify statistical significance.

The results predicting oral HPV partially supported my first hypothesis, that individual risk behaviors will have a greater effect on HPV risk for white women than black women. My first hypothesis is supported because the effect of number of partners and smoking was greater for white women than for black women. The other individual behavior variable that had an effect, oral debut, indicated that for every year later one begins performing oral sex, the risk of oral HPV goes *down* for black women, but goes *up* for white women. Because I assume later age would result in lower risk, I conclude the effect of oral debut is greater for black women than white women, which contradicts my first hypothesis.

The results predicting oral HPV risk partially support my second hypothesis, that socioeconomic status variables will have a greater effect on HPV risk for black women than white women. The effect of having insurance supported my second hypothesis: black women with health insurance lowered oral HPV risk more than white women with health insurance, who were actually at greater oral HPV risk. However, the effects of education and income reduced risk of oral HPV more for white women than for black women, which contradicts my second hypothesis.

Hypotheses

In this final section I evaluate how well the results support my hypotheses. My hypotheses were:

Hypothesis 1: Individual risk behaviors will have a greater effect on HPV risk for white women than black women.

Hypothesis 2: Socioeconomic status variables will have a greater effect on HPV risk for black women than white women.

The findings support my first hypothesis for both vaginal and oral HPV because the behaviors of smoking and greater numbers of sex partners increase risk of HPV more for white women than for black women. Both methods of finding interactions revealed that smoking would increase risk of vaginal HPV for white women more than black women. The difference in effects by race is even greater for oral HPV. The same trend is reflected in the different effects for number of partners. For every 1 interval increase in number of vaginal partners risk of vaginal HPV increases more for white women, with the interaction term showing a 35% difference in effects, and the divided-sample method showing an 11% difference in effects. This is also reflected in the findings for oral HPV where risk of HPV increases more for white women than for black women. The interaction term shows an 86% difference by race and the divided-sample term shows a 97% difference by race.

The findings partially support my second hypothesis that socioeconomic factors would have a greater influence for black women than for white women. The effect of income mitigates the risk of oral and vaginal HPV by about the same amount with no drastic differences in the effects by race. The effect of having insurance decreases the risk of vaginal HPV by 16% for white women, while it actually increases risk for black women. The opposite is true for oral HPV, where having insurance decreases risk for black women by 43%, while it increases risk for white women by 21%. The effect of education is also different by race. Graduating high school increases vaginal HPV risk more for white women than for black women, and it actually decreases oral HPV risk for white women. My second hypothesis is supported by the findings for the effect of insurance on vaginal HPV and the effect of education on oral HPV. The effect of insurance on oral HPV and the effect of education on vaginal HPV do not support my second

hypothesis. Likewise, the lack of difference in effect of income does not support my second hypothesis.

In the following chapter I discuss the theoretical implications of these findings.

CHAPTER 5: DISCUSSION

In this chapter I discuss the theoretical implications of my research findings. I began this research because I was skeptical about HPV research which cited number of sexual partners as the most predictive variable of increased HPV risk. My skepticism stemmed from knowledge that HPV is differentially distributed throughout the population by gender, race, and class. This pattern of certain social locations having greater HPV prevalence than others suggested to me that differential HPV risk warrants investigation using the sociological imagination (Mills 2000). This includes viewing increased HPV risk as a social issue related to social and historical location, rather than a personal trouble, or a choice or behavior over which an individual has control. Instead of individual/behavioral explanations for HPV risk, I wanted to see how well data on HPV supported a structural/materialist explanation.

I begin my theoretical discussion by comparing my work to the four comparison studies which inspired my research (Chaturvedi et al. 2015; D'Souza et al. 2014; Dunne et al. 2007; Markowitz et al. 2009). I outline the differences which led to finding different results. I then discuss the theoretical implications of my findings by explaining how my results support a modified version of Link and Phelan's theory of social conditions as a fundamental cause of health inequalities.

Summary of comparison studies

The studies from Chaturvedi et al. (2015), D'Souza et al. (2014), Dunne et al. (2007), and Markowitz et al. (2009) had different objectives but all found that race/ethnicity was significant in bivariate models, and insignificant in multivariate models which controlled for number of sex

partners. My research is similar in that I also used bivariate and multivariate logistic regression models to determine which variables best predict HPV.

In Table 5.1 I summarize the data source, population, objective, conclusion, weighting and variance adjustments, test statistics, criteria for multivariate model inclusion, amd interaction evaluation for each of the comparison studies.

Table 5.1: Summary of comparison studies

Study	**Chaturvedi et al. 2015**	**D'Souza et al. 2014**	**Markowitz et al. 2009**	**Dunne et al. 2007**
Years, population	NHANES 2009-2012, men and women ages 14-69	NHANES 2009-2010, men and women ages 20-69	NHANES 2003-2004, men and women ages 14-59	NHANES 2003-2004, women ages 18-59
Objective	Investigate reasons for higher oral oncogenic HPV infection among men than women by associated risk factors	Explore whether gender, age, and race differences in oral sexual behavior account for demographic distribution of oral HPV and HPV-positive oropharyngeal cancer.	Estimate prevalence of quadrivalent vaccine HPV types in United States.	Estimate prevalence of HPV among females in the United States.
Conclusion	Men have greater rates of oral oncogenic HPV infection than women, and a stronger association between sexual behavior and infection	Differences in sexual behavior by age, gender, and race explain difference in oral HPV strain 16	Quadrivalent vaccine HPV types are more prevalent among women, blacks, and older individuals than among men, white, and younger individuals. Age, lifetime number of sexual partners, and poverty are independently associated with	Age younger than 25 years, marital status, and increasing number of recent or lifetime sex partners is independently associated with HPV detection.

			HPV among women.	
Weighting and variance adjustments	NHANES weights for complex survey design, variance estimators	NHANES weights for complex survey design, variance estimators	NHANES weights for complex survey design, variance estimators	NHANES weights for complex survey design, variance estimators
Test statistics	Wald F test (P<.05)	Wald F test (P<.05)	Wald F test, Satterthwaite - adjusted F-test (P<.05)	Wald chi2 and Satterthwaite-adjusted F-test (P<.05)
Multivariate model inclusion criteria	A priori and significant bivariate results	A priori and significant bivariate results	Backwards elimination from significant bivariate associations (P<.1)	Backwards elimination if P>.05
Interactions	Tests between multivariate model variables and gender	No interactions evaluated	All pairwise interactions in main effects models	All pairwise interactions in main effects models

The comparison studies and my own work share the following characteristics: First, we all used NHANES data and focused on similar age groups. Second, each study used complex study design weights and variance estimates provided in the NHANES data. Third, each study used a combination of bivariate and multivariate models to assess HPV prevalence, and independent association with HPV status. Fourth, the studies used the same or similar test statistics to evaluate models and associations (my research used the Satterthwaite F-test).

However, the comparison studies and my own work also feature significant differences, such as the NHANES cycle years, criteria for variable inclusion, interaction evaluation, and most significantly, research objective and conclusion. The different initial research questions impacted many subsequent research decisions. Most notably, D'Souza et al (2014) was the only study to foreground race in the study objective. Chaturvedi et al (2015) and Markowitz et al (2009) focused on gender difference in both HPV prevalence and risk behaviors, while Dunne et al

(2007), like in my research, engaged with gender by focusing only on women. The emphasis on gender led Chaturvedi et al. (2015) to only evaluate possible interactions between variables included in the multivariate models and gender, ignoring the possibility of interactions by race.

Ultimately, the most meaningful difference between my research and the studies that inspired it is difference in research question. I chose my research question after reading the comparison studies and then searching for more literature on race and sexual health because my sociological training sensitized me to the importance of socially constructed identities and their relation to power.

For example, a crucial finding from my results is that sexual risk behaviors have a different effect on HPV risk for white and black women. Because I focused on racial difference from the outset of the study, I evaluated interactions between each variable (which I chose a priori), and white or black race. The four comparison studies did not evaluate race as an interaction effect either due to research focus (Chaturvedi et al. 2015), or choosing to only evaluate variables independently associated HPV risk (Dunne et al. 2007; Markowitz et al. 2009), or foregoing interactions altogether (D'Souza et al. 2014).

Since each comparison study and my own departs from a different research question it is difficult to make direct comparison between results. However, I point out that the descriptive results from my data set are at least similar to the results from an official NCHS data brief from McQuillan et al. (2017). The CDC researchers analyzed the 2011-14 NHANES cycles, including participants 18-69 years old, and found that 63.2% of black women and 36.5% of white women had vaginal HPV, while 4.5% of black women and 2.9% of white women had oral HPV. My results found that 60.37% of black women and 35.54% of white women tested positive for

vaginal HPV, while 9.65% of black women and 7.32% of white women tested positive for oral HPV. While I could not confirm my extended analyses with any currently published research, I can see my estimates for vaginal HPV status are reasonably close to CDC estimates.

My choice to focus on race and power distinguishes my research from the comparison studies and has direct implications not only for my statistical findings, but also for their theoretical implications. In the following sections I discuss how my results relate to Link and Phelan's structuralist explanation of health disparities and how I adapt their theory to highlight racial differences in sexual health.

Theoretical implications

Link and Phelan (1995) developed the structuralist theory of social conditions as a fundamental cause of health inequalities. This approach emphasizes how differential access to resources leads to different health outcomes and argues that individual behaviors are *intervening* mechanisms to health, whereas resources are the *fundamental* cause. Their theory recognizes power in terms of access to resources, broadly defined as money, knowledge, power, prestige, social support, and social networks. My research identified income as a significant predictor of vaginal HPV risk, but also found that education and insurance status are not significant predictors of vaginal or oral HPV. However, the results do suggest that education and insurance have different effects by race.

I believe Link and Phelan's theory is best suited for health outcomes strongly related to class. Their theory is a good starting point for discussing disparities because it emphasizes power in terms of resources and is not limited to specific mechanisms of increased risk, or specific health outcomes. However, I argue that Link and Phelan's theory could be adapted to better

explain how the social category of *race* relates to different *sexual health* outcomes. Link and Phelan's theory captures many material aspects of the inequalities inherent to white supremacy but fails to engage with ideological power and specificity of social location. For this reason I draw upon additional theorists to explain the relationship between race and sexual health: I use Foucault's argument that knowledge is intimately connected to power through discourse, and Hammond's advice to examine relational constructions of sexuality, and to highlight aspects of power which would otherwise disappear into schemas of common sense.

Building upon Link and Phelan, Foucault, and Hammonds, I propose that my findings regarding vaginal and oral HPV support a theory of race as a fundamental cause of inequalities in sexual health. More specifically, I argue that the historical construction of black and white women's sexuality, as well as the material and political inequalities inherent in white supremacy, have led to greater risk of STIs among black women than white women. In the following sections I explain how my research provides evidence for a theory of race as a fundamental cause using Link and Phelan's four criteria for establishing fundamental cause. I then extend this argument by pointing out how the knowledge created about black and white women's sexualities reflects and justifies ideologies of patriarchal white supremacy.

Adapting Link and Phelan's theory to race and sexual health

First I discuss my argument for adapting Link and Phelan's social conditions as fundamental cause theory to explain the relationship between race and inequalities in sexual health outcomes. To begin, I offer a brief description of the criteria they use to establish social conditions as the fundamental cause of health inequalities: 1) the social condition influences multiple disease outcomes, 2) it affects disease outcomes through multiple risk factors, 3) it

involves access to resources to minimize or cope with risk, and 4) it is reproduced over time via replacement of intervening mechanisms (Phelan, Link and Tehranifar 2010).

As we can see, Link and Phelan's theory encompasses multiple health outcomes and risk factors throughout time, and one study alone cannot prove or disprove the theory. The evidence I provide relates only to HPV and uses data collected at only one time-period: 2011-2014. Also, my research only examined data on black and white women and we should not necessarily generalize these findings to all races and genders. However, my findings, when viewed in the context of other research on STI inequalities which I discuss below, support a theory of race as a fundamental cause of inequalities in sexual health.

My findings illustrate the connection between race (the social condition in this theory), and risk of HPV (the health outcome). Even after controlling for individual behaviors and socioeconomic indicators, black women are at greater risk for oral and vaginal HPV than white women. Other research has explored different STI and HIV outcomes by race, specifically focusing on black Americans, and found that race also affects risk of chlamydia, gonorrhea, and HIV, regardless of individual risk behaviors and class differences (Hallfors et al. 2007; Halpern et al. 2004; Harawa et al. 2003; Santelli et al. 2000, Ellen, Aral and Madger 1998). My findings add to a body of literature which provides strong evidence for the first criterion of establishing a social condition as a fundamental cause of health inequalities.

The second criterion states that *multiple* risk factors are associated with the social condition and health outcomes. My research findings revealed multiple risk factors affect HPV risk, some varying by race and some not. For example, income has a significant effect on vaginal HPV risk, but does not vary substantially by race. On the other hand, education and having

health insurance had different effects by race on vaginal and oral HPV. Likewise, the individual risk factors like smoking and number of sex partners were significant predictors of vaginal HPV risk, and their effects varied by race. Based on these findings, and consistent with Link and Phelan's theory, multiple risk factors clearly influence the probability of having HPV, and race is a variable which both has its own effect and interacts with the effect of other variables.

The third criterion is that access to resources helps minimize or cope with risk. This is true for HPV in a number of ways: First, access to the HPV vaccine can prevent one from contracting HPV in the first place. There is evidence that more black, Latinx, and low-income women start the vaccine treatments, but overall more white affluent women finish the whole course, suggesting that this resource is differentially distributed by income as well as race (Jeudin et al. 2014; Niccolai, Mehta and Hadler 2011; Pierre Joseph et al. 2014). Second, access to regular health care such as pap smears can prevent high-risk HPV strains from ever developing into Invasive Cervical Cancer. Regular access to healthcare is another resource which is distributed differentially by race (Barnholtz-Sloan et al. 2009; Jeudin et al. 2014; Niccolai, Mehta and Hadler 2011). Each of these resources help people minimize or cope with risk and are distributed differently by race.

The fourth criterion is that the health inequality is reproduced over time via replacement of intervening mechanisms. As I mentioned earlier, the scope of my research project does not include tracking HPV risk throughout history. However, in the following section I review literature on medical research and media literature which illustrates that sexual health inequality is not a new phenomenon. Overall my research provides data to support Link and Phelan's second and third criteria, and I cite additional literature to provide additional evidence that race is a fundamental cause of unequal STI outcomes.

How power constructs racialized sexualities

My work focused on black and white women and found that being black had an effect on HPV risk that could not account for behavior or class alone. This is not to suggest that there is an essential or inherent difference between black and white women. Consistent with a broader social science perspective on race I conceptualize race as a social construct which has very real material effects. In other words, I do not argue that simply being raced as black leads to sexual infection and disease. I argue that being raced black and gendered female in a country that most values whiteness and maleness is associated with some variable or variables which threaten sexual health.

In this section I point out how gendered and racialized sexual narratives and knowledge have supported white racial dominance. Drawing from Foucault I assert that knowledge, whether in the form of colloquialisms or a scientific journal, both reflects and reinforces power relations. It is for this reason that we should examine scientific findings, especially those which appear to support the status quo, within their social context. I offer a brief outline of the origin and continued prevalence of knowledge about black and white women's sexuality at three times in American history: 19th century slavery, early 20th century research on venereal disease, and 1980s AIDS crisis, citing Barbara Omolade and Evelynn Hammonds (Guy-Sheftall 1995). I then explain how this context is essential for interpreting my research.

Black and upper-class white women's sexuality has been juxtaposed in America since colonial-era slavery. White slave owners had multiple incentives to have sex with enslaved black women: First, any offspring would automatically inherit their mothers' slave status and add to the workforce, and second, the white men's wives needed respite between pregnancies and were

not always sexually available to their husbands. Hegemonic discourse portrayed middle and upper-class white women as "pure women incapable of erotic feeling," while black women were seen as uncivilized and libidinous (Guy-Sheftall 1995:368).

Although women of both races were subservient to white men, white women usually aligned their interests with their husbands. "White women used the social relationship of supervisor of black women's domestic labor to act out their racial superiority, their emotional frustrations, and their sexual jealousies" in the form of physical or mental abuse (Omolade, cited in Guy-Sheftall 1995:368). This strange blend of systematized oppression and forced domestic and sexual intimacy supported the American economy and allowed it to flourish.

The discourse of black and white women's sexualities, which emerged from a time of institutionalized rape and forced labor, is also reflected in medical research on sexually transmitted diseases from the 19th and 20th centuries. Respected researchers aptly produced evidence of sexual and racial difference. For example, Thomas Murrell wrote an article for the American Medical Association, "Syphilis and the American Negro: A medico-sociologic study" stating that "95% of the negro race are likely to contract syphilis or other venereal disease including those of the educated classes" (1910:846). Like with the later Tuskeegee Syphilis Experiment, medical science provided a strong connection between blackness and sexual disease.

Of course, middle-class white women also contracted syphilis, and when they did, physicians avoided attributing their illness to personal failings. Respected physicians at John Hopkins Hospital in 1920 recommended different treatments for 'innocent' white women of the 'intelligent classes,' and black women, whom they described as 'ignorant,' 'unmoral,' and

'unmanageable'" (Williams 1920:141-145). Hammonds describes the treatment options black and white women were faced with:

> Programs for middle-class white women were designed to reduce the stigma associated with having syphilis and seeking treatment. African American women's options for treatment were limited by economic resources and the necessity of exposing themselves to censure by white professionals on an issue inextricably related to sex, in a context where their privacy and dignity could not and never had been preserved (Guy-Sheftall 1995:446).

Research on syphilis and gonorrhea proliferated in the early 20th century and produced knowledge which aligned with slavery era narratives. We can see these narratives again in popular media during the AIDS crisis in the 1980s.

Hammonds describes a particular article published in the *New York Times* in 1987 which announced that of the 50,000 women infected with HIV in New York City, 80% were black or Hispanic. The author Jane Gross argues that educating these "poor" and "reckless" women who bring "disease from the world of drug abuse to the larger community" is "an increasingly urgent task" (Guy-Sheftall 1995:436). Gross recommends more education for racially marginalized women so they can learn the proper health behaviors to avoid endangering the larger community. This is an apt example of a behavioral, or healthist approach, which puts reasonability and a moral imperative on the individual to remain healthy and prevent illness. Gross has no advice for the women who already have HIV.

I bring up these periods of American history to provide context for black and white power relations, and the type of knowledge which they produce. Doctors, journalists, and popular discourse painted Black women in general, but particularly those with STIs, as immoral and irresponsible, with no regard for their own or their family's safety, even in contexts where they were clearly victimized. The high rates of STIs among black women are continually framed

as personal, moral matters, devoid of any historical or structural context. White women from the middle and upper classes on the other hand have been portrayed as innocent victims of circumstance. These two very different explanations for why black and white women have STIs shows how the type of knowledge produced reflected power relations, as Foucault illustrates with his theory of knowledge/power. This supposedly objective knowledge provides the discourse and scientific basis to continue pathologizing and marginalizing black female sexuality. And since white, middle and upper-class Americans, whether as plantation owners, physicians, or journalists produce truth, they can use it to suit the power relations they most benefit from.

In the next section I summarize my argument that race is a fundamental cause of sexual health inequalities.

The argument for race as a fundamental cause of sexual health inequalities

I argue that race is a fundamental cause of disparities in sexual health using the results of my research, and literature on health intervention strategies, knowledge production, and historical and current power relations between black and white women. Consistent with Link and Phelan's theory of social conditions as a cause of health inequalities, my research illustrates that individual behaviors such as number of sex partners or smoking have a different effect on HPV risk depending on one's social condition, as black women or white women. It also illustrates that access to certain resources, such as income, affects HPV risk.

However, my research, even when interpreted through Link and Phelan's theory of social conditions as a fundamental cause of health inequalities, fails to illustrate how narratives of racialized sexuality have persisted throughout history and supported the prevailing power

relations. Therefore, I propose adjusting Link and Phelan's theory to more specifically address race and sexuality.

Link and Phelan's theory alone best describes health differences by class. A theory specific to race and sexuality must recognize that American society has treated black and white women's sexuality as inherently different since the country began. The decades of different treatment, different narratives, and of course the different resources have put women at different levels of sexual health risk. Black women particularly are at greater health risk because their social position has been marginalized on multiple levels: race, gender, and often class. It is no coincidence that black women are at higher risk of STIs; it is a result of patriarchal white supremacy.

It is essential to situate any study of American sexuality in the context of centuries of racial and gender oppression in order to interpret data and produce effective health intervention strategies. The studies which inspired my research (Chaturvedi et al. 2015; D'Souza et al. 2014; Dunne et al. 2007; Markowitz et al. 2009) failed to engage with race or power relations beyond what class and demographic variables account for. An awareness of material power relations, such as Link and Phelan provide, as well as narrative power, which Foucault and Hammonds engage with, provides a more nuanced and contextually appropriate lens through which researchers should interpret data on race and sexual health.

In the final section of my thesis I summarize my conclusions and explore directions for future research.

CHAPTER 6: CONCLUSION

In this section I discuss the conclusions I have drawn about my thesis results and theoretical approach to studying race and sexuality. I then discuss policy implications, and directions for further research.

My findings revealed that while number of sex partners is a significant predictor of vaginal and oral HPV, it fails to explain differences in HPV between black and white women. Number of sex partners, as well as education and health insurance status have different effects on HPV risk by race, and even comparing women of similar behaviors and class, black women are at greater HPV risk than white women.

These findings guide me to advice and questions sexual health researchers should consider: 1) Conceptualize race as more than just a demographic covariate. Even if it is not independently associated with a health outcome it may interact with other predictive variables. 2) Consider if your interpretation of the findings support prevailing power relations. Does the narrative of your research reflect or challenge stereotypes? How would introducing more social and historical context change how you interpret your findings? 3) Consider the context in which your data was collected, and how it fits in to historical research on your topic. Consider how the statistical procedures you use may lose or ignore social context.

Policy implications

Intervention strategies which focus on individual behavior, such as number of sex partners, will be less effective for certain groups. This would lead to health disparities growing larger, rather than narrowing. Interventions should take not only access to resources into account,

but also the cultural meaning attached to various practices or resources, and the knowledge directly or implicitly created by the proposed intervention. Additionally, researchers should consider whether the intervention strategy addresses the *fundamental* cause, or a *proximate* cause of the health outcome. If asking people to make different choices, consider whether those choices are equally open, and will have the same effect for everyone.

Directions for further research

My research findings prompt many more questions. First, my research focused on data and knowledge about black and white women. But black and white constructions are not the only relational constructions. Further research is needed to examine relational constructions of sexuality between other racial and ethnic groups. Similarly, my research did not highlight differences among black women and among white women. Singular racial groups are not monolithic and research is needed to examine the power relations and relational constructions of sexuality *within* racial groups.

Second, my research shows that black women are at greater risk of HPV regardless of individual or structural variables. Further research is needed to find which variables associated with race cause this disparity. We need research on the health effects of being black in a society organized by white supremacy which attends to more than just material inequalities between races. While difficult to measure, research on the psychic toll of being a racial or ethnic minority in America could reveal more direct connections between racial systems of oppression and unequal health outcomes.

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APPENDIX A: ADDITIONAL RESULTS FOR TABLES 4.4 AND 4.5

Table 4.4 extended: Bivariate associations with vaginal HPV

Variable	Logit Coefficient	Standard Error	T	P	Odds Ratio
black**	1.038901	0.117643	8.83	0	2.826109
somecollege**	-0.30025	0.109496	-2.74	0.01	0.74063
hsgrad*	-0.45624	0.193914	-2.35	0.025	0.633663
collegegrad**	-0.59835	0.126797	-4.72	0	0.549719
anyhi	-0.33503	0.181467	-1.85	0.074	0.715315
fampovrat**	-0.25239	0.038584	-6.54	0	0.776938
debut**	-0.11454	0.023622	-4.85	0	0.891777
condom**	0.413409	0.106777	3.87	0.001	1.511964
smoker**	0.720411	0.109722	6.57	0	2.055277
vagnum**	0.058404	0.013068	4.47	0	1.060144
vagnumsr**	0.426421	0.077471	5.5	0	1.531766
vagnum AND vagnumsr**	-0.03247 0.709104	0.00733 0.093723	-4.43 7.57	0 0	0.968054 2.032169
nosex	-1.73079	.3569491	-4.85	0	0.177144

* p<0.05; ** p<0.01

Table 4.5 extended: Bivariate associations with oral HPV

Variable	Logit Coefficient	Standard Error	T	P	Odds Ratio
black*	0.666866	0.305255	2.18	0.036	1.948121
somecollege*	-0.69360	0.235956	-2.94	0.006	0.499776
collegegrad	-1.01048	0.545827	-1.85	0.073	0.364043
hsgrad*	-1.04003	0.422673	-2.46	0.019	0.353441
anyhi	-0.43113	0.363181	-1.19	0.244	0.649775
fampovrat*	-0.26382	0.093208	-2.83	0.008	0.768111
oraldebut	0.01245	0.022962	0.54	0.591	1.012528
drinksperweek	0.008472	0.013869	0.61	0.546	1.008508
smoker**	1.575079	0.301622	5.22	0.000	4.831125
oralnum	0.005441	0.003163	1.72	0.095	1.005456
oralnumsr*	0.211131	0.089838	2.35	0.025	1.235074
oralnum AND oralnumsr*	-0.18333 1.564915	0.110369 0.684747	-1.66 2.29	0.106 0.029	0.832491 4.782266
nooral	-0.44159	0.635554	-0.69	0.492	0.643011

* p<0.05; ** p<0.01

APPENDIX B: ADDITIONAL RESULTS FOR TABLE 4.6

Table 4.6 extended: Risk of vaginal HPV for black and white women ages 20-59

Vaginal HPV risk Logit Coefficient (standard error)	**Model 1**	**Model 2**	**Model 3**	**Model 4**	**Model 5**
age	-0.028 (0.006)** OR=0.97	-0.029 (0.006)** OR=0.97	-0.033 (0.007)** OR=0.97	-0.033 (0.007)** OR=0.97	-0.034 (0.007)** OR=0.97
fampovrat	-0.190 (0.043)** OR=0.83	-0.147 (0.043)** OR=0.86	-0.197 (0.048)** OR=0.82	-0.154 (0.049)** OR=0.86	-0.158 (0.051)** OR=0.85
anyhi	-0.119 (0.194) OR=0.89	-0.099 (0.192) OR=0.91	-0.116 (0.205) OR=0.89	-0.101 (0.204) OR=0.90	-0.086 (0.207) OR=0.92
hsgrad	0.375 (0.302) OR=1.45	0.414 (0.292) OR=1.51	0.275 (0.318) OR=1.32	0.311 (0.300) OR=1.36	0.336 (0.293) OR=1.40
condom	0.301 (0.125)* OR=1.35	0.232 (0.128) OR=1.26	0.227 (0.145) OR=1.25	0.152 (0.146) OR=1.16	0.122 (0.149) OR=1.13
debut	-0.080 (0.029)** OR=0.92	-0.075 (0.028)* OR=0.93	0.000 (0.030) OR=1	0.004 (0.030) OR=1	0.005 (0.030) OR=1.01
smoker	0.467 (0.132)** OR=1.60	0.559 (0.145)** OR=1.75	0.390 (0.145)* OR=1.48	0.490 (0.159)** OR=1.63	0.486 (0.163)** OR=1.63
black		0.829 (0.144)** OR=2.29		0.827 (0.154)** OR=2.29	1.955 (0.324)** OR=7.06
vagnumsr			0.497 (0.073)** OR=1.64	0.495 (0.072)** OR=1.64	0.569 (0.089)** OR=1.77
blackXvagnumsr					-0.433 (0.117)** OR=0.65

intercept	2.236 (0.427)**	1.874 (0.432)**	-0.105 (0.515)	-0.455 (0.523)	-0.657 (0.567)
R2	0.12	0.16	0.25	0.28	0.26
N	1,411	1,411	1,410	1,410	1,410

* $p<0.05$; ** $p<0.01$

APPENDIX C: ADDITIONAL RESULTS FOR TABLE 4.7

Table 4.7 extended: Interactions between race and every other variable, vaginal HPV

Interaction	Logit Coefficient	Standard Error	T	P	Odds Ratio	R2
fampovratXblack	0.045446	0.079893	0.57	0.573	1.046495	0.2752
anyhiXblack	0.360233	0.248632	1.45	0.157	1.433664	0.277
hsgradXblack	-0.06602	0.473676	-0.14	0.89	0.93611	0.2775
condomXblack	0.046626	0.28699	0.16	0.872	1.04773	0.2777
debutXblack	0.060014	0.04862	1.23	0.226	1.061851	0.2742
smokerXblack	-0.18893	0.366168	-0.52	0.609	0.827842	0.2757
vagnumsr Xblack**	-0.43339	0.117199	-3.7	0.001	0.648306	0.2631
nosexXblack*	-1.89196	0.564061	-3.35	0.002	0.150776	0.1327

* p<0.05; ** p<0.01

APPENDIX D: ADDITIONAL RESULTS FOR TABLE 4.8

Table 4.8 extended: Interactions between race and other variables using divided-samples, vaginal HPV

Vaginal HPV risk Logit Coefficient	**Model 4: Black**	**Model 4: White**	**Model 6: Black**	**Model 6: White**
age	-0.032 OR=0.97	-0.038 OR=.96	-0.018 OR=0.98	-0.036 OR=0.96
fampovrat	-0.163 OR=0.85	-0.185 OR=.83	-0.214 OR=0.81	-0.153 OR=0.86
anyhi	0.186 OR=1.20	-0.171 OR=.84	0.337 OR=1.40	-0.053 OR=0.95
hsgrad	0.256 OR=1.29	0.705 OR=2.0	0.361 OR=1.43	0.503 OR=1.65
drinksperweek	0.023 OR=1.02	0.031 OR=1.03	0.019 OR=1.02	0.048 OR=1.05
debut	0.052 OR=1.05	0.023 OR=1.02		
condom	0.126 OR=1.13	0.206 OR=1.23		
smoker	0.289 OR=1.34	0.342 OR=1.41	0.026 OR=1.03	0.618 OR=1.86
vagnum	-0.061 OR=0.94	-0.032 OR=0.97		
vagnumsr	0.699 OR=2.01	0.816 OR=2.26		
nosex			-1.752 OR=0.17	-2.882 OR=0.06
intercept	-0.640	-1.440	1.010	0.628
R2	0.18	0.27	0.08	0.18
N	478	797	602	962

* $p<0.05$; ** $p<0.01$

APPENDIX E: ADDITIONAL RESULTS FOR TABLE 4.9

Table 4.9: Risk of oral HPV for black and white women ages 20-59

Oral HPV risk Logit Coefficient (Standard Error)	**Model 7**	**Model 8**	**Model 9**	**Model 10**	**Model 11**
age	0.005 (0.015) OR=1.01	0.007 (0.016) OR=1.01	-0.001 (0.015) OR=1.0	0.002 (0.015) OR=1.0	0.002 (0.015) OR=1.0
fampovrat	-0.174 (0.149) OR=0.84	-0.144 (0.149) OR=0.87	-0.217 (0.145) OR=0.80	-0.183 (0.146) OR=0.83	-0.189 (0.148) OR=0.83
anyhi	-0.033 (0.466) OR=0.97	-0.004 (0.466) OR=1	-0.032 (0.455) OR=0.97	-0.007 (0.462) OR=0.99	-0.003 (0.454) OR=1
hsgrad	-0.166 (0.483) OR=0.85	-0.140 (0.487) OR=0.87	-0.279 (0.477) OR=0.76	-0.234 (0.492) OR=0.79	-0.247 (0.488) OR=0.78
drinksperweek	-0.001 (0.015) OR=1	-0.002 (0.015) OR=1	-0.014 (0.018) OR=0.99	-0.015 (0.018) OR=0.99	-0.015 (0.018) OR=0.99
oraldebut	0.025 (0.020) OR=1.03	0.018 (0.023) OR=1.02	0.084 (0.032)* OR=1.09	0.077 (0.035)* OR=1.08	0.075 (0.033)* OR=1.08
smoker	1.275 (0.415)** OR=3.58	1.314 (0.424)** OR=3.72	1.112 (0.368)** OR=3.04	1.163 (0.378)** OR=3.20	1.149 (0.384)** OR=3.16
black		0.567 (0.358) OR=1.76		0.622 (0.380) OR=1.86	3.133 (1.752) OR=22.94
oralnum			-0.248 (0.143) OR=0.78	-0.250 (0.144) OR=0.78	-0.378 (0.226) OR=0.69
oralnumsr			2.079 (0.943)* OR=8.0	2.097 (0.951)* OR=8.14	2.869 (1.334)* OR=17.62

blackXoralnum					0.305 (0.230) OR=1.36
blackXoralnumsr					-1.963 (1.402) OR=0.14
intercept	-3.874 (1.102)**	-4.051 (1.147)**	-7.598 (1.860)**	-7.843 (1.850)**	-8.800 (2.011)**
R2	0.13	0.14	0.65	0.66	0.82
N	1,357	1,357	1,357	1,357	1,357

* $p<0.05$; ** $p<0.01$

APPENDIX F: ADDITIONAL RESULTS FOR TABLE 4.10

Table 4.10: Interactions between race and every other variable, oral HPV

Interaction	**Logit Coefficient**	**Standard Error**	**T**	**P**	**OR**	**R2**
fampovratXblack	0.155631	0.208567	0.75	0.461	1.168395	0.664
anyhiXblack	-0.36111	0.971817	-0.37	0.713	0.696901	0.6596
hsgradXblack	0.865514	0.927229	0.93	0.358	2.376226	0.6597
drinksperweekXblack	-0.03361	0.043728	-0.77	0.448	0.966953	0.6604
oraldebutXblack*	-0.11454	0.040392	-2.84	0.008	0.891774	0.659
smokerXblack	-0.59638	0.800301	-0.75	0.462	0.550801	0.6617
oralnumXblack	-0.00394	0.079508	-0.05	0.961	0.996068	0.6598
oralnumsrXblack	-0.20813	0.500502	-0.42	0.68	0.812103	0.6699
oralnumXblack AND oralnumsrXblack	0.305093 -1.962753	0.230176 1.401944	1.33 -1.4	0.194 0.171	1.356751 0.140471	0.8188
nooralXblack	0.15865	0.777434	0.2	0.84	1.171928	0.1522

* $p<0.05$; ** $p<0.01$

APPENDIX G: ADDITIONAL RESULTS FOR TABLE 4.11

Table 4.11: Interactions between race and other variables using divided-samples, oral HPV

Oral HPV risk Logit coefficient	**Model 10: Black**	**Model 10: White**	**Model 12: Black**	**Model 12: White**
age	0.041 OR=1.04	-0.012 OR=0.99	0.024 OR=1.02	0.007 OR=1.01
fampovrat	-0.062 OR=0.94	-0.216 OR=0.81	-0.122 OR=0.89	-0.125 OR=0.88
anyhi	-0.567 OR=0.57	0.193 OR=1.21	-0.411 OR=0.66	0.230 OR=1.26
hsgrad	0.018 OR=1.02	-0.525 OR=0.59	0.073 OR=1.08	-0.460 OR=0.63
drinksperweek	-0.015 OR=0.99	-0.012 OR=0.99	-0.016 OR=0.98	0.003 OR=1
oraldebut	-0.099 OR=0.91	0.149 OR=1.16		
smoker	0.651 OR=1.92	1.305 OR=3.69	0.933 OR=2.54	1.528 OR=4.61
oralnum	-0.007 OR=0.99	-0.471 OR=0.62		
oralnumsr	0.200 OR=1.22	3.573 OR=35.62		
nooral			-0.692 OR=0.50	-0.780 OR=0.46
intercept	-2.382	-10.717	-3.526	-3.810
R2	0.16	0.88	0.10	0.16
N	457	900	609	991

* $p<0.05$; ** $p<0.01$