SOCIOECONOMIC AND RACIAL-ETHNIC INEQUALITIES IN HUMAN PAPILLOMAVIRUS VACCINATION:
EMPIRICALLY TESTING FUNDAMENTAL CAUSE, HEALTH LIFESTYLE, AND PROSOCIAL MECHANISMS OF HEALTH DISPARITIES

by

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Socioeconomic and racial-ethnic inequalities in human papillomavirus vaccination: Empirically testing fundamental cause, health lifestyle, and prosocial mechanisms of health disparities

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Abstract

Socioeconomic and racial-ethnic inequalities in health are well documented, but the pathways underlying this relationship are not fully understood. To elucidate these pathways, I generate and test hypotheses about three facets of adolescent human papillomavirus (HPV) vaccination.

Fundamental cause theory contends health inequalities will shrink when policies support the equal distribution of interventions across populations. Using a triple-difference design, I analyzed the 2008–2009 and 2011–2013 National Immunization Survey–Teen (N=4,579 parents) to examine whether vaccine mandates shape provider recommendation and adolescent uptake of HPV vaccines, by socioeconomic status (SES) and race-ethnicity. I find mandates are associated with (a) improvements in provider recommendation for some SES and racial-ethnic groups (but not corresponding patterns of uptake) and (b) declining HPV vaccine uptake in the overall population.

Health lifestyle theory suggests that social groups engage in patterns of health behaviors that are reproduced within families. Analyzing the 2009 University of North Carolina (UNC) Mother–Daughter Communication Survey (N=951 mothers), I used multinomial logistic regression models to test whether mother–daughter communication about vaccines is associated with SES, race-ethnicity, and vaccine uptake. I find (a) Black (versus White) mothers are less likely to initiate conversations about HPV vaccination with their daughters and (b) mother–daughter communication increases the likelihood of HPV vaccination.

Research on prosocial behavior suggests that having fewer material resources may result in more positive attitudes toward and greater willingness to partake in health interventions that benefit others. Analyzing the 2010 UNC HPV Immunization in Sons Study (N=919 adolescent
boys and parents), I used seemingly unrelated regression models to test whether SES and race-ethnicity are associated with prosocial attitudes toward and willingness to vaccinate against HPV. I find (a) lower (versus Higher) educated parents and (b) Black and Hispanic (versus White) parents and adolescent boys report greater prosocial vaccination attitudes—some of which are associated with greater willingness to vaccinate.

These findings inform three important knowledge gaps for understanding health inequalities: the potential impact of adolescent-focused policy for mitigating health inequalities, the intergenerational reproduction of health inequalities within families, and the salience of prosocial attitudes for motivating health behaviors.
Lay Summary

Using the case of human papillomavirus (HPV) vaccination, this research focuses on understanding how socioeconomic status (SES) and race-ethnicity shape health. Analyzing US data, I examine whether SES and racial-ethnic inequalities in HPV vaccination are shaped by vaccine mandates, mother–daughter communication, and prosocial attitudes. I find that: (1) vaccine mandates facilitate provider recommendation and uptake of HPV vaccines for some SES and racial-ethnic groups, but lower rates of HPV vaccine uptake overall; (2) mother–daughter communication encourages HPV vaccine uptake but Black (versus White) mothers are less likely initiate conversations about HPV vaccination with their daughters; and (3) some lower (versus higher) educated parents and racial-ethnic minority (versus White) parents and adolescent boys are more willing to vaccinate to help prevent the spread of disease to others. These findings highlight the importance of policy, mother–daughter communication, and prosocial attitudes for shaping SES and racial-ethnic inequalities in health.
Preface

Andrea Polonijo identified and designed the research program and analyzed all research data for this dissertation. Data in chapter 3 come from the publicly available National Immunization Survey–Teen. Data in chapter 4 come from University of North Carolina (UNC) Mother–Daughter Communication Survey. Data in chapter 5 come from the UNC HPV Immunization in Sons Study. The University of British Columbia Behavioral Research Ethics Board approved the analyses conducted in chapters 4 and 5 (certificate number H13-02444).

A version of chapter 5 has been published: Polonijo, A. N., Carpiano, R. M., Reiter, P. L., & Brewer, N. T. (2016). Socioeconomic and racial-ethnic disparities in prosocial health attitudes: The case of human papillomavirus (HPV) vaccination for adolescent males. *Journal of Health and Social Behavior, 57*, 390–406. A. N. Polonijo conducted all statistical analyses and interpretations and wrote and revised the entire manuscript. R. M. Carpiano provided feedback on data interpretation and reviewed the final manuscript. P. L. Reiter and N. T. Brewer collected the original data, provided feedback on data interpretation, and reviewed the final manuscript.
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<th>Full Form</th>
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<td>ACIP</td>
<td>Advisory Committee on Immunization Practices</td>
</tr>
<tr>
<td>ASA</td>
<td>American Sociological Association</td>
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<tr>
<td>CDC</td>
<td>Centers for Disease Control and Prevention</td>
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<td>DC</td>
<td>District of Columbia</td>
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<tr>
<td>HPV</td>
<td>Human Papillomavirus</td>
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<tr>
<td>LPM</td>
<td>Linear Probability Model</td>
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<tr>
<td>MER</td>
<td>Marginal Effect at Representative Value</td>
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<tr>
<td>MICE</td>
<td>Multiple Imputation by Chained Equations</td>
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<tr>
<td>NCHS</td>
<td>National Center for Health Statistics</td>
</tr>
<tr>
<td>NCIRD</td>
<td>National Center for Immunization and Respiratory Diseases</td>
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<tr>
<td>NCSL</td>
<td>National Conference of State Legislatures</td>
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<tr>
<td>NIS–Teen</td>
<td>National Immunization Survey–Teen</td>
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<tr>
<td>OR</td>
<td>Odds Ratio</td>
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<tr>
<td>SES</td>
<td>Socioeconomic Status</td>
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<tr>
<td>UNC</td>
<td>University of North Carolina</td>
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<td>US</td>
<td>United States</td>
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<td>VFC</td>
<td>Vaccines for Children</td>
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Dedication

For Melba
Chapter 1: Introduction

How are people’s disease risk and general health shaped by their social position? Despite dramatic historical changes in the leading causes of death and their associated risk factors, inequalities in health have persisted such that individuals of lower socioeconomic status (SES) and racial-ethnic minority status experience poorer health than their higher SES and racial-ethnic majority counterparts (Link, Northridge, Phelan, & Ganz, 1998; Williams, 2012). Documentation of such inequalities exists across a broad range of health outcomes including mortality rates, disease incidence, and subjective health assessment ratings (Carpiano, Link, & Phelan, 2008; House, 2002; House, Lantz, & Herd, 2005; Williams & Sternthal, 2010). Similar inequalities have also been observed across an array of health-related behaviors, including diet (Darmon & Drewnowski, 2008; Hattery & Smith, 2011), exercise (Gryswackz & Marks, 2001; Saint Onge & Krueger, 2011), health service usage (Eisenberg & Power, 2000; Newacheck, Hung, Park, Brindis, & Irwin, 2003; Weinick, Zuvekas, & Cohen, 2000), and uptake of health-promoting technologies such as cancer screening tests (Garner, 2003; Link, 2008), medications (Gebo et al., 2005; Goldman & Lakdawalla, 2005), and vaccinations (Linn, Guralnik, & Patel, 2010; Polonijo & Carpiano, 2013).

A substantial body of sociological and social epidemiological inquiry suggests that material and psychosocial factors may explain the existence of SES and racial-ethnic inequalities in health. Material factors include those differences in tangible goods and services shaped by social and economic conditions (e.g., housing location and quality, accessibility of healthy food and other amenities including health services), that may influence individuals’ exposure to environmental health risks and affect their likelihood of adopting health promoting or health damaging behaviors, encountering life problems, or experiencing stress (Carpiano et al., 2008;
Lynch, Smith, Kaplan, & House, 2000; Marmot, 2005; Williams, Mohammed, Leavell, & Collins, 2010). Psychosocial factors concern the social meanings of one’s relative or absolute position in the social hierarchy, which may induce experiences of stigmatization and/or stress, and may lead to unhealthy coping behaviors and disease (Carpiano et al., 2008; Lynch et al., 2000; Williams & Sternthal, 2010; Wilkinson, 1997). The unequal distribution of material and psychosocial factors among social groups has been central to theories accounting for the relationship between social position and health, which will be investigated in this dissertation.

Two leading sociological theories have sought to explain the relationship between social position and health, and uncover some of the mechanisms that account for their association. Fundamental cause theory contends that greater access to resources (i.e., money, knowledge, prestige, power, and beneficial social connections) embodied by social position (including SES and race-ethnicity) allows higher status individuals to take advantage of new or existing health technologies to better avoid and treat disease (Link & Phelan, 1995). Health lifestyle theory offers a complementary perspective, suggesting that social classes engage in distinct collective patterns of behavior (or “lifestyles”) that have either a positive or negative impact on health, and arise as a result of individuals aligning their health-related choices with what is structurally possible given their resources and social class (Cockerham, 2005). Nevertheless, recent research on prosocial attitudes and behavior (see Piff, Kraus, Keltner, Côté, & Cheng, 2010) enhances understanding of the relationship between social position and health, suggesting that individuals with fewer material resources have more positive attitudes toward—and by extension may be more willing to partake in—health interventions that benefit others in their communities. Together, these theories concern the material and psychosocial factors that may drive social position-based inequalities in health attitudes, behaviors, and outcomes.
In this dissertation, I seek to understand how people’s disease risk and general health are shaped by their social position by testing aspects of fundamental cause theory, health lifestyle theory, and prosocial health attitude perspectives with respect to a recently introduced medical technology: human papillomavirus (HPV) vaccination. The first HPV vaccine entered the market in 2006 and has been targeted primarily toward adolescents to help prevent cervical (as well as other, less prevalent forms of) cancer—a disease for which SES- and race-ethnicity-based inequalities have been well documented (Barnholtz-Sloan et al., 2009; Garner, 2003). In relation to the three aforementioned theoretical perspectives, I examine three distinct facets of HPV vaccination:

1. The impact of mandates requiring HPV vaccines for school attendance on SES and racial-ethnic inequalities in HPV vaccination among adolescent girls.

2. SES and racial-ethnic differences in patterns of vaccine-related communication and decision-making among mothers and their adolescent daughters.

3. SES and racial-ethnic differences in prosocial attitudes toward HPV vaccination among adolescent boys and their parents.

In doing so, I aim to trace the reproduction of SES and racial-ethnic inequalities in health across generations and identify how inequalities may be linked to attitudes toward, decision-making about, and uptake of new health technologies.

The following introductory chapter is organized into three parts. I begin by describing the central concepts used in this dissertation, including health inequalities, SES, and race-ethnicity. Next, I introduce the theoretical perspectives and knowledge gaps that inform the objectives of my dissertation, and provide an overview of the empirical case of HPV vaccination. Finally, I
detail the purpose and objectives of the dissertation and significance of this research, and conclude with an overview of the layout of the forthcoming chapters.

**Conceptualizing Health Inequalities, SES, and Race-Ethnicity**

The three central concepts of this dissertation—health inequalities, SES, and race-ethnicity—are widely used, but are each complex. Therefore, it is necessary to first review these concepts before detailing the objectives of this dissertation.

**What is a health inequality?** While there are many definitions of “health inequality”, there is widespread consensus that the term refers to differences in health, disease, and mortality that exist between distinct segments of the population (Carter-Pokras & Baquet, 2002). Health inequalities also encompass differences in health-related behaviors (e.g., exercise, health service use, vaccination) that may shape health outcomes (Meyer, Yoon, & Kaufman, 2013). In the United States, the term “health disparity” is often used interchangeably with the term “health inequality” (Carter-Pokras & Baquet, 2002). Both concepts are typically measured by comparing the health of one segment of the population to either (a) another population segment, or (b) the general population (Department of Health and Human Services, 2000).

In the United States, health inequalities are particularly pronounced and well-documented among SES and racial-ethnic groups (Agency for Healthcare Research and Quality, 2013), which are the central foci of this dissertation. Such SES- and race-ethnicity-based inequalities are persistent across a wide range of health outcomes, including infant and adult mortality, acute and chronic infectious and noninfectious diseases, and psychiatric morbidity (Krieger, Williams, & Moss, 1997; Williams et al., 2010; Braveman, Cubbin, Egeter, Williams, & Pamuk, 2010). For instance, low-SES and racial-ethnic minority Americans have disproportionately higher rates of periodontitis (gum disease; Thornton-Evans et al., 2013), diabetes (Beckles & Chou, 2013),
obesity (May, Freedman, Sherry, & Blanck, 2013), tuberculosis (Khan, Magee, & Grant, 2013), and mental health issues (Zack, 2013) when compared to their higher-SES and racial-ethnic majority counterparts. Differences in life expectancy provide a profound example of health inequalities that are particularly striking when taking into account combined education (a key indicator of SES) and race-ethnicity. Recent data show that White men and women who attain a bachelor’s degree have life expectancies that are 14.2 and 10.3 years longer, respectively, than Black men and women who do not complete high school (Olshansky et al., 2012).

What is SES? The concept of “SES” is widely used in empirical studies, yet the term is rarely explicitly defined. Often the term is conflated and/or used interchangeably with “social class”, though these terms are conceptually distinct (see Muntaner, 2010; Veenstra, 2007; Wohlfarth, 1997) and measured using similar indicators (Carpiano et al., 2008). The differences between SES and social class are rooted in conflicting theoretical ideas about the nature of social inequality—a topic that has been central to the discipline of sociology since its inception. Classical theoretical ideas put forth by Karl Marx (1867/1992) and Max Weber (1922/1978; 1946), as well as the more contemporary theoretical ideas proposed by Pierre Bourdieu (1984; 1986) are key to understanding the distinct conceptualizations of social class and SES that are commonly used in the literature on health inequality today.

Contemporary conceptualizations of social class have drawn heavily on the classical work of Marx (1867/1992), who theorized that relationships between individuals and the means of production form the foundation of social inequality. From a Marxist perspective, those individuals who own and control the means of production are able to use their economic advantage to exploit workers who are dependent on selling their labor to survive (Marx, 1867/1992). These interdependent economic relationships between social groups form the basis of
Marxist-based conceptualizations of class, which remain influential in contemporary scholarship (see Wright, 2005). With regard to the relationship between class and health, it is theorized that individuals’ relationship to the means of production shapes their exposure to unique risk factors that result in the unequal distribution of disease and mortality (Galobardes, Lynch, & Smith, 2007). Collectively, studies using Marxist-inspired measures of social class such as occupational grade and status, business ownership, and control over organizational assets, have yielded results indicating a positive relationship exists between health and higher levels of ownership and managerial control (Veenstra, 2006; Muntaner et al., 2010). Contradictions to hypotheses found within this body of literature, however, also suggest that more complex factors affect health than solely one’s relationship with the economic labor market (for a review see Muntaner et al., 2010).

An alternative approach to defining social class that has recently been applied within the health inequality literature has drawn on the contemporary theoretical and empirical work of Bourdieu (1984; 1986). Bourdieu (1984) defined social classes as social groups defined by their relationship to the social space according to the possession and utilization of three key forms of capitals: economic, cultural, and social. Economic capital refers to economic resources, such as one’s income or assets, while cultural capital refers to the knowledge, skills, and formal education of the individual that function as a resource in society (Bourdieu, 1986). In contrast, social capital refers to those resources that one can draw upon via their social networks (Bourdieu, 1986). From a Bourdieusian perspective, the unequal distributions of these three distinct forms of capital determine the boundaries of social classes that tend to share similar social preferences and tastes. Those with the greatest amounts of capitals belong to the highest-class position. Regarding health, it is predicted that Bourdieusian-based conceptions of social
class are correlated with patterns of behavior (e.g., dietary choices, participation in physical activity, alcohol consumption) that shape overall health and well-being (Veenstra, 2007). While the application of Bourdieusian-based measures of social class to research on health inequalities has only recently begun to receive empirical attention (see Christensen & Carpiano, 2014; Gatrell, Popay, & Thomas, 2004; Veenstra, 2007), such conceptions of social class have been highly influential for shaping health lifestyle theory (see Cockerham, 2013), which is outlined in more detail later in this chapter.

Distinct from both Marxist and Bourdieusian conceptualizations of social class, SES remains conceptually distinct as it implies that a distinctly gradational relationship exists between social position and health. Modern conceptualizations of SES have been profoundly influenced by the work of Weber (1922/1978; 1946), who theorized that society is hierarchically stratified along multiple dimensions that interact to create groups of individuals from common social positions that share similar opportunities in life. Weber (1922/1978) identified economic class, status group, and political party as three primary sources of power that determine an individual’s position within the social hierarchy. Both economic class and status group remain central to modern conceptualizations of SES. According to Weber (1922/1978), economic class is based on the type and number of exchanges that individuals can pursue in the marketplace with their economic resources (e.g., their income, accumulated wealth, and assets). Status group, on the other hand, is defined by Weber (1922/1978) as being dependent on factors such as occupation, education, and consumption patterns that shape the degree to which individuals are positively or negatively perceived by others. Within the vast literature on health inequality, measures of SES have most often relied on multiple indicators of occupation, income, and/or educational attainment, reflecting Weber’s multidimensional approach to understanding social
stratification (Adler et al., 1994; Elo, 2009; Williams et al., 2010). In relation to health, it has been hypothesized that SES shapes the nature of individuals’ life opportunities, which produce circumstances that are either beneficial or disadvantageous to health and longevity (Elo, 2009; Link, Carpiano, & Weden, 2013).

Whether conceptualized as social class or SES, research evidence demonstrates that a positive relationship between social position and health has endured over time, despite the elimination of and changes in risk factors for disease (Carpiano et al., 2008; Link et al., 1998; Phelan, Link, Diez-Roux, Kawachi, & Levin, 2004). For the purposes of this dissertation, I review literature investigating health inequalities based on both social class and SES, however I focus my hypotheses, analyses, and discussion on SES. This decision is driven primarily by the data available for my analyses, which includes hierarchal indicators of education and income (i.e., level of schooling completed, annual income or income-to-poverty ratio), yet lacks indicators of occupation and/or social capital that would be appropriate for social class-based analyses.

Gradational associations between SES and a range of health outcomes have been demonstrated in multiple studies from both the developing and developed world (Adler & Rehkoph, 2008; Elo, 2009; Marmot, 1999). Overall, people of higher SES have longer life expectancies, enjoy better physical and mental health, and suffer less from disease and disability compared to their lower SES counterparts (Elo, 2009). For children, parental SES has been found to be predictive of health and mortality, suggesting that early SES-based life conditions affect health throughout the life course (Blackwell, Hayward, & Crimmins, 2001; Haas, 2008; Palloni, 2006). A number of SES-based inequalities in health risk (e.g., smoking, physical inactivity) and protective (e.g., disease screening, vaccination) behaviors have also been documented, which
overall suggest that higher SES individuals engage in cumulatively healthier behaviors than their lower SES counterparts (National Center for Health Statistics [NCHS], 2012).

**What does race-ethnicity entail?** Sociologists have long sought to delineate the meaning of race, identify how definitions of race change over time, and understand how race functions as a system of categorical classification (American Sociological Association [ASA], 2003). Contemporary definitions of race emphasize that race is a socially constructed category, reflecting historical oppression, exploitation, and social inequality (Williams & Sternthal, 2010). Racial categorizations are based on the association of individual and group characteristics with readily observable physical features such as skin color or hair type (Saperstein & Penner, 2012). The belief that race reflects real differences in physical, intellectual, moral, or spiritual superiority or inferiority, serves as the basis for the differential distribution of power, resources, and privileges in society and leads to the stigmatization and marginalization of non-White groups (Williams et al., 2010).

Though often used synonymously, the terms race and ethnicity are conceptually distinct. Ethnicity involves the categorization of social groups based on shared a nationality, ancestry, language, history, and/or cultural norms and traditions (Williams, 2012). The US government’s Office of Management and Budget (1997) officially recognizes five categories of race (White, Black, American Indian/Alaska Native, Asian, and Native Hawaiian/Pacific Islander) and one category of ethnicity (Hispanic). In reality, however, it is difficult to disentangle race from ethnicity, and the distinction between these concepts is often blurred (Williams et al., 2010). For example, though technically considered an ethnic group, the majority of Hispanic Americans prefer to identify themselves as a racial group (Tucker & Kojetin, 1996). Hence, I employ the
commonly used hybrid term “race-ethnicity” to encompass both race and ethnicity throughout this dissertation.

Research has revealed persistent, significant differences in life expectancy, disease-specific morbidity and mortality, and causes of death between racial-ethnic groups (ASA, 2003). Though, up until the early 20th century, these inequalities were thought to reflect innate biological differences between groups (Krieger, 1987), the contribution of biology to racial-ethnic differences in health is now considered to be relatively small (Chae, Nuru-Jeter, Lincoln, & Francis, 2011). Much of the existing US-based research on race-ethnicity and health has focused on comparisons of the three most sizable racial-ethnic groups, including non-Hispanic Whites (hereafter “Whites”), non-Hispanic Blacks (hereafter “Blacks”), and Hispanics, which are the three groups that I focus on in this dissertation. Blacks have poorer health status, earlier onset of disease, shorter life expectancies, and higher rates of mortality for 10 of the 15 leading causes of death in the United States when compared to Whites (Williams, 2012). While Hispanics have a slightly longer life expectancy than Whites, they have higher rates of several leading causes of death including diabetes, hypertension, and liver disease (Adler & Rehkoph, 2008).

There is widespread agreement that racial-ethnic status and SES are highly correlated (Braveman et al., 2010; Nazroo, 2003). When compared to Whites, both Blacks and Hispanics lag in high school and college completion rates, have less accumulated wealth at all levels of income (e.g., savings, assets), and are two to three times more likely to live in poverty (Williams et al., 2010). However, racial-ethnic inequalities in health persist even after taking into account SES differences, indicating that SES and race work as distinct systems of social stratification that jointly contribute to shaping health inequalities (Adler & Rehkoph, 2008; Braveman et al., 2010;
Williams & Sternthal, 2010). The direct effect of race on health may be the product of a number of factors, including segregation and discrimination. Racial-ethnic minorities are more likely to live in segregated communities with high levels of concentrated poverty, where neighborhood-level factors (e.g., availability of recreation facilities, access and quality of health services, exposure to environmental toxins) elevate one’s risk of illness and death (Williams, 1999). Racial-ethnic minorities are also more likely to experience harassment and discrimination, which can restrict resource access and trigger stressors that ultimately affect a number of health outcomes (Nazroo, 2003). Experiences of racial-ethnic discrimination are also linked to delays in healthcare seeking and lower adherence to medical treatment regimens, which can have a negative impact on disease management and progression, as well as mortality (Williams, 2012).

Hence, despite the strong association between race-ethnicity and SES, these two categorizations of social position may affect health through distinct pathways (Adler & Rehkoph, 2008).

**Theoretical Perspectives on Health Inequalities**

Having explained the three central constructs in this dissertation, I now focus on the three theoretical perspectives that I will apply with respect to vaccination for understanding how SES and race-ethnicity may produce health inequalities: fundamental cause theory, health lifestyle theory, and prosocial health attitudes. The first two of these perspectives, fundamental cause theory and health lifestyle theory, are leading sociological theories that have sought to explain the relationship between social position and health, and uncover some of the mechanisms that account for their association. The third perspective is not a formal theory per se, but rather a group of closely interrelated ideas generated from sociological and psychological scholarship pertaining to SES- and race-ethnicity-based patterns of prosocial attitudes and behaviors. Collectively, this third perspective leads to hypotheses that may enhance our understanding of
the relationship between SES, race-ethnicity, and health when the advantages of participating in a health intervention or adhering to a medical treatment extend beyond oneself to benefit others. Knowledge gaps in relation to these three theoretical perspectives offer fruitful areas for theoretical testing and expansion, which inform the primary objectives of this dissertation. Hence, examining these theoretical perspectives together may highlight the complexities of complementary mechanisms that underlie the relationship between SES, race-ethnicity, and health.

**Fundamental cause theory.** Fundamental cause theory contends that, due to the unequal distribution of health-beneficial resources in a society, health inequalities emerge and persist because persons who are more advantaged in terms of knowledge, money, status, and beneficial social connections are better positioned to avail themselves of health-promoting resources and innovations (Link & Phelan, 1995; Link & Phelan, 1996; Phelan & Link, 2013). By taking advantage of these health-promoting resources and innovations, more advantaged persons are able to better avoid diseases and their negative consequences, consequently allowing them to experience greater health and longevity than their less advantaged counterparts (Link & Phelan, 1995; Link et al., 1998). The theory articulates that fundamental causes become apparent under conditions of change, such as changes in diseases, treatments, and risk knowledge, because those resources that fundamental causes embody can be transported from one situation to another (Link, 2008; Phelan et al., 2004; Saldana-Ruiz, Clouston, Rubin, Colen, & Link, 2013; Wang, Clouston, Rubin, Colen, & Link, 2012). Hence, as “health-related situations change, those with the most resources are best able to avoid diseases and their consequences” (Link & Phelan, 1996, p. 492; see also Chang & Lauderdale, 2009; Rubin, Colen, & Link, 2010). From this perspective, health and social policy are closely linked to health inequalities, and this link can be weakened if
policies are implemented that distribute health enhancing knowledge, resources, and/or interventions equally across populations (Link, 2008; Phelan, Link, & Tehranifar, 2010; Rubin, Clouston, & Link, 2014).

A growing body of research has empirically tested fundamental cause theory in relation to SES and race-ethnicity and found support for its proposed mechanisms across a variety of health outcomes (see reviews by Phelan et al., 2010; Carpio et al., 2008). Consistent with fundamental cause theory, empirical tests have demonstrated: (a) more pronounced inequalities in relatively preventable diseases (e.g., heart disease) versus less preventable diseases (e.g., brain cancer; Phelan et al., 2004; Tehranifar et al., 2009), (b) the emergence of inequalities in preventive screening after the implementation and endorsement of diagnostic tools (e.g., mammography, Pap smear, and colorectal cancer screening; Link et al., 1998; Saldana-Ruiz et al., 2013), and (c) the exacerbation of inequalities in disease and mortality following the introduction of medical interventions (e.g., highly active anti-retroviral therapy for HIV/AIDS and statin drugs for high cholesterol; Rubin et al., 2010; Chang & Lauderdale, 2009).

Despite these insights provided by previous tests of fundamental cause theory, three key factors have yet to be evaluated empirically. First, the role that health and social policies play in mediating the fundamental cause relationship between SES, race-ethnicity, and health requires investigation. Second, few studies have considered how inequalities emerge at specific stages in the adoption of a medical treatment—from knowledge about a treatment to its uptake. Third, limited research has specifically examined this theory with respect to health-promoting innovations administered early in the life course that may have latent effects on morbidity and mortality that emerge at a later life stage (for an exception to the latter two factors, see Polonijo
These gaps in existing knowledge offer a logical direction for future research.

**Health lifestyle theory.** Health lifestyle theory offers a complementary perspective to fundamental cause theory for understanding health inequalities by emphasizing the role of structural factors in shaping the health behaviors of social groups, which ultimately affect health outcomes (Cockerham, 2005). Specifically, health lifestyle theory emphasizes the interplay between structural conditions and individual agency, and takes into consideration the tastes and preferences of social groups in an effort to understand how the personal routines of individuals merge to form distinct patterns of group-based behaviors (Cockerham, 2005; Cockerham, 2014). The theory assumes that people make realistic choices with regard to their health-related behaviors based on what is structurally possible, given the resources that they have available to achieve their wants and needs (Cockerham, 2013). At the aggregate level (e.g., societal group), these choices form “collective patterns of health related behavior”—or health lifestyles—that generally fall into one of two categories: good or bad (Cockerham, 2005, p. 55; Cockerham, 2014). The binary nature of health lifestyles means that they tend to have either positive or negative effects on health, illness, and mortality (Cockerham, 2013; Cockerham, 2014). From this theoretical perspective, lifestyles are one of the “primary mechanisms by which health is socially manufactured or undermined” (Cockerham, 2013, p. 7).

Empirical tests of health lifestyle theory have demonstrated the validity and utility of the concept of health lifestyles by highlighting the existence of social group-based patterns of health behaviors across a number of outcomes in a variety of geographic settings (Abel, 1991; Christensen & Carpiano, 2014; Cockerham, Hinote, Cockerham, & Abbott, 2006; Cockerham, Snead, & DeWaal, 2002). For example, studies incorporating class and/or SES-based indicators
of social position have observed differences in diet, exercise, alcohol consumption, smoking, and participation in annual physician checkups (Christensen & Carpiano, 2014; Grzywacz & Marks, 2001; Narcisse, Dedobbeleer, Contandriopoulos, & Ciampi, 2009; Snead & Cockerham, 2002). The vast majority of studies have indicated that those of higher social position have the healthiest lifestyles, while such lifestyles worsen as social standing decreases (for a review see Cockerham, 2013).

In terms of empirically testing health lifestyle theory, three key issues have yet to be explored. First, while previous applications of health lifestyle theory have focused on an array of health-related behaviors, research applying health lifestyle theory to preventive health treatment seeking remains scant. Second, although a substantial amount of attention has been paid to identifying group-based health lifestyles, few studies have set out to specifically observe racial-ethnic group-based differences in health lifestyles. Among the limited number of studies that have examined health lifestyles in relation to race-ethnicity, differences in exercise, diet, alcohol consumption, and cigarette smoking have been observed between racial-ethnic groups in the United States (Johnson & Hoffman, 2000; Saint Onge & Krueger, 2011; Gryswackz & Marks, 2001; Hattery & Smith, 2011). Third, health lifestyles have rarely been considered in relation to age or stage in the life course and few studies have explicitly examined health lifestyles among adolescents (for exceptions, see Burdette, Needham, Taylor, & Hill, 2017; Frohlich, Potvin, Gauvin, & Chabot, 2002; Frohlich, Potvin, Chabot, & Corin, 2002; Stead, McDermott, MacKintosh, & Adamson, 2011). With regard to the life course, it remains unclear the extent to which (a) adolescents possess agency over their own health behavior and/or are influenced by their parents and peers, and (b) parents maintain the agency to dictate the health-related behavior of their adolescent children. These gaps in understanding offer a logical direction for the
theoretical expansion of health lifestyle theory to better encompass not only SES, but also race-ethnicity and aspects of agency specific to one’s life course stage.

**Prosocial attitudes.** A third rarely considered, yet potentially useful perspective for understanding health inequalities concerns the role of SES and racial-ethnic differences in prosocial attitudes. The term “prosocial” means a benefit to others, which may or may not also benefit the self (Swap, 1991). Extensive sociological scholarship on social networks, social capital, and status groups suggests that lower-SES and racial-ethnic minority individuals are more prosocially oriented with regard to their attitudes and behaviors (see Portes, 1998; Petev, 2013). Such a tendency to act prosocially may be explained, in part, by scholarship on the close-knit nature of these groups and their strong community ties (Blau, 1974; Granovetter, 1983), as well as group-based norms that facilitate the sharing of resources, even when resources are scarce (Dominguez & Watkins, 2003; Portes, 1998). Recent psychological investigation complements sociological explanations and suggests that having fewer resources motivates individuals to act in a more prosocial manner (Piff et al., 2010). Other research suggests that the propensity to act prosocially carries over and is applicable to decisions concerning health behaviors that provide a social benefit (Shim, Chapman, Townsend, & Galvani, 2012; Vietri, Li, Galvani, & Chapman, 2012). These include behaviors such as vaccination or condom use, which provide protection against disease to both the self and others, as well as purely altruistic behaviors such as the donation of blood or organs, which benefit the recipient but not the donor (Morgan & Miller, 2002; Hyde, Knowles, & Wright, 2013; Vietri et al., 2012). Together, this body of work leads to hypotheses that lower SES and racial-ethnic minority individuals (compared to higher SES and White individuals) hold more positive attitudes toward prosocial health behaviors—and by extension, may be more willing to participate in such behaviors.
Though limited research to date has considered the relationship between SES, race-ethnicity, and prosocial health attitudes, the examination of SES and racial-ethnic differences in prosocial health attitudes offers a new and exciting direction for understanding the mechanisms that underlie health inequalities. Specifically, investigation of SES and racial-ethnic differences in prosocial health attitudes may help to ascertain whether social position is relevant for determining one’s willingness to participate in medical interventions that pose collective health benefits. Additionally, the incorporation of a prosocial attitude perspective may complement fundamental cause and health lifestyle theories, by drawing attention to conflicts that may arise between one’s prosocial attitudes and their ability to participate in prosocial health behaviors (which, may be dictated by factors such as socioeconomic constraints or insufficient knowledge about a beneficial health behavior).

The Case of HPV Vaccination

To investigate the aforementioned key issues pertaining to fundamental cause theory, health lifestyle theory, and prosocial attitude explanations for health inequalities, HPV vaccination offers a particularly useful empirical case. HPV is the most prevalent sexually transmitted infection in the United States, affecting an estimated 79 million Americans, with 14 million new diagnoses every year (Centers for Disease Control and Prevention [CDC], 2016a). Although the vast majority of HPV cases resolve on their own without treatment, HPV’s causal link to genital warts, nearly all cases of cervical cancer, an estimated 90% of anal cancers, 60% of certain oropharyngeal cancers, and 40% of penile, vaginal, and vulvar cancers makes it a serious health issue (Jemal et al., 2013; Pitts & Tufts, 2013). Of 26,000 new cases of HPV-attributable cancers diagnosed in the United States annually, 70% occur among females, the majority (64%) of which affect the cervix (Wu et al., 2012). Cervical cancer is a disease that
disproportionately affects low-SES, Black, and Hispanic women (Garner, 2003; Jemal et al., 2013; Watson et al., 2008; Wu et al., 2012). Largely accounting for these cervical cancer inequalities is underutilization of Pap smear screening among these groups, leading to delayed treatment until later stages of the disease and decreased odds of survival (Garner, 2003; Newmann & Garner, 2005; Singh, Miller, Hankey, & Edwards, 2004).

The US Advisory Committee on Immunization Practices (ACIP) recommends routine HPV vaccination for 11- and 12-year-olds and catch-up vaccination for older unvaccinated adolescents (Markowitz et al., 2014). Initially, two vaccines—Gardasil and Cervarix—were approved by the US Food and Drug Administration for HPV prevention. Gardasil was approved for females in 2006 and males in 2009, and licensed to help protect against cervical, vaginal, and vulvar cancer in females, as well as anal cancer and genital warts in both sexes, that are caused by four specific types of HPV (Markowitz et al., 2014). Cervarix was added to the market in 2009 and licensed only for females to help protect against cervical cancer caused by two specific types of HPV (Markowitz et al., 2014). In 2014, a third vaccine—Gardasil 9—was licensed for males and females aged 9 to 26 to help protect against cervical, vulvar, vaginal, and anal cancers and genital warts that are caused by nine specific types of HPV (CDC, 2016a). While Gardasil 9 is the only HPV vaccine that remains available in the United States today, it was not yet available when the data used in this dissertation were collected (2008–2013).

For maximum efficacy, the CDC and the American Academy of Pediatrics issued initial recommendations that HPV vaccines be administered as a series of three inoculations over a span of six months, and completed before the onset of sexual activity (CDC, 2016a; Rouzier & Giordanella, 2010). Given the emergence of evidence suggesting that just two inoculations were as efficacious as the three-shot series (Dobson et al., 2013; Kreimer et al., 2011), since October
2016 ACIP has advised that non-immunocompromised people aged 15 and under should receive just two innoculations, no less than five months apart (CDC, 2016a). All discussion of vaccine series completion in this dissertation refers to receiving three shots, as this is consistent with the routinely recommended number of shots in the years in which the data used in this dissertation were collected.

Most private health insurance covers the complete HPV vaccine series for adolescents, while uninsured, underinsured, Medicaid-eligible, and American Indian and Alaska Native adolescents are eligible for no-cost HPV vaccination through the national Vaccines for Children (VFC) program (Markowitz et al., 2014). It should be noted that both adolescents and health providers must register to participate in the VFC program, and that adolescents may still be required to pay an office visit fee to be vaccinated (CDC, 2016b); hence, barriers to HPV vaccine access may still exist even if one qualifies for the VFC program. HPV vaccines may also be obtained in safety-net clinics, mainly serving low-income and racial-ethnic-minority adolescents, which aim make healthcare more accessible for these groups (Tsui et al., 2013). Despite ACIP recommendations, VFC coverage, and the availability of HPV vaccines in safety-net clinics, as of 2015 only 50% of adolescent males and 63% of adolescent females had initiated the HPV vaccine series, while only 29% of boys and 42% of girls had completed the series (Reagan-Steiner et al., 2016).

Studies have identified recommendation by a healthcare provider as the key correlate of vaccine acceptance and uptake among both adolescents and their parents (Gilkey, Moss, McRee, & Brewer, 2012; Holman et al., 2014; Reiter et al., 2013). Healthcare providers are generally looked to as trusted sources of information about vaccines, while having greater trust in providers is positively associated with intentions to vaccinate (Dubé et al., 2018; Fu, Zimet,
Latkin, & Joseph, 2017; Griffioen et al., 2012). Acceptability of HPV vaccination for adolescents is also positively associated with greater knowledge about the vaccine and HPV, greater perceived vaccine safety and efficacy, greater anticipated regret that adolescents will contract HPV or HPV-related cancer after not being vaccinated, older age of the adolescent, and access to the vaccine at no cost (Donahue, Stupiansky, Alexander, & Zimet, 2014; Holman et al., 2014; Reiter, McRee, Gottlieb, & Brewer, 2010; Reiter, McRee, Kadis, & Brewer, 2011; Trim, Naji, Elit, & Roy, 2012). For adolescent boys, a perceived lack of direct benefit has been identified as a barrier to vaccination (Holman et al., 2014). Although much of the early publicity about HPV vaccines focused on (disproven) speculations that HPV vaccines would encourage promiscuity and sexual risk-taking among teens (see Smith, Kaufman, Strumph, & Levesque, 2015), studies have not consistently identified parental concerns about sexual activity as a barrier to adolescent HPV vaccination—and those that have suggest that these concerns are a barrier for only a small minority of parents (Brewer & Fazekas, 2007; Schuler, Reiter, Smith, & Brewer 2011; Trim et al., 2012).

**HPV vaccination and health inequalities.** To date, the case of adolescent HPV vaccination has elicited a number of concerns. Documenting socioeconomic and racial-ethnic inequalities in health provider recommendation and uptake of HPV vaccines has been a primary focus of both sociological and social epidemiological inquiry (Fisher, Trotter, Audrey, MacDonald-Wallis, & Hickman, 2013; Jeudin, Liveright, del Carmen, & Perkins, 2014; Niccolai, Mehta, & Hadler, 2011; Polonijo & Carpiano, 2013). Research suggests that low (versus high) SES and Black (versus White) adolescent girls are less likely to receive health provider recommendations to be vaccinated against HPV (Polonijo & Carpiano, 2013) and that the racial gap in provider recommendation has waned (but not disappeared) over time (Burdette,
While inequalities in vaccine uptake have also evolved over time (Burdette, Webb, et al., 2017), recent vaccination coverage estimates suggest that lower (versus higher) income and Black and Hispanic (versus non-Hispanic White) adolescents are more likely to initiate the HPV vaccine series, while lower SES and Black adolescent girls are less likely to complete the vaccine series and realize the full protective benefits of HPV vaccination (Curtis et al., 2014; Reagan-Steiner et al., 2015; Walker et al., 2017). Hispanic adolescents, though initially less likely to complete the vaccine series, now have higher rates of completion than non-Hispanic Whites (Reagan-Steiner et al., 2016).

As HPV vaccines are primarily targeted toward adolescents and most vaccine-related decision-making occurs within the family unit, their availability has also raised consciousness about the agency of adolescents (versus their parents) in the vaccination decision-making process (Katz et al., 2010; Mathur, Mathur, & Reichling, 2010; McRee, Reiter, & Brewer, 2010; Vietri, Chapman, Li, & Galvani, 2011). Moreover, because HPV vaccination was initially exclusively available for females and HPV-related cancers are more prevalent in women than men (Jeudin et al., 2014), questions have been raised about the willingness of adolescent boys and their parents to participate in HPV vaccination efforts (Reiter et al., 2013). Vaccinating low-SES, Hispanic, and Black adolescent males against HPV may be particularly important for protecting vulnerable females because young adults tend to have sexual partners from within their communities (Jeudin et al., 2014).

Given that HPV vaccines (a) have been unequally recommended to and adopted by different SES and racial-ethnic groups, (b) involve a degree of parent-child negotiation and decision-making when administered to their target population of adolescents, and (c) can be considered a prosocial health intervention, HPV vaccination offers an ideal empirical case for
testing hypotheses regarding fundamental cause theory, health lifestyle theory, and prosocial health attitudes. Despite this, relatively few empirical investigations have utilized sociologically informed theory to understand the social processes that underlie HPV vaccination. Theoretical perspectives that aim to explain the complex relationship between SES, race-ethnicity, and health may offer a fruitful line of inquiry for understanding inequalities in HPV vaccine-related attitudes, decision-making, and uptake, which may ultimately shape patterns of disease and mortality. Additionally, analysis of the case of HPV vaccination may also help to elicit some of the underlying mechanisms driving SES- and race-ethnicity-based inequalities in health with regard to the uptake of new medical technologies and provide an opportunity to test and expand upon existing sociological theory.

**Purpose and Significance of the Study**

With the aim of advancing knowledge about the mechanisms underlying SES and racial-ethnic inequalities in health, this dissertation uses the case of HPV vaccination to empirically test hypotheses based on fundamental cause theory, health lifestyle theory, and prosocial health attitudes. Specifically, I test these hypotheses by examining three distinct, yet interrelated facets of HPV vaccination.

First, with respect to fundamental cause theory, I examine the potential role of vaccination policy in reducing health inequalities that typically emerge when a new medical intervention becomes available. Based on the premises of the theory, inequalities should be smaller when policy supports the equal distribution of interventions across populations. To test this facet of fundamental cause theory, I analyze the impact of mandates requiring HPV vaccination for school attendance on SES and racial-ethnic inequalities in (a) adolescents receipt of a health provider recommendation to vaccinate, and (b) adolescents uptake of the vaccine. In
doing so, I not only provide a theoretically-informed test of the role of health policy with regard to the fundamental cause relationship, but also contribute to understandings of the emergence of inequalities at distinct stages in treatment adoption. Moreover, I shed light on the potential impact of adolescent-focused health policy for shaping health outcomes at later stages in the life course.

Second, with respect to health lifestyle theory, I examine how structural conditions shape SES- and race-ethnicity-based health lifestyles and consider their contribution to the reproduction of health inequalities across generations. Specifically, I investigate inequalities in HPV vaccine decision-making in the family context through an analysis of SES and racial-ethnic differences in (a) adolescent-parent communication about vaccines and (b) adolescent-parent agency in vaccine decision-making and uptake. For this analysis, I focus on three issues concerning (1) communication between mothers and daughters about HPV vaccination (and other vaccines administered in adolescence), (2) the impact of mother–daughter communication on vaccine uptake, and (3) the agency of parents versus adolescents in HPV vaccine conversation initiation and uptake. In doing so, I not only contribute to understanding the generation and reproduction of SES- and race-ethnicity-based health inequalities, but also address gaps in the health lifestyle theory literature with regard to the intergenerational transmission of health lifestyles within families and the significance of individual agency during adolescent life course stage.

Third, with respect to prosocial health attitudes, I investigate the potential roles that SES and race-ethnicity may have in shaping prosocial health attitudes—attitudes that may underlie certain prosocial health behaviors, including HPV vaccination. Given that HPV vaccination protects the vaccine recipient as well as their future sexual partners and communities against
disease, this medical technology can be considered prosocial in nature. Bringing together sociological and social psychological literatures, I generate and test a series of hypotheses concerning SES- and race-ethnicity-based prosocial attitudes toward HPV vaccination among adolescent boys and their parents. By engaging in this line of inquiry, I invite consideration of the role that SES- and race-ethnicity-based attitudes may play in shaping personal willingness to participate in future prosocial health behaviors. I also shed light on a previously under examined mechanism that may shape health inequalities.

In sum, by testing theoretically-motivated hypotheses related to these three complementary areas of inquiry, this dissertation contributes to the existing literature on SES- and race-ethnicity-based health inequalities by (a) extending discussion of the roles of material and psychosocial resources for shaping health inequalities, and (b) drawing specific attention to the emergence and intergenerational reproduction of inequalities in adolescence. Moreover, it contributes to specific knowledge surrounding SES- and race-ethnicity-based attitudes toward, decision-making about, and uptake of HPV vaccines, which may also be applicable to other vaccines and health interventions administered in adolescence.

**Organization**

The subsequent chapters are organized in the following manner. In chapter 2, I present an in-depth review of the literature related to the three theoretical perspectives that I have introduced above and provide an overview of key details pertaining to HPV vaccination that are relevant to my empirical tests. Building on this review of existing knowledge, in chapter 2 I also outline my hypotheses concerning SES- and race-ethnicity-based health inequalities in relation to the theoretical perspectives and the empirical case of HPV vaccination. In chapters 3, 4, and 5, I proceed to empirically test my hypotheses with regard to fundamental cause theory, health
lifestyle theory, and prosocial health attitudes, respectively. Each of these chapters will begin with a brief overview of the hypotheses to be tested, detail the methodology and analytic strategy in relation to these hypotheses, present the findings of the analyses, and discuss their theoretical implications. Given that each of the analytic chapters relies on a unique dataset for analysis, rather than discussing the methodology in a stand-alone chapter, for the purpose of clarity, the methods are presented separately within each of the analytic chapters. In chapter 6, I conclude with a discussion of the broader implications of the findings reported in chapters 3 through 5 and their relevance for understanding health inequalities. In this concluding chapter, I also acknowledge and address my study’s strengths and limitations, and identify promising directions for future research.
Chapter 2: Literature Review and Hypotheses

In the present chapter, I explicate how the three perspectives guiding my dissertation—fundamental cause, health lifestyle, and prosocial attitudes—inform my specific hypotheses regarding the case of HPV vaccination. This elaboration is organized in three sections. First, I summarize theory and previous research on SES- and race-ethnicity-based health inequalities in relation to fundamental cause theory, health lifestyle theory, and prosocial health attitudes. Second, I discuss how HPV vaccination offers an important empirical case for evaluating aspects of these theories and review selected relevant literature on this public health issue. Third, I detail the aims and hypotheses of this dissertation.

Fundamental Cause Theory

Fundamental cause theory aims to understand how social factors may shape health inequalities via resource differentials and social mechanisms that link resources to health through psychosocial and material pathways (Link & Phelan, 1995; Carpiano et al., 2008). The theory emphasizes that social position (including SES and race-ethnicity) has had an enduring association with health over time, despite the evolution of diseases and risk factors (Link & Phelan, 1995; Phelan et al., 2004). This relationship is analogous to what Lieberson (1985) conceptually describes as a “basic cause”—whereby one variable has a lasting effect on another despite changing mechanisms, due to the emergence or increased prominence of new mechanisms upon the decline of others. House and co-authors (see House et al., 1990; House et al., 1994) were the first to propose that the enduring relationship between SES and mortality were the product of such basic causes. However, this work did not offer an explanation as to how basic causes are able to reproduce their effects when intervening mechanisms—such as changes
in risk factors, disease screening tools, or medical treatments—are eliminated or change (Phelan et al., 2004).

**The role of flexible resources.** Building on the earlier work of Lieberson (1985) and House et al. (1990; 1994), Link and Phelan (1995), in their seminal article on “Social Conditions as Fundamental Causes of Disease,” propose that social gradients in health persist because higher social position embodies greater access to flexible resources that impact individual health behaviors and overall health, regardless of the mechanisms relevant at any given time (Link & Phelan, 1995). These resources, such as money, knowledge, prestige, power, and beneficial social connections, work to protect health by “influencing whether people know about, have access to, can afford, and are motivated to engage in health-enhancing behaviors” (Phelan et al., 2004, p. 267). Contemporary examples of such health-enhancing behaviors include eating a healthy diet, exercising regularly, staying up to date on vaccinations, or knowing about and asking one’s healthcare provider about disease screening or treatments (Phelan et al., 2004). Flexible resources may also shape one’s access to broad contexts (e.g., neighborhoods, occupations, social networks) that may influence one’s exposure to risk and protective factors (Phelan et al., 2004). For instance, healthcare access and quality may be lacking in impoverished neighborhoods, manual labor jobs may be associated with dangerous working conditions and substandard health benefits, and social networks consisting of high-status peers may expose individuals to valuable health-promoting knowledge (Phelan et al., 2004).

**Changes in intervening mechanisms across time.** From a fundamental cause perspective, flexible resources affect a person’s ability to avoid risks and minimize the impact of disease once it occurs, regardless of the prevailing diseases, risk factors, or treatments (Link & Phelan, 1995). While addressing current risks may temporarily improve inequalities in disease
and mortality, fundamental cause theory posits that their effects cannot be expected to be long lasting because intervening mechanisms can be expected to change (Phelan et al., 2004). For example, while improvements in housing quality, sanitation, and vaccination eradicated infectious diseases common in the 19th century (e.g., diphtheria, smallpox, tuberculosis), chronic diseases (e.g., cancers, heart disease) linked to new risk factors (e.g., smoking, diet, exercise) have emerged as major causes of mortality (Phelan & Link, 2005). Despite this dramatic change in risk factors and disease regimes, gradients in mortality persisted across time (Phelan et al., 2010). Hence, Link and Phelan (1995) contend that social factors, such as SES and race-ethnicity, are “fundamental causes of disease” because they “affect multiple disease outcomes through multiple mechanisms, and consequently maintain an association with disease even when intervening mechanisms change” (Link & Phelan, 1995, p. 80). Key to the fundamental cause argument is the proposition that inequalities will arise when humans develop the capacity to control specific health conditions because the benefits of health interventions and innovations will not be evenly distributed (Carpiano et al., 2008; Link, 2008; Saldana-Ruiz et al., 2013; Wang et al., 2012). Since the initial articulation of fundamental cause theory, over 20 years ago, the theory has remained influential and prompted empirical testing and expansion.

**Empirical support for fundamental cause theory.** A review of empirical tests of fundamental cause theory reveals results overwhelmingly consistent with SES- and race-ethnicity-based fundamental cause predictions. Such theoretical tests have generally addressed one of three types of health inequalities: (1) inequalities in preventable versus less preventable diseases, (2) inequalities in diagnostic screening, treatment uptake, and disease-specific mortality before and after the introduction of new knowledge, screening tools, or medical treatments, or (3) inequalities in the uptake of disease screening or medical treatments over time.
**Inequalities in preventable versus less preventable diseases.** Three noteworthy studies have investigated inequalities in preventable versus less preventable diseases. Using US data, Phelan et al. (2004) found that individuals of lower SES have higher likelihoods of dying from diseases that are relatively preventable (e.g., heart disease, cervical cancer) versus diseases that are less preventable (e.g., brain cancer, multiple sclerosis). Similarly, Tehranifar et al. (2009) observed racial-ethnic inequalities in cancer survival in the United States that widened as cancers became more amenable to medical intervention. Using data from 19 European countries, Mackenbach et al. (2015) identified relative SES-based inequalities in mortality that were generally larger for causes of death amenable to behavior change, medical intervention, or injury prevention versus non-preventable causes.

Though not a comparison of preventable versus less preventable disease, an investigation of racial inequalities in stage of diagnosis and survival from ovarian cancer by Kim, Dolecek, and Davis (2010) complements the above studies. As predicted, in the general absence of symptoms or an effective diagnostic screening tool, the authors find no racial differences in stage of ovarian cancer diagnosis. However, after ovarian cancer is diagnosed (making it amenable to medical intervention), less favorable survivor outcomes exist for Blacks versus Whites (Kim et al., 2010).

**Inequalities following the introduction of new medical knowledge, treatments, and/or diagnostic tools.** A number of empirical tests of fundamental cause theory have documented the emergence or exacerbation of SES- and race-ethnicity-based health inequalities following the introduction of new medical knowledge, treatments, or diagnostic tools. Revealing the emergence of inequalities, Miech (2008) identifies how SES-based inequalities in cocaine use abruptly emerged in the 1980s and 1990s, following a cultural redefinition of cocaine use as
being unhealthy, which led to a decrease in usage by higher SES groups but not their lower SES counterparts. Similarly, Saldana-Ruiz et al. (2013) discovered the emergence of SES-based inequalities in colorectal cancer mortality following advances in colorectal screening recommendations and diagnostic tools. Likewise, Clouston, Rubin, Colen, and Link (2014) discovered the emergence is SES-based inequalities in county-level suicide rates in the United States, following the introduction of selective serotonin reuptake inhibitors to treat depression and prevent suicide. Highlighting the exacerbation of inequalities, Rubin, Colen, and Link (2010) reported that, though lowering overall HIV/AIDS related mortality, the introduction of highly active anti-retroviral therapy increased SES- and race-ethnicity-based inequalities in HIV/AIDS mortality.

Other studies have identified the reversal of health inequalities, favoring Whites and those of higher SES, following the dissemination of new medical knowledge or treatments. Rubin, Clouston, and Link (2014) found that, after knowledge of the causal link between smoking cigarettes and lung cancer was disseminated in the United States, lung cancer mortality rates that were initially greater in higher SES counties became greater in lower SES counties (while, over the same period, rates of relatively unpreventable pancreatic cancer remained unchanged). Similarly, Chang and Lauderdale (2009) found that, once cholesterol lowering statin drugs became available, the initially positive association between income and cholesterol levels reversed to become negative.

**Inequalities in disease screening and treatment uptake over time.** In addition to the preceding findings, a few studies have documented SES and/or racial-ethnic differences in disease screening over time. Link et al. (1998) identified the emergence of SES-based inequalities in mammography screening after the widespread implementation and endorsement
of this diagnostic tool. With regard to HPV vaccination in the years following the vaccines’ initial approval (2008–2010), Polonijo and Carpiano (2013) demonstrated that SES- and race-ethnicity-based inequalities persisted across distinct stages treatment adoption, including knowledge, health professional recommendation, and uptake.

Together, this body of empirical research supports that SES and race-ethnicity-based health inequalities are: (a) most pronounced for diseases that are amenable to prevention, and (b) shaped by differences in health behaviors and/or utilization of health promoting innovations, following the introduction of new health-promoting knowledge, treatments, or screening tools.

**Mechanisms of fundamental causes.** The elucidation of mechanisms, or pathways, by which fundamental causes affect health has been an important focus of theoretical expansion. In an ethnographic study of disease management in two diabetes clinics, Lutfey and Freese (2005) highlight the existence of multiple mechanisms that sustain the relationship between social position and health. The authors observed differences in diabetes patient’s cognitive abilities (e.g., communication skills, ability to follow treatment regimens) and motivations (e.g., the costs and benefits of complying with treatment, the relative magnitude of proposed lifestyle adjustments), as well as external structural constraints (e.g., occupational demands, financial constraints, health insurance) and organizational features of the clinics (e.g., doctor specializations, in-clinic educational resources, continuity of care), which allowed higher SES patients to better manage their disease. The authors also identified several “compensatory inversions”, whereby the best resources were disproportionately allocated to higher SES patients, who had the least need for them (Lutfey & Freese, 2005, p. 1345).

Lutfey and Freese’s (2005) research also highlights the existence of countervailing mechanisms, which run counter to the direction of the fundamental cause relationship but have
effects that are cumulatively smaller than the mechanisms that produce the fundamental relationship. Specifically, the authors observed a conflict between status maintenance and optimal health behaviors among White and middle-class girls, who went against medical advice for disease management in an effort to achieve a thin physique. While this conflict created pathways that worked against the fundamental cause relationship, the overall balance of mechanisms sustained the relationship between social position and health outcomes (Lutfey & Freese, 2005).

In a further elaboration on the discussion of mechanisms, Freese and Lutfey (2011) propose four key metamechanisms that help to explain the durable relationship between social position and health. More conceptually abstract than mechanisms, the authors posit that metamechanisms explain the generation of “multiple concrete mechanisms that reproduce a particular relationship in different places and different times” (Lutfey & Freese, 2011, p. 69). Their four proposed metamechanisms include: means, habitus, institutions, and spillovers.

Means describes the purposive use of resources to improve one’s health. Some examples of means include paying for a personal trainer, accessing a health specialist, and taking advantage of disease screening technologies (Freese & Lutfey, 2011).

In contrast to the purposive nature of means, habitus describes the less conscious norms, preferences, and lifestyles of different groups (e.g., dietary preferences, occupational choice) that may affect health outcomes. The term habitus will be revisited and expanded upon later in this chapter, in relation to health lifestyle theory.

Shifting away from ideas suggesting individual agency, the metamechanism of institutions refers to the ways in which institutions differentially treat individuals and better facilitate health gains for certain groups (Freese & Lutfey, 2011). For example, empirical work
has shown that, despite presenting with uniform symptoms and diseases, patient characteristics such as SES and race-ethnicity (in addition to other factors such as gender or age) may influence the accuracy of physician diagnosis and quality of follow-up care (Arber et al., 2004; Lutfey & Freese, 2005; McKinlay, 1996).

Lastly, the metamechanism of spillovers refers to the actions of others in one’s social network that may benefit an individual even when they do not engage in any purposeful action themselves (Freese & Lutfey, 2011). Spillovers may have the greatest impact on those who are socially close and, because social distance is lower among people with similar SES, higher status individuals are more likely to benefit from new health knowledge—even if they do not take personal action on a particular health issue (Freese & Lutfey, 2011). For example, despite not caring about their health, individuals may still benefit from the actions of community members who champion for better health and social services, refrain from smoking in public spaces, or choose to vaccinate themselves. In their examination of HPV vaccination to test fundamental cause mechanisms, Polonijo and Carpiano (2013) suggest that spillover effects may be particularly pertinent to children and adolescents, as they are embedded in a network of family members whose actions and resources may have consequences for their health and well-being.

In addition to the aforementioned work, Polonijo and Carpiano (2013) further the discussion surrounding mechanisms underlying fundamental causes by introducing the concept of latent mechanisms. Latent mechanisms describe how preventive treatments administered at one point in the life course may, when taken up unequally, have an effect on disease-related morbidity and mortality that is “not immediately evident, but rather, would be revealed at a later stage of life” (Polonijo & Carpiano, 2013, p. 115). Specifically, they suggest that inequalities in
adolescent HPV vaccination may act as a latent mechanism to reproduce inequalities in HPV-related cancers, which are typically observed in later adulthood.

Areas for expansion of fundamental cause theory. Overall, the fundamental cause literature offers concrete examples of the ways in which human action can shape social patterns of morbidity and mortality and exemplifies how seemingly well-intentioned interventions and medical technologies can unintentionally increase health inequalities between SES and racial-ethnic groups (Link et al., 1998; Polonijo & Carpiano, 2013; Rubin et al., 2010). However, there are three key areas where more research is needed to understand the mechanisms by which SES and racial-ethnic inequalities emerge and persist across time.

First, fundamental cause theory posits that the association between medical innovations and SES and race-ethnicity will shrink if policies are implemented that distribute interventions equally across populations (Link, 2008), though this conjecture has received limited empirical attention. An analysis of the relative impact of SES as a fundamental cause in the United States versus Canada (a neighboring country with publicly funded, universal healthcare) posits that policy acts as a buffer to the relationship between SES and health for highly preventable disease (Wilson, 2009). Observations of the persistence of SES-based health inequalities in Western European countries with highly developed social welfare states, however, have led others to speculate that the impact of policy is limited because (a) new mechanisms may emerge that replace mechanisms attenuated or eliminated by social policy and (b) people of higher social position may be able to make better use of social resources (Mackenbach et al., 2008; Mackenbach, 2012). Further empirical testing of whether policy that aims to make health interventions equally accessible to all is associated with a reduction in SES and racial-ethnic health inequalities is an important direction for future inquiry.
Second, more research is needed to better elucidate how flexible resources (i.e., knowledge, power, prestige, and beneficial social connections) operate to create health inequalities. Observing how inequalities emerge at specific stages in the adoption of a medical treatment—from knowledge about a treatment through to treatment uptake—may be helpful in this endeavor. Given that very few studies have traced the emergence of disparities at distinct stages of treatment adoption (for an exception see Polonijo & Carpiano, 2013), this remains a logical area for future theoretically motivated testing.

Third, there is a dearth of fundamental cause theory research that considers health-promoting innovations that are administered early in the life course. Observing how treatments administered in childhood and/or adolescence may be unequally taken up by different SES and racial-ethnic groups is important for posing conjectures as to how early life course health interventions may latently shape patterns of disease-specific morbidity and mortality in adulthood. Moreover, drawing attention to early life course interventions may also be productive for better understanding how the attitudes and actions of adults (in particular, parents and health providers) spillover and contribute to shaping health inequalities among children and adolescents.

**Health Lifestyle Theory**

A complementary perspective to attempt to explain social inequalities in health and elucidate some of the underlying mechanisms driving SES- and race-ethnicity-based health inequalities is health lifestyle theory. The theory contends that daily behaviors—or lifestyles—are one of the “primary mechanisms by which health is socially manufactured or undermined” (Cockerham, 2013, p. 7). Rather than being driven purely by individual will or free choice, the theory asserts that the decisions people make with regard to food, exercise, smoking or other
health behaviors are shaped by structural factors such as SES and race-ethnicity that constrain the options that individuals have to choose from (Cockerham, 2013). Accordingly, health lifestyles tend to cluster in patterns that reflect one’s social position (Cockerham, 2013).

**Agency, structure, and habitus: The foundations of health lifestyles.** Formally articulated by Cockerham (2005), the roots of health lifestyle theory can be traced back to Weber’s (1922/1978) classic concept of lifestyles. For Weber (1922/1978), lifestyles were status-based phenomena by which people who sought to be part of a particular status group were required to adopt a congruent lifestyle. Because of this adoption, status groups were then stratified by the consumption patterns of their members. Weber’s (1922/1978) lifestyle concept takes into account agency and structure—concepts that are central to the discipline of sociology. Specifically, Weber (1946) notes that the life choices or personal agency of individuals is constrained by the life chances that they are afforded according to their class or status position (Weber, 1946). That is, while individuals may choose a lifestyle, the social context (or structural conditions) provided by their life chances limits the lifestyles that they can realistically adopt (Cockerham, Rutten, & Abel, 1997).

In addition to Weber’s scholarship, health lifestyle theory draws upon the more contemporary work of Giddens (1991; 1984), who maintained that (a) modern society has fostered a diversity of lifestyle choices, which forces even the lowest classes to make decisions, (b) individuals’ lifestyle choices tend to cluster and form predictable patterns based on social position, and (c) structure is both a constraining and enabling force. Giddens (1984) argues that while structure may restrict the choices available to people, the resources afforded by structure (e.g., finances, status) can also help individuals realize their choices. Health lifestyle theory also invokes Bauman’s (1999) observation that all choices—no matter what the circumstances—are
constrained by the options available and the social norms that govern the desirability and appropriateness of preferences. Structural influence is evident in an array of consumption patterns signaling membership to social class (Baumann, 1999; Giddens, 1991).

Health lifestyle theory also embodies Bourdieu’s (1984) notion of “habitus”—the routine social orientations of individuals toward action (i.e., tastes, preferences, values), which are produced as a result of living under class conditions. Through empirical exploration of survey data in 1960s France, Bourdieu identified distinct class-based preferences in terms of cultural tastes, in a number of domains including food, music, and leisure activities (Bourdieu, 1984). For example, Bourdieu observed a preference for cheap and nutrient dense foods among the working class, while professionals preferred tasty, healthy, light, and low-fat meals (Bourdieu, 1984). Bourdieu (1984) theorized that these class-based preferences were reproduced across generations and shaped primarily by parent’s economic resources and cultural capital—the knowledge, skills, and formal education of the individual that function as a resource in society. Bourdieu argued that while culture serves to unconsciously engrain habitus in individuals, enacting these choices reproduces the class culture itself (Bourdieu, 1984).

**A formal theory of health lifestyles.** Cockerham’s (2005; 2013) formal articulation of health lifestyle theory draws on the aforementioned foundations by emphasizing the interplay between life choices (e.g., school choice, health-related decisions) and life chances (e.g., SES, race-ethnicity, gender, age) and considering tastes and preferences, and the interplay between actors and conditions. The theory conjectures that people have goals, needs, and desires that they align with their probabilities for realizing them and assumes people will make realistic choices based on what is structurally possible, given their available resources and social position (Cockerham, 2005; Cockerham, 2014). At the aggregate level, individual choices form
“collective patterns of health-related behavior”—or health lifestyles, which tend to be binary and have either positive or negative effects on health (Cockerham, 2005, p. 55; Cockerham, 2013). According to health lifestyle theory, health knowledge and economic resources shape individuals’ health lifestyles, while social norms and other social supports (for example, the health-product industry) reinforce distinct health lifestyles (for example, practices related to exercise and diet) according to social position that influence the behaviors of other group members (Cockerham, 2005).

**The role of collectivities for shaping health lifestyles.** In addition to the role of SES, race-ethnicity, and other variables, health lifestyle theory speaks directly to the role of families—as collectivities—in the transmission of health lifestyles. Cockerham (2013, p. 143) defines collectivities as “collections of actors linked together through specific social relationships and networks, such as the workplace, kinship, religion, and politics”. It is hypothesized that, as a collectivity, families share their norms, values, and ideals in the primary socialization of children, and transmit knowledge about their social status, cultural orientation, and medical history (Cockerham, 2013). While children may either accept or reject the social perspectives of their family, they are nonetheless presented with the views of their kin, which shape the social world in which they live and may have a long-lasting influence on their preferences and behaviors (Cockerham, 2013).

Complementary life course research indicates that as children transition into adolescence, health decision-making shifts from the parent to the adolescent (Umberson, Crosnoe, & Reczek, 2000). At the same time of transition, adolescents usually experience a rapid expansion of social networks to include peers, whose norms and values may contradict those of parents (Cockerham,
2013; Umberson et al., 2000). Such peer groups may function as collectivities that either bolster or weaken the influence of family values on adolescents’ health behaviors and health lifestyles.

**Empirical support for health lifestyle theory.** Empirical work has found support for health lifestyle theory in a variety of contexts. In a comparative analysis of Germany and the United States, Abel (1991) demonstrated the validity of the concept of health lifestyles by identifying three distinct health lifestyle groups in both nations that were characterized by sets of shared behaviors. Research exploring health lifestyles in post-Soviet countries has also demonstrated that poor health practices are deeply engrained in an individual’s habitus (Cockerham et al., 2002) and that anti-socialists, as a collectivity, have healthier lifestyles compared to individuals who want to return to communism (Cockerham, Hinote, Cockerham, & Abbott, 2006). Cockerham (2000) also found that the longevity of middle-aged blue collar workers in Russia, were adversely affected by societal and group norms that led them to participate in behaviors that put them at risk for cardiovascular disease, including heavy drinking, smoking, high-fat diets, and a lack of exercise (see also Cockerham et al., 1997; Cockerham, Hinote, & Abbott, 2006). While the majority of studies applying health lifestyle theory have focused on adult populations, recent research by Burdette, Needham, et al. (2017) identified clusters of health behaviors that formed distinct health lifestyles among US adolescents. Directly applicable to the present investigation of health lifestyles is additional research that has addressed the specific roles of (1) SES, (2) race-ethnicity, and (3) collectivities for reproducing and shaping health lifestyles.

**SES and the reproduction of health lifestyles.** Empirical work has demonstrated that SES indicators, such as income and education, have a powerful influence on health lifestyles in adulthood (Cockerham, 2013). Research evidence overwhelmingly demonstrates that the most
affluent and educated individuals tend to have the healthiest lifestyles, while such lifestyles worsen as social position decreases (Cockerham, 2013). Studies using income and educational indicators of social position have observed that adults of higher rank in the social hierarchy tend to have healthier diets, higher rates of participation in physical activity, lower rates of participation in risk behaviors such as smoking and excessive drinking, and are more likely to have regular physical checkups with physicians and dentists (Andrews, Hill, and Cockerham, 2017; Christensen & Carpiano, 2014; Grzywacz & Marks, 2001; Narcisse et al., 2009; Snead & Cockerham, 2002). Those of higher class and/or SES are also the first to have knowledge of new health risks and, given that they have fewer structural limitations that prohibit the adoption of healthy behaviors, are the first to adopt new beneficial health practices (Cockerham, 2005; Carpiano et al., 2008). Research on the impact of parental SES on adolescent health lifestyles has yielded less consistent findings, with some studies identifying a positive association between parental SES and healthy lifestyles and others finding no association (Leech, McNaughton, & Timperio, 2014).

**Race-ethnicity and the reproduction of health lifestyles.** Alongside income- and education-based indicators of social position, race-ethnicity has been identified as being influential for shaping health lifestyles (Cockerham, 2013). Although relatively few studies have examined racial-ethnic differences in health lifestyles, the findings of these studies are consistent with the health lifestyle concept. For example, Saint Onge and Krueger (2011) identify patterns in the types of exercise engaged in by racial-ethnic groups in the United States, with Whites disproportionately partaking in facility-based exercise (e.g., swimming, golf, tennis), Blacks participating in more team and fitness activities, and Mexican Americans engaging in more team sports. In line with the structural limitations acknowledged by health lifestyle theory, the authors
suggest that lack of access (or perceived access) may prevent racial-ethnic minority groups from participating in the facility-based exercises that are favored by Whites. Other work by Grzywacz and Marks (2001) suggests that participation in exercise declines more steeply for Blacks than Whites across the life course, which may be caused by both the earlier development of functional health problems among Blacks and greater concentration of Blacks in unsafe neighborhoods that restrict motivation to participate in outdoor activities. With regard to diet, Hattery and Smith (2011) identify lower nutrition diets among Blacks (compared to Whites), which they attribute to the disproportionate number of Blacks that live in poverty. Other racial-ethnic comparisons health lifestyles in the United States have shown that White adults smoke, drink alcohol, exercise, and practice weight control more than Black adults (Cockerham, 2013), that White adolescents engage in riskier lifestyles than their Black counterparts, (e.g., smoking, drinking, not wearing seatbelts; Blum et al., 2000; Burdette, Needham, et al., 2017), and that Black adolescents have more sedentary lifestyles than other racial-ethnic groups (Boone-Heinoin, Gordon-Larsen, & Adair, 2008). Together, such evidence highlights the existence of race-ethnicity-based health lifestyles in both adolescence and adulthood.

**Collectivities and the shaping of health lifestyles.** Although there is a lack of empirical research on the reproduction of health lifestyles within the family context, research by Christensen (2011), Frohlich and colleagues (Frohlich, Potvin, Gauvin, & Chabot, 2002; Frohlich, Potvin, Chabot, & Corin, 2002), and Wickrama and colleagues (Wickrama, Conger, Wallace, & Elder, 1999) sheds light on how both families and social structures shape the health lifestyles of adolescents. In an examination of childhood body weight, Christensen (2011) finds that the perceptions and practices of parents are embodied in children’s habitus, disposing them to certain dietary, physical activity, body formation, and healthy living behaviors. In relation to
adolescent smoking, Frohlich, Potvin, Gauvin and Chabot (2002) find that parental education and parental smoking habits as well as local contextual factors (e.g., the prevalence of anti-smoking signage) influence pre-adolescents’ likelihood of smoking initiation, while Frohlich, Potvin, Chabot, and Corin (2002) find that higher SES neighborhoods tend to be more discouraging of smoking than more disadvantaged communities. In relation to the intergenerational transmission of health-risk behaviors, Wickrama et al. (1999) find that parents' health-risk lifestyles (assessed via smoking, excessive drinking, poor eating, no exercise, and inadequate sleep) are predictive of adolescent health-risk lifestyles, net of familial SES, and that the transmission of health lifestyles is particularly strong along gender lines (e.g., from mothers to girls and from fathers to boys). Together, this research supports the notions that health lifestyles are reproduced across generations and that higher (compared to lower) SES adolescents are more likely exist within health-promoting structures.

Other research by Stead et al. (2011) demonstrates the influential role of peer groups for shaping health lifestyles in adolescence. These authors’ investigation of the emotional, social, and symbolic aspects of food for adolescents in the United Kingdom identified unhealthy eating habits among adolescents as being symbolic of a high social status (i.e., being cool or popular) among peers. While eating unhealthy foods such as chocolate bars, potato chips, and brand-name soda signaled belonging within higher status peer groups, consuming healthy foods was considered socially risky as it signified lower social standing and prompted ridicule and marginalization by higher status peers. This study illustrates the potentially salient role of peers as collectivities in adolescence for shaping health behaviors.
Areas for expansion of health lifestyle theory. Research inquiry into the reproduction of SES- and race-ethnicity-based health inequalities remains a fertile site for empirical testing, particularly with regard to three key areas.

First, while studies have applied health lifestyle theory to identify and promote understanding of inequalities in a variety of health behaviors (e.g., diet, exercise, smoking), there has yet to be any substantial focus on the application of health lifestyle theory to understanding inequalities in the uptake or utilization of health promoting medical interventions (e.g., vaccination, diagnostic screening tools, drug treatments). Thus, whether health lifestyle theory is applicable for understanding group-based participation in these interventions has yet to be determined. The application of health lifestyle theory to understanding SES and racial-ethnic differences in the uptake of health promoting interventions is thus a logical direction for future empirical testing.

Second, though a few studies have sought to apply health lifestyle theory to understand racial-ethnic differences in health behaviors, the reproduction of health lifestyles within racial-ethnic groups remains an under-investigated domain. Additional examination of race-ethnicity in relation to health lifestyles may be particularly important for better understanding the structural facilitators and constraints that shape distinct behavioral patterns among racial-ethnic groups. Hence, application of health lifestyle theory to understanding race-ethnicity-based patterns of health behavior remains a key area for further investigation.

Third, the processes by which families reproduce SES- and race-ethnicity-based health lifestyles across generations is an additional important—yet relatively underexplored—site for theoretical testing and expansion. Specifically, the role of families in shaping health lifestyles in adolescence has yet to receive much theoretically motivated empirical attention. As children
transition into adolescence, the degree of agency that they (versus their parents) possess with regard to their health related behavior remains largely unknown. Consideration of the social structures that shape adolescent health lifestyles and the salience of individual agency at this stage in the life course may help to determine the degree to which adolescents make their own health-related choices and/or are influenced by their families and peers.

**Prosocial Attitudes**

In addition to fundamental cause theory and health lifestyle theory, prosocial attitudes are a third complementary approach that may help to enhance understanding of the mechanisms underlying race-ethnicity and SES-based inequalities in health. As noted in chapter 1, prosocial attitudes become relevant for consideration when participating in health-enhancing behaviors (such as vaccinations, condom use, and blood donation) serves a social good—that is, when the benefits of participation or uptake extend beyond oneself to benefit others (Swap, 1991). While a formal theory concerning the link between prosocial health attitudes and health inequalities has yet to be articulated, a substantial body of sociological and psychological literature suggests that (a) SES and race-ethnicity shape individuals’ prosocial attitudes, and (b) prosocial attitudes, in turn, shape individuals’ willingness to participate in health behaviors that offer a benefit to others. Four related literatures offer important insights for formulating hypotheses regarding SES and racial-ethnic differences in prosocial health attitudes: (1) SES and prosocial behavior, (2) social networks, (3) social capital, and (4) opportunity hoarding.

**SES and prosocial behavior.** First, emerging literature on SES and prosocial behavior suggests, somewhat counter-intuitively, that having fewer resources motivates individuals to act in a more prosocial manner (Piff et al., 2010). A commonsense assumption is that, given that lower SES individuals face restrictions from economic resources, educational opportunities, and
social institutions, and are exposed to greater interpersonal stress (Gallo, Bogart, Vranceanu, & Matthews, 2005; Wilkinson, 1997), this group would prioritize their own self-interest over the welfare of others, and make decisions primarily for their own benefit (Piff et al., 2010). Following this line of reasoning, the costs associated with prosocial behavior—which divert attention away from the self and onto others—should deter lower SES individuals from acting in a prosocial manner (Piff et al., 2010). Contrary to these assumptions, an emerging body of research provides support for an alternative hypothesis: that, despite possessing fewer resources, lower SES individuals are more likely to engage in prosocial behavior because they have increased concern over the needs and welfare of others, possess more egalitarian values, and are motivated by feelings of compassion (Piff et al., 2010). This hypothesis alludes to the idea that, while higher SES individuals possess sufficient resources to act as a buffer against life’s disturbances, lower SES individuals must rely more on their social bonds and are thus more aware of others in their social environment and more prosocial in their orientation (Granovetter, 1983; Piff et al., 2010; Wright, 2009).

Recent empirical inquiry highlights the propensity for lower SES individuals to act in a more prosocial manner than higher SES individuals do. In a series of laboratory experiments, Piff and colleagues (2010) found lower SES people, relative to their higher SES counterparts, to be more generous, more supporting of charity, more trusting towards strangers, and more helpful to individuals in distress. Despite having fewer material resources and being lower in social rank, lower SES individuals were more likely to help increase the welfare of others, even when doing so was costly for themselves (Piff et al., 2010). Such findings are complementary to those of Kraus, Piff, and Keltner (2009) and Kraus and Keltner (2009), which suggest that lower SES individuals are more sensitive to others in their social environments and more socially engaged
in their relationships. This work also resonates with findings concerning the economics of philanthropy in the United States that reveal that persons with the lowest incomes give the largest percentage of their earnings to charity (Andreoni, 2001).

Collectively, this body of research suggests that, despite having fewer material resources, lower SES individuals may have more positive attitudes toward—and, by extension, be more willing to engage in—prosocial health behaviors. Research has yet to directly address the role of race-ethnicity in relation to prosocial attitudes and behaviors in the United States. However, given that race-ethnicity is strongly associated with resource access and general social marginalization, and has impacts on health comparable to SES (Phelan et al., 2010; Williams & Sternthal, 2010), it can be hypothesized that similar patterns of prosocial attitudes and behaviors among lower SES groups may also be prevalent among racial-ethnic minority groups. The sociological literature reviewed in the following sections provides further evidence to support such claims.

Social networks. Scholarship on social networks offers insights regarding why individuals of more marginalized social positions may be more prosocially oriented, while individuals of higher social positions may hold more individualistic orientations. Persons of lower (versus higher) social position tend to have narrower, more homophilous networks (i.e., consisting of a close-knit group of individuals of similar social position); more informal ties (i.e., ties to relatives and/or local others that are not associated with formal membership within a particular organization); and stronger ties to local community and kin (Blau, 1974; Granovetter, 1983; Horvat, Weininger, & Laureau, 2003; Petev, 2013). In terms of community, these homophilous ties may be shaped by discrimination and residential segregation processes that increase the propensity that persons of a specific SES and/or racial-ethnic minority group will
live within the same neighborhoods or local communities, thereby contributing to the concentration of disadvantage within a community (Williams, 2012). Economic insecurity may force marginalized individuals to rely on network ties and help explain their strong homophilous networks (Granovetter, 1983). The strong informal ties that characterize lower SES and certain racial-ethnic minority group networks have been identified as a fundamental source of instrumental resources such as loans, childcare, and basic needs—illustrating the propensity for prosocial behavior within these groups even when resources are scarce (Dominguez & Watkins, 2003; Horvat et al., 2003; Stack, 1974).

**Social capital.** Literature on social capital may also help to explain prosocial behavior among lower SES and racial-ethnic minority groups, by providing insight into norms regarding sharing and reciprocity that emerge from the informal network relationships described in the previous section. Bourdieu (1984; 1986, p. 248) provided one of the seminal sociological discussions of social capital, defining it as the “aggregate of the actual or potential resources which are linked to possession of a durable network of more or less institutionalized relationships of mutual acquaintance or recognition.”¹ Bourdieu’s definition emphasizes that social capital consists of two parts: (1) the quality and amount of resources that one can draw on, and (2) the social relationships that allow one to utilize these resources to their advantage. Bourdieu (1986) also noted that social capital exchanges are generally characterized by

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¹ Many definitions of social capital exist within and beyond sociology (e.g., see Bourdieu, 1984; Coleman, 1990; Putnam, 2000). For the purpose of this dissertation, I rely exclusively on the conceptualization of social capital provided by Bourdieu (1984), which focuses primarily on the class-based reproduction of social capital across generations. Hence, Bourdieu’s conceptualization may be particularly relevant for understanding SES-based health inequalities that occur in adolescence, which is a central focus of this dissertation. Bourdieu’s conceptualization of social capital has also been highly influential for health lifestyle theory, which is one of the three key theoretical perspectives tested in this dissertation.
unstipulated obligations, indeterminate timelines, and norms of reciprocity, and require some form of motivational force to occur.

Building on Bourdieu’s scholarship, Portes (1998) suggests that motivations to exchange social capital can generally be characterized as one of two forms: consummatory or instrumental. Consummatory motivations are based on a sense of obligation to behave in a particular manner, of which a sense of bounded solidarity—whereby individuals in a common social situation learn to identify with, support, and exhibit altruism toward one another—may be particularly relevant (Portes, 1998). In addition to being applicable to SES groups who may share a sense of common social position (Portes, 1998), bounded solidarity may also be applicable to some racial-ethnic minority groups, who often exist in tight knit communities (Dominguez & Watkins, 2003; Stack, 1974). Instrumental motivations are based on norms of reciprocity and enforceable trust, whereby those who give expect to be compensated in the future (Portes, 1998). As low-SES and some racial-ethnic minority groups may need to rely on their social networks in order to acquire basic resources, instrumental motivations may also be particularly relevant for these groups (Dominguez & Watkins, 2003).

Both consummatory and instrumental motivations for sharing social capital have been observed in studies of the networks of low-SES and racial-ethnic minority mothers. Dominguez and Watkins (2003) and Nelson (2000) identify a strong logic of reciprocity within low-SES networks that weakens when the perceived social position of the giver is higher than that of the recipient. Moreover, within low-SES African American and Latin American networks, Dominguez and Watkins (2003) observed norms of return based on trust and the assurance that those in similar social circumstances are understanding and sympathetic of daily needs. These findings suggest that the tendency of low-SES and some racial-ethnic minorities toward
prosocial behavior may be rooted both in a sensitivity to the needs of community members (consummatory motivations) as well as the expectation that prosocial behaviors will be reciprocated by community members of similar social standing (instrumental motivations).

**Opportunity hoarding.** Fourth and finally, Wright’s (2009) concept of opportunity hoarding complements social network- and social capital-based explanations regarding prosociality among lower status individuals by informing conjectures regarding why individuals of higher social position may be less prosocially oriented and more likely to possess individualistic attitudes. Drawing on the seminal stratification scholarship of Weber (1946), Wright (2009) argues that privileged social position allows individuals to gain economic advantages that causally disadvantage individuals excluded from accessing these positions. In the context of health, social position, including one’s SES and racial-ethnic group, may enable those who are more privileged to access the best health-related care, information, and resources, which negatively affects the health outcomes of more disadvantaged individuals. For example, higher SES communities may have greater access to doctors due to a demand (and ability to pay) for their services, enabling them better access to new medical technologies that are limited in supply and maintaining the flow of the most up-to-date health information within this group (Lutfey & Freese, 2005). Given that more advantaged group members have greater access to the knowledge and financial resources that allow them to take advantage of medical technologies, they are more likely to participate in their uptake (Phelan et al., 2010). Nevertheless, it can be conjectured that they may be more motivated by individualistic than prosocial reasons to do so, since those persons around them are similarly privileged.

Wright’s work on opportunity hoarding among the socially advantaged is complemented by Scrambler’s (2001) “greedy bastards” hypothesis of health inequalities, which posits that the
highest SES individuals are primarily motivated by self-interest. Using data concerning widening inequalities in health, income, and material wealth in the United Kingdom, Scrambler (2001) argues that the capitalist system encourages the elite members of society to seek out increased profits at the expense of the majority of the population, which creates and exacerbates health inequalities.

**Converging literatures.** With regard to prosocial health attitudes, these four intersecting literatures concerning (1) SES differences in prosocial behavior, (2) social networks, (3) social capital, and (4) opportunity hoarding collectively provide substantial explanation for posing three conjectures about social position-based differences in prosocial attitudes, which are interesting areas for future health research.

First, members of lower SES and some racial-ethnic minority groups may be more likely than their higher SES and White counterparts to endorse and engage in prosocial health behaviors—even if they do not have a strong connection to the individuals that their behavior directly benefits.

Second, the homophilous informal network ties that are prevalent within the networks of marginalized individuals and the norms that facilitate exchanges of social capital within these networks increases the likelihood that lower SES and racial-ethnic minority individuals, despite facing a scarcity of resources, will endorse and engage in prosocial health behaviors.

Third, to the extent that lower SES and racial-ethnic minority individuals have experienced resource scarcity and, in general, the negative impacts of opportunity hoarding, they are more likely to sympathize with the position of similar others, and be more prosocially motivated to endorse and engage in health behaviors that positively affect other members of their communities.
Testing Fundamental Cause, Health Lifestyle, and Prosocial Attitude Mechanisms of Health Inequalities via the Case of HPV Vaccination

As a relatively newly introduced medical technology that: (a) involves decision-making by adolescents and their parents (Mathur et al., 2010; McRee et al., 2010), (b) helps protect against cancers that disproportionately affect low-SES and racial-ethnic minority groups (Jeudin et al., 2014), and (c) provides both an individual and social benefit (Hechter et al., 2013; Quadri-Sherriff et al., 2012), HPV vaccination offers an ideal empirical case to examine the mechanisms whereby SES and race-ethnicity shape health outcomes across generations. While theory-informed sociological investigations of vaccination have been relatively rare, the case of HPV vaccination also offers a productive avenue for testing and expanding upon fundamental cause, health lifestyle, and prosocial attitude perspectives concerning the emergence of health inequalities. Specific ways in which each of these theoretical perspectives may be applied to the case of HPV vaccination are discussed below.

**Fundamental cause theory and HPV vaccination.** Although immunization has been proposed as one of the most effective means of reducing health inequalities, for HPV vaccination to reduce existing and future health inequalities, uptake must be both (a) widespread and (b) adopted in an equitable manner (Boyce & Holmes, 2012). Fundamental cause theory elicits concerns about the disproportionate uptake of medical technologies by higher SES and racial-ethnic majority groups and the exclusion of their lower SES and racial-ethnic minority counterparts, which may influence future patterns of disease incidence and mortality. The case of HPV vaccination offers an example of one such recently developed medical technology to test whether health inequalities persist when mechanisms for disease prevention change.
Research suggests that adolescents and their parents have varying levels of knowledge about HPV vaccination as well as face specific socio-cultural and financial barriers to vaccine acceptance and access that may perpetuate future SES and racial-ethnic inequalities in cervical and other (less common) HPV-related cancers (Allen et al., 2010; Barnholtz-Sloan et al., 2009; Polonijo & Carpiano, 2013). However, fundamental cause literature also suggests that, if implemented equally across social groups, the association between social status and the uptake of a medical intervention may be weakened and inequalities in health should decrease (Phelan et al., 2010). Studies have yet to test the impact of policy in relation to fundamental cause theory within the context of the United States or establish whether policy that makes medical interventions equally accessible weakens the relationship between SES and health outcomes.

While legislation varies widely across the United States, since 2006 at least 25 jurisdictions have enacted policy that either supports public education about HPV vaccines, funds vaccinations for girls, or requires that girls be vaccinated—which presumably removes some barriers to HPV vaccination (National Conference of State Legislatures [NCSL], 2018). In light of these policy differences, it can be predicted that any inequalities in HPV vaccination would be smaller in jurisdictions where these policies are in place, versus jurisdictions where no such policies exist. This difference in vaccination policy and strategy between jurisdictions offers a novel natural experiment to test whether such policies facilitate the equal diffusion of HPV vaccines across SES and racial-ethnic groups. As such, between-jurisdiction differences in HPV vaccination policy are one example of how HPV vaccination can be used to test a facet of fundamental cause theory. In addition to this policy evaluation focus, this empirical test is useful for informing fundamental cause theory because it concerns a treatment aimed specifically at a
young adolescent population (a population which little prior fundamental cause theory research has examined) and has the opportunity to prevent the latent onset of disease in adulthood.

**Health lifestyle theory and HPV vaccination.** Like fundamental cause theory, health lifestyle theory would predict that individuals of lower SES and racial-ethnic minority status will be less likely to take full advantage of HPV (and other) vaccines or to incorporate cervical (and other) cancer prevention behaviors into their health lifestyles. From this perspective, higher SES and White individuals should be the first to have knowledge about the HPV vaccine and have fewer structural limitations that would prohibit vaccine uptake, resulting in a positive association between SES, White racial-ethnic status, and vaccine uptake. Health lifestyle theory would also predict that, due to higher SES being indicative of increased cultural capital, health literacy, and existence within health-promoting community contexts, as household SES increases, parents should have a greater likelihood of talking to their children about health-related issues (such as HPV vaccination) and transmitting health-promoting messages across generational lines. Given the high correlation between SES and race-ethnicity, similar associations can be predicted such that White (versus non-White) parents are also disproportionately likely to transmit health-promoting messages across generational lines.

Given that adolescents are embedded in both familial and social networks of peers, two additional predictions based on health lifestyle theory can be made with regard to HPV vaccination. First, even if specific higher-SES and White families do not transmit information about HPV vaccination to their adolescent family members, these adolescents may be disproportionately likely to hear about HPV vaccination from their (similar SES and/or White) peers. Second, higher SES and White adolescents may face fewer structural barriers to obtaining HPV vaccines (e.g., access to providers that administer the vaccine, means to pay for all costs
associated with vaccination either through insurance or out-of-pocket), allowing them greater agency to exercise their choice to be vaccinated when compared to lower SES or racial-ethnic minority adolescents.

Existing HPV vaccination research suggests that adolescents play an active role in the negotiation of vaccine-related decisions (Mathur et al., 2010; McRee et al., 2010), but also reveals that their preferences may be different from their parents (Vietri et al., 2011). Other related research suggests that parents (as opposed to peers) are a particularly influential source of HPV-related information for economically disadvantaged African American youth—yet a profound lack of awareness about HPV vaccination exists among these adolescents (Miller, Wickliffe, Jahnke, Linebarger, & Humiston, 2014). Untangling the nuances of adolescents’ personal agency in their own health-related decision-making at this period of their life, and identifying how such agency may be tied to SES and race-ethnicity is crucial for gaining a deeper understanding of the reproduction of SES- and race-ethnicity-based health lifestyles across generations.

**Prosocial attitudes and HPV vaccination.** Though often viewed as a personal protection against acquiring disease, vaccination against contagious diseases can be conceptualized as a prosocial health behavior (Hershey, Asch, Thumasathit, Mazaros, & Waters, 1994; Pywell, 2000; Wheelock, Thomson, & Sevdalis, 2013). When vaccinated, individuals change the dynamics of disease transmission by conferring protection on unvaccinated individuals through decreased risk of disease exposure (Vietri et al., 2012). When vaccine coverage reaches a sufficiently high level, it is possible to achieve herd immunity, whereby high levels of immunity in the general population mean that even individuals with no direct contact with vaccinated individuals may be protected against disease. Hence, it is possible to eradicate
diseases without vaccinating an entire population. In such situations, vaccines can be a greater benefit to society than they are to just the individual vaccine recipient because they contribute to protecting a community in which one is embedded (Quadri-Sheriff et al., 2012; Vietri et al., 2012).

Previous research suggests that prosocial attitudes are an important factor that underlies vaccination decisions. Early work on the factors motivating vaccine-related decisions by Hershey et al. (1994) identified altruism—a form of prosocial behavior whereby the action is intended to benefit only the recipient but not the actor—as a key motivator of vaccination. Other work by Hisnanick and Coddington (2000) suggests that the concept of altruism is particularly relevant to the vaccine-related decisions that parents make on behalf of their children. Consistent with these earlier findings, inquiry by Vietri et al. (2012) found that when considering vaccination, individuals are sensitive to the amount of good that they can do for others, while Voytek, Jones, and Metzger (2011) found “helping others” to be a significant motivating factor for individuals who agreed to participate in vaccine trials. Similarly, a recent systematic review of studies examining the role of herd immunity in parents’ vaccination decisions found that, when asked, between 30% and 60% of parents viewed community benefit as an important reason to vaccinate their children against an array of different diseases (Quadri-Sherriff et al., 2012). With regard to the specific case of HPV vaccination in adolescent boys, Schuler, DeSousa, and Coyne-Beasley (2014) found that most parents report that their decisions to vaccinate their sons were likely to be influenced by the protective benefit that the vaccine conferred to their sons’ female partners.

In line with previous research on SES- and race-ethnicity-based patterns of sociability, we might expect lower SES and racial-ethnic minority adolescent boys and their parents to possess more prosocial attitudes toward HPV vaccination as a means to reduce disease incidence
among sex partners, girlfriends/wives, and communities. Examining possible SES and racial-ethnic differences in prosocial attitudes toward HPV vaccination may help to unpack the relationship between SES, race-ethnicity, and prosocial health behaviors, and elicit a better understanding of the social attitudes that underlie health decisions.

**Hypotheses**

Having discussed the ways in which fundamental cause, health lifestyle, and prosocial attitude perspectives may be applied to the case of HPV vaccination, I now turn to specific hypotheses on the relationship between SES, race-ethnicity, and inequalities in HPV vaccination. Such hypotheses relate to the three facets of HPV vaccination that I will analyze in chapters 3, 4, and 5, respectively.

**Vaccine mandates and health inequalities: Testing fundamental cause theory.**

Guided by fundamental cause theoretical explanations of health inequalities, in chapter 3, I test hypotheses regarding the impact of jurisdiction-imposed HPV vaccine mandates for adolescent girls on SES- and race-ethnicity-based inequalities in provider recommendations of HPV vaccines and HPV vaccine uptake. Specifically, via a comparison of relative vaccination disparities in jurisdictions with versus without HPV vaccine mandates, I seek to determine whether—as fundamental cause theory predicts (Link, 2008)—mandates requiring HPV vaccination for school attendance support the more equal diffusion of HPV vaccines across SES and racial-ethnic groups.

In 2007, both Virginia and the District of Columbia (DC) mandated HPV vaccination for grade 6 girls for school attendance (Pitts & Tufts, 2013; NCSL, 2018). While adolescents in these jurisdictions must still access the vaccine through a health provider, Medicaid is required to cover the cost of the vaccine series and parents can only opt-out of vaccination for their
daughters after reviewing materials provided by the board of health (Pitts & Tufts, 2013; NCSL, 2018). Without such mandated vaccination for school attendance in the rest of the country, this difference in vaccination policy and strategy offers a natural experiment situation to test whether, consistent with fundamental cause-based predictions, predicted SES- and race-ethnicity-based inequalities in HPV vaccination are reduced when significant barriers to knowledge and access are removed. In light of this, I test three hypotheses regarding the potential impact of state-mandated vaccination for shaping provider recommendations of HPV vaccines and adolescent HPV vaccine uptake:

Relative to the pre-mandate period, in jurisdictions with mandates (versus those without), there will exist smaller observed SES- and race-ethnicity-based inequalities in:

Hypothesis 1a. receipt of a health provider recommendation to vaccinate against HPV,
Hypothesis 1b. initiation of the HPV vaccine series, and
Hypothesis 1c. completion of the HPV vaccine series.

Mother–daughter communication and vaccination: Testing health lifestyle theory.

Grounded in health lifestyle theory, in chapter 4, I investigate SES- and race-ethnicity-based differences in vaccine decision-making within the family context. Specifically, I focus on four issues concerning mother–daughter communication. First, I examine whether SES- and/or race-ethnicity-based inequalities exist in a mother’s likelihood of talking with her daughter about tetanus, meningitis, and HPV vaccines. Second, I consider whether mothers’ talking with their daughters about the tetanus, meningitis, and HPV vaccines has an impact on vaccine uptake.

Given that these three vaccines are all recommended for 11- and 12-year-olds, but HPV is

In 2015, after the initiation of this dissertation, Rhode Island became the third US jurisdiction to mandate HPV vaccination for school attendance. I focus only on the Virginia and DC mandates, as sufficient data to assess the impact of the Rhode Island mandate are not yet available.
sexually transmitted, tetanus is most often the result of environmental contamination, and meningitis is usually contracted via contact with a human carrier of the disease, comparison of these three vaccines allows me to consider whether SES- and race-ethnicity-based patterns of communication and uptake are unique to the case of HPV, or whether they are generalizable to other vaccines (and, potentially, other medical treatments). Third, I investigate whether SES- and/or race-ethnicity-based differences exist with regard to a daughter’s initiation of conversation with her mother about HPV vaccination. Fourth, I assess whether a daughter’s initiation of conversation about HPV vaccination with her mother has an impact on vaccine uptake. In relation these areas of examination, the following hypotheses are be tested:

Hypothesis 2a. Higher SES and White mothers will be more likely to have discussions with their daughters about tetanus, meningitis, and HPV vaccines, when compared to lower SES, Black, and Hispanic mothers.

Hypothesis 2b. Daughters whose mothers communicate with them about tetanus, meningitis, and HPV vaccines will be more likely to be vaccinated, when compared to daughters whose mothers did not communicate with them about the vaccines.

Hypothesis 2c. Higher SES and White adolescent girls will be more likely to initiate conversation about HPV vaccination with their mothers, when compared to lower SES, Black, and Hispanic adolescent girls.

Hypothesis 2d. Adolescent girls who initiate conversation with their mothers about HPV vaccination will have higher rates of HPV vaccine uptake compared to adolescent girls who did not initiate such conversations.

**SES and racial-ethnic differences in willingness to vaccinate: The role of prosocial attitudes.** Informed by literature on prosocial attitudes and behaviors, in chapter 5 I investigate
hypotheses related to SES- and race-ethnicity-based differences in attitudes toward HPV vaccination as a method of collective disease prevention. Specifically, I examine whether SES- and race-ethnicity-based differences exist in both adolescent boys’ and their parents’ attitudes toward HPV vaccination as a means to: (a) prevent future girlfriends, spouses, and/or sex partners from getting genital warts and some cancers, and (b) prevent the spread of genital warts and some cancers within their community. For comparison, I also consider SES and racial-ethnic differences in attitudes toward HPV vaccination as a means to prevent genital warts and some cancers at the personal level (i.e., in the adolescent recipient of the vaccine). In light of the literature on prosocial attitudes, it can be expected that lower SES and racial-ethnic minority individuals will be more prosocially oriented with regard to vaccine uptake. Accordingly, the following five hypotheses are tested:

**Hypothesis 3a.** Lower (versus higher) SES and Black and Hispanic (versus White) parents will place greater importance on HPV vaccination to prevent genital warts and cancers in their sons’ (a) future sex partners, (b) future girlfriends/wives, and (c) the community.

**Hypothesis 3b.** Lower (versus higher) SES and Black and Hispanic (versus White) adolescent boys will place greater importance on HPV vaccination to prevent genital warts and cancers in (a) their future girlfriends/wives and (b) the community.

**Hypothesis 3c.** Among parents, possessing more positive prosocial attitudes toward HPV vaccination will be positively associated with willingness to vaccinate one’s son against HPV.
Hypothesis 3d. Among adolescent boys, possessing more positive prosocial attitudes toward HPV vaccination will be positively associated with willingness to be vaccinated against HPV.

Hypothesis 3e. Prosocial attitudes will serve as an indirect pathway between SES and race-ethnicity and willingness to vaccinate.
Chapter 3: A Fundamental Cause Approach to Testing the Impact of Policy on Health Inequalities

In this chapter, I examine the impact of jurisdiction-imposed mandates requiring vaccination for school attendance on the diffusion of HPV vaccination across SES and racial-ethnic groups. As described in the previous chapter, the analyses in this chapter test an important conjecture of fundamental cause theory: that health inequalities will be reduced when policies are implemented that distribute health enhancing knowledge, resources, and/or interventions equally across populations (Link, 2008).

Through applying fundamental cause theory to the case of HPV vaccine mandates, I predict that the implementation of school-based vaccine mandates in DC and Virginia will be associated with smaller observed SES- and race-ethnicity-based inequalities in:

1a. health provider recommendation of HPV vaccines,

1b. HPV vaccine series initiation, and

1c. HPV vaccine series completion.

I test these hypotheses by leveraging multiple years of data collected by the National Immunization Survey–Teen (NIS–Teen), the most comprehensive source of data for analyzing patterns of SES and racial-ethnic differences in vaccination outcomes within and across jurisdictions in the United States.

I begin this chapter by outlining the methods and analytic strategy used to test my hypotheses. I then present the findings of my analyses, in relation to my hypotheses. Finally, I conclude with a discussion of the theoretical and practical implications of my findings and the strengths and limitations of my analyses.
Methods

**Survey data.** This analysis uses data from the 2008, 2009, 2011, 2012, and 2013 waves of the NIS–Teen. The NIS–Teen is a nationally representative US survey administered annually (since 2006) by the National Center for Immunization and Respiratory Diseases (NCIRD) and the NCHS of the CDC. This repeated cross-sectional survey aims to identify vaccination coverage among adolescents and groups at risk of vaccine-preventable diseases (CDC, NCIRD, & NCHS, 2014).

The NIS–Teen was administered via a random-digit-dialing household telephone survey to identify households with adolescent’s aged 13 to 17 (CDC et al., 2014). Given the increasing number of cell-phone-only households (approximately 47% of children lived in cell-phone-only households in 2014; Blumberg & Luke, 2014), in 2011 the NIS–Teen introduced a dual-frame sampling approach and collected interviews from both landline and cell-phone-only households (CDC et al., 2014). The response rate for the landline-based sample ranged from 51.1% to 58.7% across the five years of interest, while the response rate for the cell-phone-based sample ranged from 22.4% to 23.6% in the three years following its introduction to the survey (CDC et al., 2014).

The NIS–Teen interviewed the parent most knowledgeable about their adolescent’s vaccination history, after which (with parental consent) the adolescent’s immunization provider was contacted in an effort to verify responses (CDC et al., 2014). Due to the limited number of cases with healthcare provider-verified data, I limited my analyses to data collected from parents in the household survey. Parent recall has been found to be a valid measure of HPV vaccine uptake (Dorrell, Jain, & Yankey, 2011). A study of parent-reported vaccination using NIS–Teen 2008 data found substantial to near perfect agreement between parent recall and provider
reported data for HPV vaccine series initiation and completion (kappa = .74 to kappa = .92; Dorrell et al., 2011). While the wide publicity that the HPV vaccines have received may make them easier to recall than other vaccines administered in adolescence, the sexually transmitted nature of HPV may have made conversations with healthcare providers about the vaccine particularly memorable (Dorrell et al., 2011).

**Analytic sample to evaluate the policy change.** The NIS–Teen first collected data specific to HPV vaccination in 2008, one year after the Advisory Committee on Immunization Practices recommended the vaccine as part of the immunization schedule for adolescent girls (Jain, Singleton, Montgomery, & Skalland, 2009). While Virginia and DC passed mandates to require the HPV vaccine for grade six girls to enter school in April 2007, the mandates did not come into effect until October 2008, which further delayed their initiation until the start of the 2009 school year (i.e., August or September 2009; see Virginia Code, 2007). As the annual NIS–Teen data collection began in January (eight to nine months before the start of the school year) and was administered to parents of teens aged 13 and older (typically in grade eight or above), the direct impact of the HPV vaccine mandates would first be observable among 13- and 14-year-old girls in the 2011 survey wave.

I made three important considerations when determining the final analytic sample. First, given that vaccine mandates would have (as of 2011) primarily affected 13- and 14-year-old girls, I limited my analyses to this group. Second, given documented regional variation in physicians’ HPV vaccination practices (Daley et al., 2010), I further restricted my sample to adolescents living in the South Atlantic census region. This census region consists of Virginia and DC, where HPV vaccination is required for school attendance, as well as Delaware, Florida, Georgia, Maryland, North Carolina, South Carolina, and West Virginia, where similar mandates
were not in place during the time period of interest. Third, due to the relatively small number of 13- and 14-year-old girls sampled from the two jurisdictions with HPV vaccination mandates (between \( n = 150 \) and \( n = 242 \) across survey years), I pooled the yearly data from (a) 2008 and 2009 (i.e., the period before the mandates would have affected my sample), and (b) 2011, 2012, and 2013 (i.e., the period after the mandates would have affected my sample). Data from 2010 were excluded because some (but not all) adolescents in my sample would have been affected by the policy change in that year. To be sure that specific survey years were not driving my pooled period results, I conducted supplementary analyses using disaggregated years.

My final analytic sample consisted of 4,539 girls for which complete data was available for all variables of interest (87% of the total sample). All missing data were due to non-response in three survey variables: income, provider recommendation, and vaccine uptake. Each of these variables had 5% missingness. Data were more likely to be missing from low-income and non-White respondents, as well as from respondents who were surveyed in post-mandate period. I attempted multiple imputation by chained equations to impute missing data as an alternative to the complete case analysis presented in this chapter, however the imputation models did not converge when they included the categorical interaction variables necessary for evaluating the policy change.

**Study variables.** Table 3.1 details the descriptive statistics for all dependent, independent, and control variables, by jurisdiction and period. Health professional recommendation, vaccine series initiation, and vaccine series completion were the three dependent variables of interest.

**Health provider recommendation.** Health provider recommendation was measured as a binary variable (coded no = 0; yes = 1), based on a single question asking respondents whether a
Vaccine series initiation and completion. Vaccine uptake was measured in the household survey by asking the respondent to report the number of times the adolescent had received HPV shots—as recorded on the adolescent’s immunization shot card and recalled from memory. The NIS–Teen created a summary count variable based on these two sources of information. From this summary variable, I created two binary variables: (1) “vaccine series initiation,” for teens who received at least one shot (zero injections = 0; one or more injections = 1) and (2) “vaccine series completion,” for teens who received three shots (coded zero, one, or two injections = 0; three injections = 1)—which corresponds with the routinely recommended number of shots across all survey years analyzed.

Independent variables. The independent variables included jurisdiction, period, SES, and race-ethnicity. The binary variable jurisdiction was based on the respondent’s regional jurisdiction of residence and was created to compare South Atlantic states where HPV vaccination was not mandated for school attendance (Delaware, Florida, Georgia, Maryland, North Carolina, South Carolina, and West Virginia; hereafter “non-mandated jurisdiction”; coded as 0), with DC and Virginia (hereafter “mandated jurisdiction”; coded as 1), where HPV vaccines were mandated for school attendance.

A variable for period was created based on the year the survey was conducted. This variable was coded dichotomously in order to compare combined 2008 and 2009 survey years (i.e., the period before the sample living in mandated jurisdictions would have been affected by the mandates; coded as 0) with combined 2011, 2012, and 2013 survey years (i.e., the period
after the sample living in mandated jurisdictions would have been affected by the mandates; coded as 1).

SES was measured using household income and mother’s education, modeled as separate variables. Income was based on the poverty threshold for the given year and recorded categorically as: “above poverty > $75,000” (hereafter referred to as “high income”, the referent group), “above poverty but ≤ $75,000” (hereafter “middle income”), and “below poverty” (hereafter “low income”). This categorical income variable was chosen over an alternative continuous measure of income available in the NIS–Teen because (a) the categorical measure had less missing data, allowing me to run analyses on a larger sample and (b) preliminary analyses using a continuous income variable did not yield significant results. Mother’s education was recorded categorically as “less than high school”, “high school”, “some college”, and “college/university degree”. Due to the small number of cases that had not completed high school in mandated jurisdictions (n = 37 in the pre-mandate period; n = 48 in the post-mandate period), I recoded mother’s education using three categories: college/university degree (the referent category; hereafter referred to as “college degree”), some college, and high school degree or less (hereafter, “high school”).

Race-ethnicity was recorded in the NIS–Teen as the race-ethnicity (“non-Hispanic White”, “non-Hispanic Black”, “Hispanic”, or “other/mixed race-ethnicity”) of the adolescent that the responding parent was answering on behalf of. Due to the small number of sampled Hispanic (n = 33 in the pre-mandate period and n = 44 in the post-mandate period) and other/mixed race-ethnicity teens (n = 35 in the pre-mandate period and n = 49 in the post-mandate period) within mandated jurisdictions, I combined these groups to create a three-category race-ethnicity variable coded as: non-Hispanic White (the referent group; hereafter
referred to as “White”), non-Hispanic Black (hereafter “Black”), and Hispanic or other/mixed race-ethnicity (hereafter “other race-ethnicity”). Given the other race-ethnicity sample was still relatively small in mandate jurisdictions (even after merging the Hispanic and other/mixed race-ethnicity groups; \( n = 68 \) in the pre-mandate period and \( n = 93 \) in the post-mandate period), I considered combining the Black and other race-ethnicity categories to create a binary “White” versus “other” race-ethnicity variable. I ultimately retained three distinct racial-ethnic categories because recent US surveillance data indicate Black and Hispanic adolescent girls experience differing inequalities in vaccine completion (i.e., Black [versus White] adolescents are less likely to complete the vaccine series, while Hispanic [versus White] adolescents are more likely to complete the vaccine series; Reagan-Steiner et al., 2016).

**Control variables.** Demographic variables relevant to HPV vaccine recommendation and uptake were controlled for using a series of dummy variables. These included: *mother’s age* (coded under 45 years old = 0; 45 years old and older = 1), *mother’s marital status* (coded married = 0; single/divorced/separated/widowed = 1), *number of children in the household* (coded two or more = 0; only one = 1), and the *respondent’s relationship to the teen* that they were answering on behalf of (coded mother or female guardian = 0; other guardian = 1).

**Statistical analyses.** I used a difference-in-difference-in-difference or triple difference approach (see Berck & Villas-Boas, 2016; Raifman, Moscoe, & Austin, 2018) to evaluate the impact of HPV vaccine mandates on social inequalities in provider recommendation and vaccine uptake. This triple-difference approach was ideal for testing my hypotheses because it allowed me to examine SES- and race-ethnicity-based inequalities in outcomes across jurisdictions and time periods, while (a) controlling for any omitted factors that may have influenced HPV vaccination differently for adolescents in mandated and non-mandated jurisdictions that were
constant across time and (b) removing any omitted factors that influenced HPV vaccination differently across time periods for individuals in both mandated and non-mandated jurisdictions. In applying this approach, I specified a series of linear probability models (LPMs) and then computed and graphed marginal effects to interpret the models’ interaction terms (discussed in the model specifications below). The LPMs used with this approach have been shown to be an acceptable—and easier to interpret—alternative to logistic regression models for analyzing binary outcomes (Mood, 2010). LPMs were well suited for this context because (a) I was primarily interested in marginal effects (specifically, whether outcomes changed for different groups after the policy change) rather than the non-linearity of any relation, and (b) preliminary analyses conducted using logistic regression models yielded nearly identical findings (results not shown; Mood, 2010).

Below, I detail the procedures used to investigate my hypotheses pertaining to provider recommendation and vaccine uptake. Due to the complex sampling design of the NIS–Teen, all of the described analyses were conducted using Stata/SE 13’s survey design (svyset) feature (StataCorp, 2013)—based on published guidelines regarding sampling weights and variance estimation for analyses within and across years (see CDC et al., 2014). For all analyses, p values < .05 were considered statistically significant.

**Procedures for analyzing inequalities in provider recommendation.** Analyses for examining the impact of HPV vaccine mandates on social inequalities in provider recommendation involved three steps. First, I examined overall inequalities in provider recommendation by specifying a model that regressed recommendation on SES, race-ethnicity, and all control variables.
Second, I used three triple-difference models (see Berck & Villas-Boas, 2016; Raifman, Moscoe, & Austin, 2018) to assess the impact of the policy change on SES- and race-ethnicity-based inequalities in provider recommendation. In the first triple-difference model, I analyzed the effect of the mandates on income-based inequalities by regressing provider recommendation on (a) the three-way interaction term \( \text{income} \times \text{jurisdiction} \times \text{period} \), (b) two-way interactions for \( \text{income} \times \text{jurisdiction} \), \( \text{income} \times \text{period} \), and \( \text{jurisdiction} \times \text{period} \), and (c) main effects for \( \text{income} \), \( \text{jurisdiction} \), and \( \text{period} \), as well as education, race-ethnicity, and all control variables. In this model, the coefficients for low- and middle-income groups derived from the three-way interaction represent the effects for adolescent girls in the sample who were subject to mandated HPV vaccination in the post-mandate period and therefore whose outcomes capture the impact of the policy change (Marcet, 2011). The two-way interaction \( \text{income} \times \text{jurisdiction} \) controls for factors that affect recommendation differently for income groups within mandated jurisdictions and are constant across time periods. \( \text{Income} \times \text{period} \) controls for factors that affect recommendation differently across time for the different income groups, but are constant across jurisdictions. \( \text{Jurisdiction} \times \text{period} \) controls for factors that affect recommendation similarly over time for all individuals in mandated jurisdictions. \( \text{Income}, \text{jurisdiction}, \) and \( \text{period} \) control for factors that, respectively, affect provider recommendation: (a) differently for income groups, regardless of jurisdiction and consistently across time, (b) differently within a mandated-jurisdiction constantly across time periods, and (c) constantly across time periods within a mandated jurisdiction. To evaluate the effect of the mandates on education- and race-ethnicity-based inequalities on provider recommendation I repeated the above triple-difference model—substituting education and then race-ethnicity for income in my two- and three-way interactions—and interpreted the results in a similar manner.
Third, to aid in the interpretation of the three-way interaction results, I used Stata’s margins command (StataCorp, 2013) to calculate and graph marginal effects at representative values, holding all other variables constant at their respective modes (see Williams, 2012). The marginal effects at representative values can be interpreted as the discrete change in the predicted probabilities for the dependent variable (HPV vaccine recommendation, initiation, or completion) for each category of an independent variable (education, income, or race-ethnicity) relative to its referent group—at each time period and within each jurisdiction—for individuals with modal values on all other variables.

**Procedures for analyzing inequalities in vaccine uptake.** The procedures for examining the impact of HPV vaccine mandates on social inequalities in vaccine uptake first entailed repeating the three steps described above, replacing vaccine series initiation and then vaccine series completion as the dependent variables in each model. Then, given that provider recommendation is a well-established predictor of HPV vaccine uptake (Gilkey et al., 2012; Holman et al., 2014), I repeated each uptake model with the addition of provider recommendation as an independent variable. As the overall findings of the models predicting initiation and completion with and without the provider recommendation variable were similar, I present the results of models that include the provider recommendation variable and indicate any differences (in terms of direction or significance) between the two sets of models.

**Results**

The descriptive statistics for all study variables are reported in Table 3.1. Of note, across time periods, the weighted sample of adolescents drawn from vaccine-mandated jurisdictions was of higher SES than the weighted sample drawn from non-mandated jurisdictions: 41% to 47% of adolescents in mandated jurisdictions had mothers with a college degree versus 36% in
non-mandated jurisdictions; 52% to 53% of adolescents in mandated jurisdictions lived in high-income households, versus 34% to 38% in non-mandated jurisdictions. This difference is consistent with the varying demographics of the mandated versus non-mandated jurisdictions at large; both DC and Virginia have higher than average (a) median annual household incomes and (b) levels of college attainment (US Census Bureau, 2016; Ryan & Siebens, 2012). Race-ethnicity was similar in mandated and non-mandated jurisdictions across time, an exception being that a higher percentage of the adolescents living in mandated jurisdictions were White in the post-mandate period (62%) versus in the pre-mandate period (52%).

Examining the absolute levels of health professional recommendation, vaccine series initiation, and vaccine series completion in mandated and non-mandated jurisdictions in the periods before and after the vaccine mandates came into effect, is useful for interpreting the findings of my specific hypothesis tests. In the period before the introduction of vaccine mandates, nearly half of the adolescents living in both non-mandated jurisdictions (50%) and mandated jurisdictions (46%) had received a provider recommendation to vaccinate. In the same time period, 30% had initiated and 16% had completed the vaccine series in non-mandated jurisdictions, while 34% had initiated and 22% had completed the vaccine series in mandated jurisdictions.

In the period following (versus before) the introduction of vaccine mandates, more adolescents had received a provider recommendation in both non-mandated (58%) and mandated jurisdictions (60%). In this same period, vaccine initiation rates increased to 41% and 37% in non-mandated and mandated jurisdictions, respectively, while rates of vaccine series completion rose to 20% in non-mandated jurisdictions and fell to 17% in mandated jurisdictions. Additional analyses (not shown in table) of pooled 2008, 2009, and 2011 data waves also indicate that the
overall level of awareness about HPV vaccination remained stable across years, and that the vast majority of parents in both non-mandated (93%) and mandated (97%) jurisdictions had heard about HPV vaccines.

Table 3.1. Descriptive statistics for study variables by time period and mandate jurisdiction.

<table>
<thead>
<tr>
<th></th>
<th>Total Sample ((N = 4,579))</th>
<th>Pre-Mandate Period ((2008–2009))</th>
<th>Post-Mandate Period ((2011–2013))</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(N) (%^a) (n) (%^a)</td>
<td>(N) (%^a) (n) (%^a)</td>
<td>(N) (%^a) (n) (%^a)</td>
</tr>
<tr>
<td>Dependent Variables</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Received (vs. did not receive) recommendation</td>
<td>2,660 54.71</td>
<td>726 49.61</td>
<td>226 45.86</td>
</tr>
<tr>
<td>Initiated (vs. did not initiate) vaccine series</td>
<td>1,761 36.74</td>
<td>466 30.11</td>
<td>163 34.09</td>
</tr>
<tr>
<td>Completed (vs. did not complete) vaccine series</td>
<td>919 18.62</td>
<td>250 16.18</td>
<td>92 21.51</td>
</tr>
<tr>
<td>Independent Variables</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Income</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High income</td>
<td>2,179 38.13</td>
<td>623 38.40</td>
<td>221 52.79</td>
</tr>
<tr>
<td>Middle income</td>
<td>1,734 41.9</td>
<td>621 46.27</td>
<td>155 37.00</td>
</tr>
<tr>
<td>Low income</td>
<td>666 19.97</td>
<td>173 15.53</td>
<td>66 10.21</td>
</tr>
<tr>
<td>Mother’s education</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>College degree</td>
<td>2,132 37.45</td>
<td>614 36.17</td>
<td>212 41.44</td>
</tr>
<tr>
<td>Some college</td>
<td>1,178 26.41</td>
<td>401 26.48</td>
<td>111 27.40</td>
</tr>
<tr>
<td>High school</td>
<td>1,269 36.15</td>
<td>402 37.35</td>
<td>119 31.17</td>
</tr>
<tr>
<td>Race-ethnicity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>2,863 54.89</td>
<td>963 55.93</td>
<td>211 57.77</td>
</tr>
<tr>
<td>Black</td>
<td>941 24.32</td>
<td>236 25.37</td>
<td>163 25.63</td>
</tr>
<tr>
<td>Other race-ethnicity</td>
<td>775 20.79</td>
<td>218 18.70</td>
<td>68 16.59</td>
</tr>
<tr>
<td>Control Variables</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mother &lt; 45 years old (vs. (\geq 45) years old)</td>
<td>2,695 63.04</td>
<td>890 63.46</td>
<td>237 62.28</td>
</tr>
<tr>
<td>Mother married (vs. other)</td>
<td>3,270 66.44</td>
<td>1,058 71.70</td>
<td>279 72.06</td>
</tr>
<tr>
<td>(\geq 2) children in household (vs. only 1)</td>
<td>3,161 75.11</td>
<td>970 76.07</td>
<td>284 70.38</td>
</tr>
<tr>
<td>Mother respondent (vs. other adult)</td>
<td>3,594 78.93</td>
<td>1,152 81.13</td>
<td>353 81.02</td>
</tr>
</tbody>
</table>

Note: “Weighted percentages.
I hypothesized that there would be smaller observed SES and race-ethnicity-based inequalities in HPV vaccine recommendation, initiation, and completion in jurisdictions with HPV vaccine mandates (versus those without), once these mandates came into effect. Next, I outline the results of my specific hypothesis tests.

**Hypothesis 1a: Health provider recommendation.** Model 1 in Table 3.2 shows the results of the LPM regressing health provider recommendation on income, education, race-ethnicity, and all control variables, without any interaction terms. This model shows that the probability of receiving a health provider recommendation to vaccinate was 7- to 15-percentage-points lower for adolescents who (a) lived in a middle- or low-income (versus high-income) household, (b) had parents who attained no more than high school education (versus a college degree), or (c) were of Black (versus White) race-ethnicity, net of time period and jurisdiction. Another noteworthy finding from this model is that, an adolescent’s probability of receiving a provider recommendation increased by 11 percentage points in the post-mandate (versus pre-mandate) period, net of SES, race-ethnicity, and jurisdiction.
### Table 3.2 Linear probability models for health provider recommendation by SES, race-ethnicity, time period, and mandate jurisdiction.

<table>
<thead>
<tr>
<th></th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
<th>Model 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Post-mandate period</td>
<td>.11*** (.02)</td>
<td>.11** (.03)</td>
<td>.10** (.03)</td>
<td>.15*** (.02)</td>
</tr>
<tr>
<td>Mandated jurisdiction</td>
<td>−.03 (.03)</td>
<td>−.05 (.06)</td>
<td>−.15** (.06)</td>
<td>.00 (.05)</td>
</tr>
<tr>
<td>Middle income</td>
<td>−.08** (.03)</td>
<td>−.09* (.04)</td>
<td>−.08** (.03)</td>
<td>−.08** (.03)</td>
</tr>
<tr>
<td>Low income</td>
<td>−.15*** (.04)</td>
<td>−.08 (.06)</td>
<td>−.15*** (.04)</td>
<td>−.16*** (.04)</td>
</tr>
<tr>
<td>Some college</td>
<td>−.04 (.02)</td>
<td>−.04 (.03)</td>
<td>−.11* (.04)</td>
<td>−.04 (.03)</td>
</tr>
<tr>
<td>High school</td>
<td>−.10*** (.03)</td>
<td>−.10** (.03)</td>
<td>−.11* (.05)</td>
<td>−.10** (.03)</td>
</tr>
<tr>
<td>Black</td>
<td>−.07* (.03)</td>
<td>−.08* (.03)</td>
<td>−.07* (.03)</td>
<td>−.03 (.05)</td>
</tr>
<tr>
<td>Other race-ethnicity</td>
<td>−.04 (.03)</td>
<td>−.04 (.03)</td>
<td>−.04 (.03)</td>
<td>−.04 (.05)</td>
</tr>
</tbody>
</table>

**Post-mandate period**...
- × Mandated jurisdiction: .05 (.07)
- × Middle income: .02 (.05)
- × Low income: −.10 (.07)
- × Some college: .07 (.06)
- × High school: −.04 (.06)
- × Black: −.08 (.06)
- × Other race-ethnicity: −.14* (.07)

**Mandated jurisdiction**...
- × Middle income: .02 (.09)
- × Low income: −.10 (.07)
- × Some college: .16† (.09)
- × High school: .15 (.10)
- × Black: −.15 (.10)
- × Other race-ethnicity: −.14 (.11)

**Post-mandate period × Mandated jurisdiction**...
- × Middle income: −.09 (.13)
- × Low income: .13 (.17)
- × Some college: −.01 (.13)
- × High school: .06 (.14)
- × Black: .24 (.15)
- × Other race-ethnicity: .45* (.15)

**Constant**
- .67*** (.03)
- .66*** (.03)
- .69*** (.03)
- .64*** (.03)


*Note:* N = 4,579. Reported estimates are unstandardized slope coefficients (linearized standard errors). Models adjust for all control variables listed in Table 3.1.

\( ^* p < .10, * p < .05, ** p < .01, *** p < .001. \)

**Income-based inequalities in provider recommendation.** Model 2 in Table 3.2 shows the results of the triple-difference model used to analyze the impact of vaccine mandates on income-based inequalities in provider recommendation. The three-way interactions estimate the impact of the mandates on inequalities for middle- and low-income (versus high-income) groups. Both
three-way interaction terms are non-significant, suggesting vaccine mandates did not significantly improve provider recommendations for middle- and low-income groups. Figure 3.1 shows the marginal effects of this three-way interaction, which indicate that, in mandated jurisdictions, disparities for middle-income (versus high-income) adolescents were not observed, while significant pre-mandate-period disparities for low-income (versus high-income) adolescents (MER = −.19, or a 19-percentage-point lower probability of receipt of a recommendation for low versus high income) were no longer significant once mandates came into effect (MER = −.16). In non-mandated jurisdictions, significant disparities in provider recommendation existed for middle-income (versus high-income) adolescents in the pre-mandate period (MER = −.09), and low-income (versus high-income) adolescents in the post-mandate period (MER = −.18).

<table>
<thead>
<tr>
<th></th>
<th>Pre-Mandate Period</th>
<th>Post-Mandate Period</th>
<th>Pre-Mandate Period</th>
<th>Post-Mandate Period</th>
</tr>
</thead>
<tbody>
<tr>
<td>Middle Income</td>
<td>-.07</td>
<td>-.14</td>
<td>-.09*</td>
<td>-.07</td>
</tr>
<tr>
<td>Low Income</td>
<td>-.18*</td>
<td>-.16</td>
<td>-.08</td>
<td>-.19***</td>
</tr>
</tbody>
</table>

Note: Referent group = high income. * p < .05, ** p < .01, *** p < .001.

Figure 3.1 Marginal effects at representative values and 95% confidence intervals for provider recommendation by income, time period, and mandate jurisdiction.

**Education-based inequalities in provider recommendation.** Model 3 in Table 3.2 shows the results of the triple-difference model assessing education-based inequalities in provider
recommendation. The non-significant coefficients obtained for the three-way interaction in Model 3 suggest that vaccine mandates did not significantly improve provider recommendations for adolescents whose mothers had less than a college degree. The marginal effects, plotted in Figure 3.2, show that statistically significant educational inequalities did not exist in mandated jurisdictions in either time period. Significant educational disparities existed in non-mandated jurisdictions: (a) adolescents whose mothers had some college education (versus a college degree) had an 11-percentage-point lower probability of receipt of a provider recommendation in the pre- (but not post-) mandate period, and (b) adolescents whose parents had high school education had 11- and 15-percentage-point lower probabilities of receipt of a provider recommendation in the pre- and post-mandate periods, respectively.

![Figure 3.2 Marginal effects at representative values and 95% confidence intervals for provider recommendation by education, time period, and mandate jurisdiction.](image)

**Note:** Referent group = college degree.
* p < .05, ** p < .01, *** p < .001.

*Race-ethnicity-based inequalities in provider recommendation.* The results of the triple-difference model that examined the impact of vaccine mandates on race-ethnicity-based inequalities in provider recommendation are presented in Model 4 of Table 3.3. The significant
coefficient for the three-way interaction between *post-mandate period * mandated jurisdiction * other race-ethnicity* indicates that vaccine mandates increased provider recommendations for other race-ethnicity adolescents by 45 percentage points. While non-significant, the coefficient for the three-way interaction that included Black race-ethnicity was large (b = .24). The marginal effects of the three-way interaction term, plotted in Figure 3.3, show that in mandated jurisdictions, Black (versus White) race-ethnicity adolescents were significantly less likely to receive a recommendation in the pre- (but not post- ) mandate period (MER = -.18). In non-mandated jurisdictions, Black and other race-ethnicity (versus White) adolescents had, respectively, 11- and 10-percentage-point lower probabilities of receipt of a provider recommendation in the post-mandate period.

![Figure 3.3](image.png)

*Note:* Referent group = White.

* p < .05, ** p < .01, *** p < .001.

**Hypothesis 1b: Vaccine series initiation.** Table 3.3 shows the results for HPV vaccine series initiation by SES, race-ethnicity, health provider recommendation, time period, and mandate jurisdiction.
Table 3.3 Linear probability models for HPV vaccine initiation by SES, race-ethnicity, time period, and mandate jurisdiction.

<table>
<thead>
<tr>
<th></th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
<th>Model 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Post-mandate period</td>
<td>.04* (.02)</td>
<td>.06* (.03)</td>
<td>.00 (.03)</td>
<td>.03 (.03)b</td>
</tr>
<tr>
<td>Mandated jurisdiction</td>
<td>.02 (.03)</td>
<td>.12* (.05)</td>
<td>.10† (.05)</td>
<td>.05 (.04)</td>
</tr>
<tr>
<td>Middle income</td>
<td>-.00 (.02)</td>
<td>.03 (.04)</td>
<td>-.00 (.02)</td>
<td>-.00 (.02)</td>
</tr>
<tr>
<td>Low income</td>
<td>.12** (.04)a</td>
<td>.05 (.05)</td>
<td>.11* (.04)a</td>
<td>.11** (.04)a</td>
</tr>
<tr>
<td>Some college</td>
<td>.02 (.03)</td>
<td>.02 (.03)</td>
<td>-.01 (.04)</td>
<td>.02 (.03)</td>
</tr>
<tr>
<td>High school</td>
<td>.03 (.03)</td>
<td>.03 (.03)</td>
<td>-.02 (.04)</td>
<td>.03 (.03)</td>
</tr>
<tr>
<td>Black</td>
<td>.01 (.03)</td>
<td>.02 (.03)</td>
<td>.02 (.03)</td>
<td>-.02 (.04)</td>
</tr>
<tr>
<td>Other race-ethnicity</td>
<td>.06* (.03)a</td>
<td>.06* (.03)</td>
<td>.06† (.03)a</td>
<td>.01 (.05)</td>
</tr>
<tr>
<td>Provider recommendation</td>
<td>.43*** (.02)</td>
<td>.43*** (.02)</td>
<td>.43*** (.02)</td>
<td>.43*** (.02)</td>
</tr>
</tbody>
</table>

Post-mandate period...
- × Mandated jurisdiction  
  -21** (.07)  
- × Middle income         
  -.06 (.05)  
- × Low income            
  .09 (.06)  
- × Some college          
  .06 (.05)  
- × High school           
  .10† (.05)  
- × Black                 
  .03 (.06)  
- × Other race-ethnicity  
  .07 (.06)  

Mandated jurisdiction...
- × Middle income         
  -.14† (.08)  
- × Low income            
  -.06 (.11)  
- × Some college          
  -.12 (.08)  
- × High school           
  -.02 (.08)  
- × Black                 
  .15 (.10)  
- × Other race-ethnicity  
  -.03 (.09)  

Post-mandate period × Mandated jurisdiction...
- × Middle income         
  .30** (.11)  
- × Low income            
  .17 (.14)  
- × Some college          
  .16 (.13)  
- × High school           
  .08 (.11)  
- × Black                 
  -.08 (.13)  
- × Other race-ethnicity  
  .03 (.14)  

Constant                  | .03 (.03)     | .02 (.03)     | .06† (.03)    | .04 (.03)     |
\( R^2 \)                  | .21           | .22           | .21           | .21           |

Note: N = 4,579. Reported estimates are linear probabilities (linearized standard errors). Models adjust for all control variables listed in Table 3.1.

aEstimate non-significant when provider recommendation is excluded from the model.
bEstimate significant when provider recommendation is excluded from the model (b = .09; p < .01).
† p < .10, * p < .05, ** p < .01, *** p < .001.

The results of Model 1 (Table 3.3) demonstrate that post-mandate period (versus pre-mandate period; b = .04) was significantly positively associated with HPV vaccine initiation overall. Low income (versus high income; b = .12) and other race-ethnicity (versus White; b =
.06) were also significantly positively associated with HPV vaccine initiation; however, these estimates were non-significant when provider recommendation was excluded from in the model, indicating a suppressor effect. Health provider recommendation was the most powerful predictor of HPV vaccine series initiation, increasing adolescents’ likelihood of initiating vaccination by 43 percentage points (net of SES, race-ethnicity, period, jurisdiction, and all control variables); this finding remained consistent across the triple-difference models (Table 3.3, Models 2–4). I now turn to the triple-difference models used to test my specific hypotheses for vaccine initiation.

**Income-based inequalities vaccine series initiation.** Model 2 in Table 3.3 shows the results of the triple-difference model testing whether vaccine mandates were associated with smaller income-based inequalities in HPV vaccine series initiation. The significant three-way interaction between post-mandate period × mandated jurisdiction × middle income indicates that vaccine mandates were associated with a 30-percentage-point increase in vaccine series initiation for middle-income adolescents. The non-significant estimate for the three-way interaction term for low-income adolescents suggests vaccine mandates did not improve initiation for this group. The marginal effects (see Figure 3.4) show that there were no significant income-based disparities in mandated or non-mandated jurisdictions in the pre-mandate period. Reverse disparities in initiation emerged for low-income (versus high-income) adolescents in both mandated (MER = .24, or a 24-percentage-point greater probability of initiating vaccination) and non-mandated jurisdictions (MER = .14) in the post-mandate period.

When provider recommendation was excluded from the LPM, the results were similar in terms of direction, however (a) the MERs obtained for low-income adolescents in both jurisdictions in the post-mandate period were no longer significant, and (b) the MER for middle-
income adolescents living in mandated jurisdictions in the pre-mandate period became significant (MER = −.15, \( p < .05 \); plots not shown).

![Figure 3.4 Marginal effects at representative values and 95% confidence intervals for HPV vaccine series initiation by income, time period, and mandate jurisdiction.](image)

*Note:* Referent group = high income.  
* * \( p < .05 \), ** \( p < .01 \), *** \( p < .001 \).

**Education-based inequalities vaccine series initiation.** Model 3 in Table 3.3 shows the results of the triple-difference model testing whether vaccine mandates were associated with smaller education-based inequalities in HPV vaccine series initiation. The coefficients for the three-way interaction between post-mandate period \( \times \) mandated jurisdiction \( \times \) education were non-significant for adolescents whose parents had high school or some college education, indicating mandates did not significantly increase HPV vaccine initiation for adolescents whose mothers had less than a college degree. The marginal effects (see Figure 3.5) show the emergence of a reverse-disparity for adolescents with high-school education (versus a college degree) in mandated jurisdictions in the post-mandate period (MER = .13, or a 13-percentage-point greater probability of initiating vaccination for adolescents whose mothers had only a high school degree).
Note: Referent group = college degree.

*p < .05, **p < .01, ***p < .001.

Figure 3.5 Marginal effects at representative values and 95% confidence intervals for HPV vaccine series initiation by education, time period, and mandate jurisdiction.

Race-ethnicity-based inequalities vaccine series initiation. Model 4 in Table 3.3 presents the results of the triple-difference model that examines the impact of vaccine mandates on race-ethnicity-based inequalities vaccine initiation. The non-significant three-way-interaction estimates suggest vaccine mandates did not improve HPV vaccine initiation for either Black or other race-ethnicity adolescents. The marginal effects (see Figure 3.6) show the emergence of a reverse-disparity (MER = .09, or a 9-percentage-point greater probability of initiating vaccination) for other race-ethnicity (versus White) adolescents in non-mandated jurisdictions in the post-mandate period, though this marginal effect was non-significant when provider recommendation was excluded from the model (plot not shown).
Hypothesis 1c: Vaccine series completion. Table 3.4 shows the results for vaccine series completion by SES, race-ethnicity, provider recommendation, time period, and mandate jurisdiction. Model 1 shows the results of the LPM regressing HPV vaccine series completion on income, education, race-ethnicity, health provider recommendation, and all control variables, without any interaction terms. The results show that health provider recommendation (versus no recommendation) increased the probability of an adolescent having completed the HPV vaccine series by 22 percentage points, and this estimate was consistent across the triple-difference models (Models 2–4). Living in a low-income (versus high-income) household also increased the probability of an adolescent having completed the HPV vaccine series ($b = .06$); this estimate was no longer significant when provider recommendation was excluded from the model, indicating a suppressor effect. Being of Black (versus White) race-ethnicity decreased the probability of an adolescent having completed the vaccine series by 9 percentage points, while
mother’s education, jurisdiction, and period were non-significant. Next, I present the results of
the triple-difference analyses.

Table 3.4 Linear probability models for HPV vaccine series completion by SES, race-ethnicity, time period, and mandate jurisdiction.

<table>
<thead>
<tr>
<th></th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
<th>Model 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Post-mandate period</td>
<td>-.00 (.02)</td>
<td>.01 (.03)</td>
<td>.01 (.02)</td>
<td>-.00 (.02)</td>
</tr>
<tr>
<td>Mandated jurisdiction</td>
<td>.02 (.02)</td>
<td><strong>.10 (.05)</strong></td>
<td><strong>.10† (.05)</strong></td>
<td>.04 (.04)</td>
</tr>
<tr>
<td>Middle income</td>
<td>-.00 (.02)</td>
<td>.00 (.03)</td>
<td>-.00 (.02)</td>
<td>-.00 (.02)</td>
</tr>
<tr>
<td>Low income</td>
<td>*<em>.06</em> (.03)†**</td>
<td>.06 (.04)</td>
<td>*<em>.06</em> (.03)†**</td>
<td>*<em>.06</em> (.03)†**</td>
</tr>
<tr>
<td>Some college</td>
<td>-.01 (.02)</td>
<td>-.02 (.02)</td>
<td>-.00 (.03)</td>
<td>-.01 (.02)</td>
</tr>
<tr>
<td>High school or less</td>
<td>.01 (.02)</td>
<td>.01 (.02)</td>
<td>-.00 (.03)</td>
<td>.01 (.02)</td>
</tr>
<tr>
<td>Black</td>
<td><strong>-.09</strong>* (.02)**</td>
<td><strong>-.09</strong>* (.02)**</td>
<td><strong>-.09</strong>* (.02)**</td>
<td><strong>-.08</strong>* (.03)**</td>
</tr>
<tr>
<td>Other race-ethnicity</td>
<td>-.00 (.02)</td>
<td>-.00 (.03)</td>
<td>-.00 (.03)</td>
<td>-.06† (.04)</td>
</tr>
<tr>
<td>Provider recommendation</td>
<td><strong>.22</strong>* (.02)**</td>
<td><strong>.22</strong>* (.02)**</td>
<td><strong>.22</strong>* (.02)**</td>
<td><strong>.22</strong>* (.02)**</td>
</tr>
</tbody>
</table>

*Post-mandate period...*  
× Mandated jurisdiction  
× Middle income  
× Low income  
× Some college  
× High school  
× Black  
× Other race-ethnicity

*Mandated jurisdiction...*  
× Middle income  
× Low income  
× Some college  
× High school  
× Black  
× Other race-ethnicity

*Post-mandate period × Mandated jurisdiction...*  
× Middle income  
× Low income  
× Some college  
× High school  
× Black  
× Other race-ethnicity

Constant  
.07** (.02)  
.06* (.02)  
.07** (.02)  
.07** (.02)

_\bar{R}^2_  
.10  
.10  
.10  
.11


Note: N = 4,579. Reported estimates are linear probabilities (linearized standard errors). Models adjust for all control variables listed in Table 3.1.

*Estimate non-significant when provider recommendation is excluded from the model.
†p <.10, * p < .05, ** p < .01, *** p < .001.
**Income-based inequalities in vaccine series completion.** Model 2 (Table 3.4) displays the results of the triple-difference analyses for vaccine series completion by income. The estimates obtained from the three-way interaction were non-significant, indicating vaccine mandates did not improve HPV vaccine series completion for low- or middle-income adolescents. All marginal effects for this three-way interaction were non-significant (plot not shown).

**Education-based inequalities in vaccine series completion.** Model 3 (Table 3.4) displays the results of the triple-difference analyses for vaccine series completion by education. The estimates obtained from the three-way interaction for education were non-significant, suggesting vaccine mandates did not improve HPV vaccine series completion for adolescents whose mothers had less than a college degree. All marginal effects for this interaction were non-significant (plot not shown).

**Race-ethnicity-based inequalities in vaccine series completion.** Model 4 (Table 3.4) shows the results of the triple-difference analyses for vaccine series completion by race-ethnicity. The three-way interaction estimates in Model 4 were non-significant, indicating mandates did not improve HPV vaccine series completion for Black or other race-ethnicity adolescents. The marginal effects (see Figure 3.7) indicate Black (versus White) adolescent girls had an 8- and 10-percentage-point lower probability of completing the HPV vaccine series in non-mandate jurisdictions in the pre- and post-mandate periods, respectively.
Sensitivity analyses. To increase my confidence in my findings, I ran three key sets of sensitivity analyses. First, to determine whether similar conclusions could be made when comparing mandated jurisdictions to the entire United States (as opposed to just South Atlantic census region states), I re-ran all models with an alternative binary jurisdiction variable that compared (a) the vaccine-mandated jurisdictions of Virginia and DC with (b) all other US states. Second, to assess whether the significant findings were being driven by a specific survey year, I re-ran all analyses with the survey year coded as five separate categories (2008 = referent year). Comparing the results of these sensitivity analyses to my original findings, I observed similar patterns of results to those discussed in this chapter. The analyses that modeled each of the five survey years separately, however, were generally too underpowered to detect statistical significance.
Third, given that the relatively small sample of girls living in mandated states ($n = 442$ in the pre-mandate period and $n = 593$ in the post-mandate period) may have left some analyses underpowered to detect significant differences, I re-ran my models using binary SES variables that combined the two smallest (and, consequently, the two lowest) categories of education ($0 =$ “college degree”; $1 =$ “no degree”) and income ($0 =$ “$> $75,000”$, $1 =$ “$\leq $75,000”). The results of these models did not change the findings for race-ethnicity, however they resulted in null findings for some of the significant differences initially observed for SES. This suggests that middle-SES adolescents are distinct from both their low- and high-SES counterparts with regard to HPV vaccination, and thus should be treated as a separate category.

**Discussion**

The implementation of HPV vaccine mandates for school attendance in DC and Virginia allowed for a natural experiment to (a) examine whether these policies facilitated the equal diffusion of HPV vaccination across SES and racial-ethnic groups (b) test the fundamental cause-based hypotheses that SES and racial-ethnic inequalities will be smaller when policy supports the equal implementation of interventions across populations. From a fundamental cause perspective, more advantaged persons should be better able to mobilize their resources to take full advantage of HPV vaccination and thus—in the absence of equally distributed knowledge, technology, and/or other resources—health disparities should emerge. The focus on policy as a buffer between social position and health is an important, yet relatively under-investigated facet of fundamental cause theory, and thus this study makes a novel theoretical contribution to the research literature.

The results of my analyses of five years of US data supported some of my fundamental cause-based hypotheses, however they also yielded some findings that were contradictory to my
predictions. In addition to their theoretical relevance, these findings are useful for evaluating the
effectiveness of existing HPV vaccine mandates and may help to inform future interventions
aimed at increasing the uptake of other vaccines and medical treatments administered in
adolescence. Below, I discuss a number of factors that help explain my findings in relation to
fundamental cause theory and current research on HPV vaccination.

**Vaccine mandates and inequalities in provider recommendation.** Consistent with
previous research identifying health provider recommendation as a significant—and often the
most important—predictor of HPV vaccine uptake (e.g., Guerry at al., 2011; Rosenthal et al.,
2011; Moghtaderi & Adams, 2016), this study identified a strong association between health
professional recommendation and both initiation and completion of the HPV vaccine series. As
health professionals must administer vaccines, providers may play a key role in the creation of
inequalities in HPV vaccination via differential patterns of recommendation to adolescents and
their parents (Polonijo & Carpiano, 2013). From a fundamental cause perspective, mandates
requiring HPV vaccines for school attendance should help to prevent inequalities in provider
recommendation. Hence, I predicted that, relative to the pre-mandate period, in jurisdictions with
mandates (versus those without), there would exist smaller socioeconomic and racial-ethnic
inequalities in the receipt of a health provider recommendation to vaccinate against HPV.

In the pre-mandate period, I observed income- and race-ethnicity-based disparities in
mandated jurisdictions and income- and education-based disparities in non-mandated
jurisdictions. Supporting my predictions, no disparities in provider recommendation were
identified in mandated jurisdictions after mandates came into effect. Additionally, mandates
appeared to improve provider recommendations for other race-ethnicity adolescents and similar
patterns were observed for Black adolescents (in terms of the direction of relationships), though
these estimates did not reach statistical significance. In contrast, I identified the persistence of education- and income-based disparities—as well as the emergence of new race-ethnicity-based disparities—in non-mandated jurisdictions in the post-mandate period.

Overall, my findings for provider recommendation suggest vaccine mandates may have contributed to shrinking pre-existing disparities and preventing the emergence of new disparities in the diffusion of information about HPV vaccines via health providers for lower-SES and marginalized racial-ethnic groups. These findings are consistent with fundamental cause theory’s conjecture that policies that support the equal distribution of innovations across social groups should help to reduce health disparities—least in terms of the more equal diffusion of health-promoting information.

Previous research has found providers’ failure to discuss HPV vaccinations with patients to be an important contributor to non-vaccination—particularly for low-income and racial-ethnic minority adolescents (Guerry et al., 2011; Perkins, Brogley, Adams, & Freund, 2012)—signifying that such inequalities reflect persistent differences in provider behavior. Providers have cited fear of alienating patients based on perceptions that they possess negative vaccine-related attitudes, time constraints associated with discussing the complexities of vaccination against a sexually transmitted infection, and concerns that patients will not follow-up to complete the vaccine series as reasons for not routinely recommending HPV vaccines (Perkins & Clark, 2013). Studies have also found that the strength and quality of healthcare provider’s HPV vaccine recommendations matter: adolescents have higher odds of uptake when providers very strongly (versus not very strongly) recommended to parents that their child be vaccinated (Fu et al., 2017) and parents who initially decline HPV vaccination are more likely to vaccinate their child at a later date (versus not vaccinate at all) when their initial recommendation was of high quality. Though, even when
vaccine mandates are in place, providers report not making strong recommendations to vaccinate due to fears of losing patients from their practice (Carhart, Schminkey, Mitchell, & Keim-Malpass, 2018). In light of my findings and previous research, it is important to acknowledge that the receipt of a provider recommendation does not ensure an adolescent (or parent) will act on this advice. Hence, while mandates may facilitate the equal receipt of provider recommendations to vaccinate among SES and racial-ethnic groups, recommendations alone do not guarantee uptake.

**Vaccine mandates and inequalities in vaccine uptake.** Initiation and completion of the three-shot HPV vaccine series are two key stages of uptake. I predicted that, relative to the pre-mandate period, jurisdictions with mandates (versus those without) would have smaller observed SES and racial-ethnic inequalities in both HPV vaccine series initiation and completion. My findings for HPV vaccine initiation showed mandates were associated with improved vaccine series initiation for middle-income adolescents. Additionally, while neither SES- nor race-ethnicity-based disparities were observed in either jurisdiction in the pre-mandate period, reverse disparities in initiation emerged in the post-mandate period benefitting (1) low-income adolescents, regardless of jurisdiction, (2) adolescents whose parents had high school education in mandated jurisdictions, and (3) other race-ethnicity adolescents in non-mandated jurisdictions.

For HPV vaccine series completion, only racial-ethnic disparities were identified: Black adolescents were less likely to complete the vaccine series than Whites in non-mandated (but not mandated) jurisdictions across time periods.

My findings for vaccine uptake by income, which reveal the absence and/or reverse-disparities favoring lower-income adolescents—regardless of jurisdiction—in the post-mandate period, likely reflect the importance of additional vaccination policy targeting only low-income
adolescents. As noted in chapter 1, the VFC program covers the cost of vaccination for un- and under-insured adolescents across the United States, removing most of the financial burden associated with HPV vaccination, and low-income adolescents are target populations for safety-net health clinics that offer HPV vaccination. In contrast, adolescents with private insurance in non-mandated jurisdictions may need to pay out-of-pocket for some of the costs associated with HPV vaccination and access the vaccine through a family physician (Mirkin & Bach, 2010).

Middle-income adolescents may be in a particularly disadvantaged position for obtaining the HPV vaccine in non-mandated jurisdictions because they may be ineligible for VFC coverage and lack sufficient discretionary funds required for purchase. Mandating vaccination for grade-six girls—and consequently ensuring all health insurers cover HPV vaccines—removes a financial barrier to access, and may explain improved vaccine initiation for middle-income adolescents in mandated jurisdictions. These findings for income provide support for fundamental cause theory’s contention that policy can help weaken the relationship between social position and health.

The finding that mandates improved HPV vaccine initiation for adolescents whose parents had high school education (versus a college degree) also supports my hypotheses—and may speak to the role of education for taking part in—and opting out of—vaccination. Both the DC and Virginia mandates have broad opt-out provisions that allow parents to refuse HPV vaccination on behalf of their children after reviewing educational materials about the vaccine—and do not require a religious, medical, or philosophical justification (Perkins, Lin, Wallington, & Hanchate, 2016; Moghtaderi & Adams, 2016). While vaccine mandates may remove a barrier to vaccination for parents with lower education and improve vaccine initiation in this group, the parents with the highest level education—who may also be the most likely to question vaccines
and refuse them on behalf of their children (Reich, 2014)—may be more likely to exercise the agency necessary to opt-out of mandated HPV vaccination on behalf of their adolescent daughters.

In contrast to the above findings for vaccine initiation by SES, I did not find evidence to support my fundamental cause-based prediction that HPV vaccine mandates shaped racial-ethnic patterns of vaccine initiation. Vaccine mandates also did not appear to influence HPV vaccine series completion by either educational or income markers of SES. Unexpectedly, rates of HPV vaccine uptake (both initiation and completion) were lower in mandated (versus non-mandated jurisdictions) in the post-mandate period, and HPV vaccine series completion declined in mandated jurisdictions once mandates came into effect. This suggests low enforcement of—and adherence to—HPV vaccine mandates, which is surprising given that school mandates have been effective for (a) achieving high levels of uptake for other adolescent and childhood vaccines (i.e., Hepatitis B, varicella; Abrevaya & Mulligan, 2011; Olshen, Mahon, Wang, & Woods, 2007), and (b) preventing socioeconomic and racial-ethnic inequalities in Hepatitis B vaccine uptake in a Chicago sample (Morita, Ramirez, & Trick, 2008). However, these findings are complimentary to two recent analyses of NIS–Teen data that found similar rates of uptake among girls living in jurisdictions with (versus without) mandated vaccination for school attendance (but, did not consider their role for shaping SES or racial-ethnic inequalities; Moss, Reiter, Truong, Rimer, & Brewer, 2016; Perkins et al., 2016) as well as an analysis of clinical care data that concluded Virginia mandates had no effect on the rate of HPV vaccine initiation (Cuff et al., 2016).

Considered in tandem with this previous research, my findings may reflect three key factors unique to the implementation of HPV vaccine mandates, which make these mandates ineffective in increasing uptake overall.
First, given the broad opt-out provisions associated with HPV vaccine mandates, other authors have argued that, in practice, these mandates may function more as a form of education about vaccines for parents and less as a school-entry requirement for adolescent girls (Perkins et al., 2016). Despite being mandated, parents often do not perceive HPV vaccination to be routine or mandatory because HPV cannot be transmitted through casual contact with peers in school settings (Carhart et al., 2018; Perkins et al., 2010). Interestingly, a recent survey of Virginia parents found that (a) less than 1% believed the best time for HPV vaccination was when it was required for school, (b) only 14% believed HPV vaccination was required for their child, and (c) 100% believed their daughters were “up-to-date” on vaccinations—despite the fact that nearly 50% had not received any HPV shots (Cuff et al., 2016). While educational materials are sent to all parents of adolescent girls in mandated jurisdictions, advising them to have their child vaccinated, there is no guarantee that all parents actually read or understand the materials provided (Moghtaderi & Adams, 2016). Given that parents may or may not read or understand the provided educational materials, even their educational impact is questionable. Hence, provider recommendations—which showed strong positive correlations with vaccine uptake and completion—remain influential for encouraging uptake in both mandated and non-mandated jurisdictions.

Second, HPV vaccine mandates were accompanied by lobbying by the vaccines’ pharmaceutical manufacturers (Haber, Malow, & Zimet, 2007) and generated a considerable amount of political debate and controversial news coverage (Gollust et al., 2010), which may have eroded public support for this health policy and led to lower overall rates of vaccine uptake. Much of the public discourse surrounding mandates highlighted the moral aspects of vaccinating girls against a sexually transmitted infection and raised questions about the long-term efficacy
and possible side effects associated with the vaccine itself (Fowler, Gollust, Dempsey, Lantz, & Ubel, 2012). White parents, in particular, also exhibited widespread opposition to vaccine mandates based on the rationale that they infringe on their autonomy to make decisions for their children (Perkins et al., 2010). A qualitative study of parents in Virginia demonstrated that many parents are critical of vaccine mandates and reluctant to adhere to them—preferring to opt-out when children enter grade six and decide for themselves, on their own terms, when and if their child will be vaccinated (Pitts & Tufts, 2013). Corroborating these findings, a recent analysis of NIS–Teen data by Perkins and colleagues (2016) identified higher rates of vaccine series completion in mandated jurisdictions for older girls who would not have been affected by the vaccine mandates versus younger girls for whom the vaccine mandates were in effect (and who constituted my own analytic sample). Excluding older adolescents from the analyses presented in this chapter allowed for a stricter evaluation of the policy change: focusing on the impact of the mandates for adolescents based on geographic locale, rather than on age-based disparities which could potentially be due to cohort effects as opposed to actual policy change.

Third, HPV vaccine mandates specify that adolescent girls should initiate the HPV vaccine series before entering grade 6, but do not include a specific timeline for completion. While enforcing initiation requires the cooperation of (a) individual healthcare providers to distribute HPV vaccines and (b) schools to enforce vaccine mandates, there is no widely used public health surveillance system that tracks adolescents HPV vaccination status or reminds patients to return for follow-up inoculations (Carhart et al., 2018). Moreover, individual healthcare providers in mandated jurisdictions often neglect to remind patients to follow-up after their first HPV shot (Carhart et al., 2018). Hence, enforcing compliance with initiation by grade 6 and encouraging timely vaccine series completion is difficult. In their current form, while
mandates appear to have had a positive effect on reducing some disparities in the pathway between provider recommendation through to vaccine series completion, they may be ineffective for encouraging HPV vaccine completion among grade-six girls, and may even deter some parents from vaccinating their children at the recommended age.

**Implications for future research.** This chapter has relevant implications for medical sociology in terms of understanding the role of policy for facilitating the equal diffusion of new medical interventions across SES and racial-ethnic groups. First, it emphasizes the importance of (a) healthcare providers for encouraging vaccination (and potentially participation in other health-promoting behaviors; e.g., disease screening) among adolescents and (b) policy for mitigating inequalities in provider interactions with adolescent patients of varying social positions—interactions that may shape inequalities in adolescents’ immediate health behaviors and latent health outcomes. Given this was one of the first studies to explicitly test fundamental cause theory in relation to health policy in the United States and that it identified some inequalities in provider recommendation that were mitigated with the introduction of vaccine mandates, future research should build on this line of inquiry by examining how other policies may either mitigate or exacerbate inequalities in provider recommendations of other health-promoting interventions and diagnostic tools.

Second, this chapter identifies a number of disparities—and reverse-disparities—at distinct stages in the diffusion of HPV vaccines, as well as differences in the impact of current policy for shaping the inequalities at various stages of uptake. In doing so, it highlights the unique role that SES and race-ethnicity may play in shaping inequalities at distinct stages in the adoption of new medical technologies, and emphasizes the need for policy to take these complex inequalities into account. Given that few sociological studies have considered how health
inequalities emerge at specific stages in the adoption of new medical technologies/treatments, further examination of how inequalities emerge across stages of uptake would likely be useful for supporting the development of targeted interventions and policies that aim to equally distribute health promoting interventions equally across SES and racial-ethnic groups.

Third, this research contributes to practically evaluating current HPV vaccine mandates and understanding the limitations of these mandates for supporting both the equal and widespread diffusion of HPV vaccines. While current HPV vaccine mandates may have been implemented with the intention of supporting the universal adoption of HPV vaccines, without strict enforcement or widespread public support they may do little to (a) improve overall HPV vaccine uptake or (b) reduce the incidence of HPV-related cancers both across the entire population and within those marginalized SES and racial-ethnic groups that these cancers disproportionately affect. From a policy perspective, policies that enable individuals to either passively “fall through the cracks” or actively opt-out of participation will likely have a limited impact on shaping health inequalities (see Phelan et al., 2010).

Limitations and strengths. The NIS–Teen is ideal for testing my hypotheses, as it provides the most comprehensive data on adolescent vaccination in the United States and includes a number of questions specific to HPV vaccine recommendation and uptake. However, a number of limitations exist when working with this data.

First, this chapter analyzed parent recall data, which is subject to recall bias. Although parent recall is not a perfect source of data, analyses of NIS–Teen data have found (a) parent reported data to be “valid” and “adequate” measures of HPV vaccine uptake at the population level and (b) higher agreement between parent recall and provider verification in 13- and 14-
year-olds (i.e., the sample analyzed in this chapter) versus older teens (Dorrell et al., 2011; Hirth et al., 2016).

Previous studies assessing the validity of NIS–Teen data also suggest that SES and racial-ethnic differences in parent recall of adolescent vaccines exist: low-income and Hispanic parents are more likely to underreport HPV vaccination, while Black parents are more likely to overreport HPV vaccination (Hirth et al., 2016). My analyses, however, revealed the opposite trends: low-income adolescents had higher rates of vaccine initiation and completion, other race-ethnicity adolescents (a large proportion of which were Hispanic) had higher rates of initiation, and Black adolescents had lower rates of vaccine series completion. The fact that HPV vaccines were mandated in certain jurisdictions and not others, could also (a) mean HPV vaccines were more salient and thus easier to recall for parents living in mandated jurisdictions and/or (b) have prompted parents living in mandated jurisdictions to have responded in a manner that was consistent with adherence to the mandates. However, my findings demonstrated (a) similarly high levels of awareness about HPV vaccines in both mandated and non-mandated jurisdictions and (b) lower uptake in mandated states once mandates came effect. Thus, differences in reporting did not appear to be driving my findings, as overreporting should have resulted in biases favoring vaccine knowledge and uptake.

Second, sample size restricted the analyses that were possible. A relatively small subsample of respondents resided in mandated jurisdictions across all study years ($n = 995$), which may have left some models underpowered to detect potentially significant three-way interactions. Small numbers of Hispanic and mixed/other race-ethnicity adolescents resided in jurisdictions with mandated vaccination, which did not allow me to analyze these adolescents as independent racial-ethnic groups. Despite these challenges, the results of my analyses
corroborate with recent research examining the overall impact of HPV vaccine mandates (but did not consider SES or racial-ethnic inequalities in vaccine uptake) that found that state-level HPV vaccination mandates have not increased vaccination rates overall (Bugenske, Stokley, Kennedy, & Dorell, 2012; Moghtaderi & Adams 2016) and increase my confidence in my findings.

Third, my data were also limited in that they were repeated cross-sections (not a panel or cohort design) and cannot adequately predict causation. Moreover, though sampling weights were utilized to compensate for non-response, the overall response rate was less than 60% across survey years. Despite these limitations, this study used the best available nationally representative data on HPV vaccination in the United States that were ideal for testing whether socioeconomic or racial-ethnic inequalities exist in HPV vaccination and identified significant findings that complement previous studies.

This chapter makes novel contributions to research on the role of policy in relation to fundamental cause theory. In the case of HPV vaccine mandates, policy appeared to be particularly beneficial for mitigating racial-ethnic inequalities in provider recommendation and educational inequalities in vaccine initiation—which, may ultimately prevent future social inequalities in the development of HPV-related diseases. While this policy appeared to support the equal diffusion of HPV vaccines among adolescent girls, poor uptake of HPV vaccines in mandated states suggests specific policy weaknesses (including broad mandate opt-out provisions and a lack of attention to vaccine series completion) that ultimately prevent mandates from improving vaccine uptake overall. In chapter 6, I will discuss the broader implications of the findings from this empirical chapter for understanding health inequalities. In the next chapter, I shift attention to social inequalities in mother–daughter communication about vaccination, and
the role that communication may play for shaping the uptake of meningitis, tetanus, and HPV vaccines among adolescent girls.
Chapter 4: Social Inequalities in Vaccine Decision Making

In this chapter, I consider SES- and race-ethnicity-based differences in decision-making about vaccines for adolescent girls. As detailed earlier (in chapter 2), health lifestyle theory motivates this investigation. This theory considers how structure and agency interplay to shape the health-related choices and behaviors of different groups. I apply health lifestyle theory to better understand (a) how structural conditions shape SES- and race-ethnicity-based health lifestyles, and (b) how families may reproduce health lifestyles across generations.

Through applying health lifestyle theory to vaccination decisions, I formulate and test two sets of hypotheses about mother–daughter interactions and uptake. The first set concerns vaccine decision-making, in terms of mother–daughter conversations and uptake:

2a. higher (versus lower) SES and White (versus Black and Hispanic) mothers will be more likely to have conversations with their daughters about meningitis, tetanus, and HPV vaccines; and

2b. mothers who communicate (versus do not communicate) with their daughters about these vaccines will be more likely to have daughters who are vaccinated.

The second set considers the conversation initiator and its implications for uptake:

2c. higher (versus lower) SES and White (versus Black and Hispanic) adolescent girls will be more likely to initiate conversations about HPV vaccination with their mothers; and

2d. adolescent girls who do (versus do not) initiate conversations about HPV vaccination with their mothers will be more likely to be vaccinated against HPV.

I evaluate these hypotheses using a novel US national dataset that contained several questions about mother–daughter communication and adolescent vaccination.
Following the structure of chapter 3, I outline the methods and analytic strategy used to test my hypotheses, present the results of my analyses in relation to my hypotheses, and discuss the theoretical implications of my findings.

**Methods**

**Dataset and sample.** This analysis uses data from the University of North Carolina (UNC) Mother–Daughter Communication Survey. The survey was designed to characterize mother’s communication with their adolescent daughters about HPV vaccination and determine whether HPV-related conversations led to discussions about sexual health and the prevention of sexually transmitted infections (McRee et al., 2012). Participants were a national sample of mothers of adolescent girls aged 11 to 14 years (\(N = 951\), response rate 66%) who completed an English-language survey online in December 2009 (McRee et al., 2012).

Participating mothers belonged to a pre-existing panel of US households maintained by Knowledge Networks, a survey company that recruited a probability sample of participants for the panel using a dual-frame approach (list-assisted, random-digit-dialing, and address-based random sampling; McRee et al., 2012). For households without pre-existing Internet access, the survey company provided a laptop computer and Internet access in exchange for completing multiple surveys each month (McRee et al., 2012). Respondents who completed the survey using existing computer and Internet access received standard incentive points from the survey panel that they could accumulate and redeem for small cash payments (McRee et al., 2012). Evidence suggests that the Knowledge Networks panel is generally free of common biases found in Internet survey methodology and is similar to the US population on several demographic features, including race-ethnicity, education, and income (Baker, Bundorf, Singer, & Wagner, 2003; Dennis et al., 2009).
The demographic backgrounds of participants were similar to non-participating survey panel members, an exception being that participants were more likely to have at least a college degree (33%) compared to non-participants (21%; McRee et al., 2012). Two percent of cases were missing data for at least one of the dependent variables, and 32% of parents reported that they did not know whether their child had received at least one of the three vaccines of interest. I included all 951 participants in my analytic sample, using multiple imputation procedures (described later in this chapter) to impute values for both missing and “don’t know” responses.

**Study variables.** Details of the survey instrument design and pre-testing are reported elsewhere (see McRee et al., 2012) and the survey instrument is available to view online (www.unc.edu/~ntbrewer/hpv.htm). Table 4.1 details the descriptive statistics for all variables. Mother–daughter communication about vaccines, vaccine conversation initiator, and vaccine uptake were the three outcomes of interest.

Mother–daughter communication about meningitis, tetanus, and HPV vaccines. The survey assessed mother–daughter communication separately for meningitis, tetanus, and HPV vaccines. Mothers were asked how much they had talked with [daughter’s name] about: (1) the meningitis shot, (2) the tetanus booster or Tdap shot, and (3) the HPV vaccine. Responses for each question were: “a little”, “a lot”, or “not at all”. I recoded these variables dichotomously (did not talk about the vaccine = 0, talked about the vaccine = 1), given that only a small proportion of participants (< 5% of the total sample) reported speaking with their daughters “a lot” about either meningitis or tetanus vaccines.

HPV vaccine conversation initiator. Mothers who reported having talked to their daughters about HPV vaccination were asked a follow-up question about who initiated the conversation: “When you talked to [daughter’s name] about the HPV vaccine, who first brought
up the topic?” The conversation initiator was recorded categorically as several potential persons, including: the “responding parent”, “non-responding parent”, “daughter”, “healthcare provider”, and “someone else”. A small number of respondents indicated that the “non-responding parent” ($n = 6; < 1\%$) or “someone else” ($n = 31; 3\%$) had initiated conversations about HPV, so I recoded this variable into four categories as: no one (the referent group), a parent (i.e., the responding or non-responding parent), the daughter, and someone else. Although the number of respondents indicating that the “daughter” had initiated the conversation was small ($n = 40$), I retained this category because it was central to my hypotheses.

**Meningitis, tetanus, and HPV vaccine uptake.** Meningitis, tetanus, and HPV vaccine uptake were assessed with three questions asking whether [daughter’s name] had (1) “received a meningitis shot, sometimes called Menactra or Menomune”, (2) “received a tetanus booster, also called Td or Tdap shot”, and (3) “had any shots of the HPV vaccine”. Responses for each question were: “yes”, “no”, and “don’t know”. The number of parents who did not know their child’s vaccination status was not sufficiently large to model this response as a separate category for all three vaccines. Previous analyses using multiple imputation for “don’t know” responses have been shown to produce accurate estimates (see Rubin, Stern, & Vehovar, 1995); I thus recoded the vaccine uptake variables dichotomously ($\text{no} = 0$, $\text{yes} = 1$) using the multiple imputation procedures described later in this chapter to impute predicted values for parents who were unsure of their child’s vaccination status.

**Independent variables.** The independent variables included SES and race-ethnicity. SES was measured using mother’s education and household income, modeled as separate variables. *Mother’s education* was coded categorically in the household survey as “less than high school”, “high school diploma”, “some college/university”, and “college or university degree”. Because
only a small number of respondents had not completed high school \((n = 17; 2\%)\), I recoded this variable using three categories: college or university degree (hereafter referred to as “college degree, the referent category), some college, and high school diploma or less.

*Household income* was measured in the survey using 19 categories, with increments ranging from $4,999 to $24,999 (from less than $5000 to $175,000 or more). I took the midpoint of each income interval to approximate a continuous household income variable. Sensitivity analyses revealed similar results when I modeled income alternatively as four- and six-category scale variables.

*Race-ethnicity* was measured using the race-ethnicity of the responding mother, assessed categorically in the survey as: non-Hispanic White (hereafter, “White,” which is the referent category for all analyses), non-Hispanic Black (hereafter, “Black”), Hispanic, and non-Hispanic other or multiple races-ethnicities (hereafter, “other race-ethnicity”).

**Control variables.** My analyses controlled for several potential confounding factors relevant to vaccine communication and uptake. *Mother’s religiosity* was assessed by asking respondents “How important is religion to you?” and was measured on a four-point likert scale (not important = 1, slightly important = 2, fairly important = 3, very important = 4). I imputed the sample’s mean religiosity score (3.3) for two participants who did not answer this question. *Adolescent’s age* was modeled continuously as the respondent’s daughter’s age in years (range = 11 to 14) on the date that the mother was screened for the survey. I coded *mother’s age* (under 45 years old = 0, 45 years old and older = 1), *mother’s marital status* (married or living with partner = 0, other marital status = 1), *number of children in the household* (one or two kids = 0, three or more kids = 1), and *household urbanicity* (urban = 0, rural = 1) as dummy variables. Lastly, *geographic region of residence*, the US census region in which the respondent resided, was
recorded and modeled categorically as South (the referent category), Midwest, Northeast, or West.

**Multiple imputation procedures.** I used multiple imputation by chained equations (MICE; White, Royston, & Wood, 2011) to impute (a) missing data for the vaccine communication and conversation initiator variables (2%), and (b) vaccination status for the unknown vaccine uptake variables (32%), allowing all analyses to be conducted on a complete (non-missing) dataset \( n = 951 \). Table 4.1 details the number of cases imputed for each dependent variable.

The MICE model used to predict missing values included the imputed dependent variables and all of the (non-imputed) independent and control variables required for my analyses. I applied sampling probability weights provided by Knowledge Networks to the imputation model, to account for the survey design. These weights incorporate post-stratification adjustments using demographic distributions from the most recent data from the Census Bureau’s Current Population Survey, to reduce non-response and non-coverage bias and achieve nationally representative estimates (Dennis et al., 2009). Based on White et al.’s (2011) rule of thumb, which suggests the number of imputations \( M \) should be at least equal to the percentage of incomplete cases in the dataset, I performed 35 imputations. Therefore, all analyses conducted using multiply imputed data were based on 35 imputed datasets.

For all of the analyses described below, I first ran models using a complete case analysis (where \( n = \) the number of complete cases for the variables used in each specific model) and then repeated the models using the imputed data. A comparison of findings for these two sets of analyses yielded similar results across all models—both in terms of the direction and significance of relationships. All results reported in this chapter are based on model estimates
using imputed data.

**Analytic strategy.** I tested all hypotheses using a series of binary and multinomial logistic regression models. Each model described below includes all of the aforementioned control variables. To test hypothesis 2a, whether higher (versus lower) SES and White (versus Black and Hispanic) mothers are more likely to have conversations about meningitis, tetanus, and HPV vaccines with their daughters, I specified three separate binary logistic regression models that respectively regressed (1) meningitis, (2) tetanus, and (3) HPV vaccine communication between mothers and daughters on mother’s education, household income, and race-ethnicity.

To test hypothesis 2b, regarding whether mother–daughter communication about vaccines increases the odds of adolescent vaccine uptake, I specified three separate binary logistic regression models that respectively regressed (1) meningitis, (2) tetanus, and (3) HPV vaccine uptake on mother’s education, household income, and race-ethnicity. I then repeated each of these three vaccine uptake models, adding the respective mother–daughter vaccine communication variable to each model.

To test hypothesis 2c—that higher (versus lower) SES and Black and Hispanic (versus White) girls are more likely to initiate conversations about HPV vaccination with their mothers—I specified a multinomial logistic regression model that regressed the HPV vaccine conversation initiator on mother’s education, household income, and race-ethnicity.

To test hypothesis 2d that adolescents who initiate conversations about HPV with their mothers (versus those who do not) are more likely to initiate HPV vaccination, I specified a binary logistic regression model, regressing HPV vaccine uptake on conversation initiator, mother’s education, household income, and race-ethnicity.
### Table 4.1 Descriptive statistics for study variables.

<table>
<thead>
<tr>
<th></th>
<th>Unweighted n or Mean (Standard Deviation)</th>
<th>Weighted % or Weighted Mean (Linearized Standard Error)</th>
<th>Total n Cases</th>
<th>Imputed</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Dependent Variables</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Talked (vs. did not talk) about...</strong>&lt;sup&gt;a&lt;/sup&gt;</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Meningitis vaccine</td>
<td>325</td>
<td>35.86</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>Tetanus vaccine</td>
<td>480</td>
<td>52.45</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>HPV vaccine</td>
<td>618</td>
<td>62.06</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td><strong>HPV vaccine conversation initiator</strong>&lt;sup&gt;a&lt;/sup&gt;</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No one</td>
<td>337</td>
<td>37.94</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Daughter</td>
<td>40</td>
<td>4.46</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Parent</td>
<td>324</td>
<td>32.58</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Someone else</td>
<td>250</td>
<td>25.02</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Vaccinated (vs. not vaccinated) against...</strong>&lt;sup&gt;a&lt;/sup&gt;</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Meningitis</td>
<td>547</td>
<td>60.08</td>
<td>266</td>
<td></td>
</tr>
<tr>
<td>Tetanus</td>
<td>760</td>
<td>79.12</td>
<td>60</td>
<td></td>
</tr>
<tr>
<td>HPV</td>
<td>277</td>
<td>29.24</td>
<td>24</td>
<td></td>
</tr>
<tr>
<td><strong>Independent Variables</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Education</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>College degree</td>
<td>458</td>
<td>29.27</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Some college</td>
<td>354</td>
<td>36.57</td>
<td></td>
<td></td>
</tr>
<tr>
<td>≤ High school diploma</td>
<td>139</td>
<td>34.17</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Household income</strong></td>
<td>80,608.57 (44,430.38)</td>
<td>66,163.28 (22,393.81)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Race-ethnicity</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>716</td>
<td>62.18</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Black</td>
<td>88</td>
<td>15.69</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hispanic</td>
<td>86</td>
<td>15.63</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other race-ethnicity</td>
<td>61</td>
<td>6.50</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Control Variables</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Religiosity</td>
<td>3.31 (.99)</td>
<td>3.34 (.05)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Daughter’s age</td>
<td>12.53 (1.15)</td>
<td>12.48 (0.06)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mother aged 45 or younger (vs. older than 45)</td>
<td>569</td>
<td>72.88</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mother married or cohabitating (vs. single)</td>
<td>801</td>
<td>80.49</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 or 2 children in household (vs. 3 or more)</td>
<td>624</td>
<td>63.12</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Urban dwelling (vs. rural)</td>
<td>818</td>
<td>82.04</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Region of residence</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>South</td>
<td>290</td>
<td>24.10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Midwest</td>
<td>157</td>
<td>15.16</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Northeast</td>
<td>273</td>
<td>38.14</td>
<td></td>
<td></td>
</tr>
<tr>
<td>West</td>
<td>231</td>
<td>22.59</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


*Note: N = 951 from 35 multiply imputed datasets. aReported values for this variable are based on the mean estimates from 35 imputations.*
I conducted all analyses using Stata/SE 13. I report odds ratios (for logistic regression models), relative risk ratios (for multinomial regression models), and their respective 95% confidence intervals, with \( p \) values < .05 considered statistically significant. The aforementioned probability sampling weights, provided by the Knowledge Networks survey company, were applied to all analyses using the “svyset” feature in Stata (StataCorp, 2013).

**Results**

Examining the absolute levels of meningitis, tetanus, and HPV vaccine communication and uptake as well as the relative proportion of parents, daughters, or others who initiated conversations about HPV vaccination is useful for interpreting my findings (see Table 4.1). Approximately 62% of mothers had conversed with their daughters about HPV vaccination, while a smaller percentage of mothers had spoken with their daughters about tetanus (52%) or meningitis (36%) vaccination.

The HPV vaccine was the most discussed vaccine among mothers and daughters, yet had the lowest level of uptake: 29% of respondents had daughters who had been vaccinated against HPV, while 60% had daughters who had been vaccinated against meningitis and 79% had daughters who were vaccinated against tetanus. Parents initiated the majority of mother–daughter communication about HPV vaccination (33%), while the daughter initiated 4% of conversations, and someone else initiated 25% of conversations. Next, I present the results of my specific hypothesis tests to understand (a) relative differences regarding SES- and race-ethnicity-based inequalities in these three aspects of vaccination, and (b) the role of mother–daughter communication and conversation initiator for shaping vaccine uptake.

**Hypothesis 2a: Communication about meningitis, tetanus, and HPV vaccines.** Table 4.2 shows the results for models regressing mother–daughter communication about (1)
meningitis, (2) tetanus, and (3) HPV vaccines on SES and race-ethnicity. For meningitis and tetanus vaccination, mother–daughter communication differed by race-ethnicity, but not SES. Compared to Whites, Hispanic parents were significantly more likely to have talked about meningitis (odds ratio [OR] = 2.15) and tetanus (OR = 2.12) vaccines with their adolescent girls, while significant differences were not observed for any other racial-ethnic group. For HPV vaccination, neither SES nor racial-ethnic differences were observed in mother–daughter communication.

Table 4.2 Adjusted odds ratios (95% confidence intervals) for binary logistic regression of mother–daughter communication about meningitis, tetanus, and HPV vaccination on SES and race-ethnicity.

<table>
<thead>
<tr>
<th></th>
<th>Meningitis Vaccine Communication</th>
<th>Tetanus Vaccine Communication</th>
<th>HPV Vaccine Communication</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Education</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>College degree</td>
<td>Referent</td>
<td>Referent</td>
<td>Referent</td>
</tr>
<tr>
<td>Some college</td>
<td>.90 (0.60 – 1.34)</td>
<td>.87 (0.59 – 1.29)</td>
<td>.72 (0.47 – 1.09)</td>
</tr>
<tr>
<td>≤ High school diploma</td>
<td>1.07 (0.62 – 1.85)</td>
<td>.85 (0.49 – 1.47)</td>
<td>1.01 (0.56 – 1.83)</td>
</tr>
<tr>
<td>Income</td>
<td>1.00 (1.00 – 1.00)</td>
<td>1.00 (1.00 – 1.00)</td>
<td>1.00 (1.00 – 1.00)</td>
</tr>
<tr>
<td><strong>Race-ethnicity</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>Referent</td>
<td>Referent</td>
<td>Referent</td>
</tr>
<tr>
<td>Black</td>
<td>1.69 (0.82 – 3.47)</td>
<td>.74 (0.37 – 1.46)</td>
<td>.65 (0.30 – 1.43)</td>
</tr>
<tr>
<td>Hispanic</td>
<td>2.15* (1.04 – 4.47)</td>
<td>2.12* (1.06 – 4.25)</td>
<td>.65 (0.32 – 1.31)</td>
</tr>
<tr>
<td>Other race-ethnicity</td>
<td>1.63 (0.70 – 3.79)</td>
<td>.50 (0.23 – 1.11)</td>
<td>.64 (0.30 – 1.35)</td>
</tr>
</tbody>
</table>


**Note:** N = 951 from 35 multiply imputed datasets. Models include all control variables listed in Table 4.1. All estimates are weighted.

* p < .05; ** p < .01; *** p < .001.

**Hypothesis 2b: Uptake of meningitis, tetanus, and HPV vaccines.** Table 4.3 shows the results for models regressing meningitis, tetanus, and HPV vaccine uptake on (1) SES and race-ethnicity, and (2) SES, race-ethnicity, and vaccine communication. Neither SES nor racial-ethnic differences were observed in the uptake of any vaccine in the models that excluded the respective mother–daughter communication variables.
Table 4.3 Adjusted odds ratios (95% confidence intervals) for binary logistic regression of meningitis, tetanus, and HPV vaccine uptake on SES, race-ethnicity, and mother–daughter vaccine communication.

<table>
<thead>
<tr>
<th></th>
<th>Meningitis Vaccine Uptake</th>
<th>Tetanus Vaccine Uptake</th>
<th>HPV Vaccine Uptake</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td><strong>Education</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>College degree</td>
<td>Referent</td>
<td>Referent</td>
<td>Referent</td>
</tr>
<tr>
<td>Some college</td>
<td>1.02</td>
<td>1.04</td>
<td>.69</td>
</tr>
<tr>
<td>(.64 – 1.62)</td>
<td>(.63 – 1.71)</td>
<td>(.39 – 1.25)</td>
<td>(.37 – 1.21)</td>
</tr>
<tr>
<td>≤ High school diploma</td>
<td>1.80</td>
<td>1.86</td>
<td>1.01</td>
</tr>
<tr>
<td>(.92 – 3.54)</td>
<td>(.93 – 3.76)</td>
<td>(.45 – 2.27)</td>
<td>(.43 – 2.31)</td>
</tr>
<tr>
<td>Income</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>(1.00 – 1.00)</td>
<td>(1.00 – 1.00)</td>
<td>(1.00 – 1.00)</td>
<td>(1.00 – 1.00)</td>
</tr>
<tr>
<td><strong>Race-ethnicity</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>Referent</td>
<td>Referent</td>
<td>Referent</td>
</tr>
<tr>
<td>Black</td>
<td>2.28</td>
<td>2.11</td>
<td>2.92</td>
</tr>
<tr>
<td>(.81 – 6.47)</td>
<td>(.68 – 6.57)</td>
<td>(.90 – 9.44)</td>
<td>(1.01 – 10.00)</td>
</tr>
<tr>
<td>Hispanic</td>
<td>1.64</td>
<td>1.35</td>
<td>2.37</td>
</tr>
<tr>
<td>(.66 – 4.10)</td>
<td>(.50 – 3.60)</td>
<td>(.74 – 7.57)</td>
<td>(0.53 – 6.97)</td>
</tr>
<tr>
<td>Other race-ethnicity</td>
<td>.85</td>
<td>.70</td>
<td>.37</td>
</tr>
<tr>
<td>(.28 – 2.65)</td>
<td>(.22 – 2.64)</td>
<td>(.09 – 1.49)</td>
<td>(.10 – 6.97)</td>
</tr>
<tr>
<td><strong>Meningitis vaccine communication</strong></td>
<td>3.95***</td>
<td>2.22 – 7.01</td>
<td></td>
</tr>
<tr>
<td><strong>Tetanus vaccine communication</strong></td>
<td>3.44***</td>
<td>1.88 – 6.28</td>
<td></td>
</tr>
<tr>
<td><strong>HPV vaccine communication</strong></td>
<td>9.15***</td>
<td>(4.63 – 18.05)</td>
<td></td>
</tr>
</tbody>
</table>

*Source: UNC Mother–Daughter Communication Survey, 2009

Note: N = 951 from 35 multiply imputed datasets. Models include all control variables listed in Table 4.1. All estimates are weighted.

* p < .05; ** p < .01; *** p < .001.

After adding the corresponding mother–daughter vaccine communication variable to each model, SES remained non-significant for the uptake of all three vaccines, while differences in uptake by race-ethnicity emerged for tetanus (but not meningitis or HPV) vaccination. For tetanus vaccine uptake, the addition of the mother–daughter communication variable indicated a suppressor effect for Black (versus White) race-ethnicity, whereby the non-significant estimate in Model 1 (OR = 2.92) was of larger magnitude and significant in Model 2 (OR = 3.18).

However, this change should be interpreted with caution due to the estimates being derived from non-linear models and therefore not directly comparable (Mood, 2010). For mother–daughter communication, the Model 2 estimates indicate that having conversed with one’s daughter was
positively associated with the odds of meningitis (OR = 3.95), tetanus (OR = 3.44), and HPV (OR = 9.15) vaccine uptake.

Table 4.4 (a) Relative risks (95% confidence intervals) for multinomial logistic regression of HPV vaccine conversation initiator on SES and race-ethnicity, and (b) adjusted odds (95% confidence intervals) for binary logistic regression of HPV vaccine uptake on SES, race-ethnicity, and HPV vaccine conversation initiator.

<table>
<thead>
<tr>
<th>HPV Vaccine Conversation Initiator</th>
<th>HPV Vaccine Uptake</th>
</tr>
</thead>
<tbody>
<tr>
<td>Education</td>
<td></td>
</tr>
<tr>
<td>College degree</td>
<td>Referent</td>
</tr>
<tr>
<td>Some college</td>
<td>Referent</td>
</tr>
<tr>
<td>≤ High school diploma</td>
<td>Referent</td>
</tr>
<tr>
<td>Income</td>
<td>Referent</td>
</tr>
<tr>
<td>Race-ethnicity</td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>Referent</td>
</tr>
<tr>
<td>Black</td>
<td>.41*</td>
</tr>
<tr>
<td>Hispanic</td>
<td>.53</td>
</tr>
<tr>
<td>Other race-ethnicity</td>
<td>.73</td>
</tr>
<tr>
<td>Conversation initiator</td>
<td></td>
</tr>
<tr>
<td>No one</td>
<td>Referent</td>
</tr>
<tr>
<td>Parent</td>
<td>7.94***</td>
</tr>
<tr>
<td>Daughter</td>
<td>8.01***</td>
</tr>
<tr>
<td>Someone else</td>
<td>10.97***</td>
</tr>
</tbody>
</table>


Note: N = 951 from 35 multiply imputed datasets. Models include all control variables listed in Table 4.1. All estimates are weighted.

*p < .05; **p < .01; ***p < .001.

Hypothesis 2c: HPV vaccine conversation initiator. Results for the multinomial logistic regression of HPV vaccine conversation initiator on SES and race-ethnicity are displayed in Table 4.4. SES was not a significant predictor of whether a (a) parent, (b) adolescent, or (c) someone else (versus no one) initiated mother–daughter conversations about HPV vaccination. For race-ethnicity, Black (versus White) parents had a 59% lower likelihood of having initiated a conversation about HPV vaccines with their daughters. However, no racial-ethnic differences
were observed in having either a daughter or someone else initiate mother–daughter conversation about HPV vaccination.

**Hypothesis 2d: HPV vaccine conversation initiator and uptake.** Results for the binary logistic regression of HPV vaccine uptake on SES, race-ethnicity, and HPV vaccine conversation initiator are also displayed in Table 4.4. No significant SES or racial-ethnic differences existed for HPV vaccine uptake. However, substantially higher odds of HPV vaccine uptake existed for having a parent (OR = 7.94), daughter (OR = 8.01), or someone else (OR = 10.97) initiate a mother–daughter conversation about HPV vaccination, versus not having anyone initiate a conversation.

**Supplementary analyses.** Given the statistically significant estimates/slopes observed for Black race-ethnicity → parent-initiated conversation and parent-initiated conversation → HPV vaccine uptake, I conducted a supplemental analysis to test whether parent-initiated conversation was a significant mediator of the association between Black race-ethnicity and HPV vaccine uptake. As per collective health lifestyle theory, there is a theoretical rationale for considering parent-initiated conversation being a potential mechanism shaping disparities. To test whether this indirect pathway was significantly different from zero, I used Iacobucci’s (2012) z\textsubscript{mediation} test, which is similar to a Sobel test, but enables testing for mediation among categorical variables. The z\textsubscript{mediation} test value was −1.99, indicating that the indirect effect via parent-initiated uptake was statistically significant.

**Discussion**

Drawing on health lifestyle theory, this chapter analyzed SES and racial-ethnic differences in mother–daughter communication about meningitis, tetanus, and HPV vaccines, and considered whether differences in mother–daughter communication and conversation
initiator shaped patterns of vaccine uptake. This focus on mother–daughter communication as a potential pathway between SES and race-ethnicity and the uptake of various vaccines administered in adolescence has received only modest attention in previous research, and makes a novel contribution to the existing literature. My analyses of US national data provided support for some of my hypotheses, yet also revealed findings that were inconsistent with other predictions. These findings are insightful for better understanding the roles of families as collectivities for shaping adolescent health behaviors and reproducing health lifestyles across generations, as well reflecting on the relative agency of parents, adolescents, and others in shaping adolescent health decisions. Next, I discuss my results with regard to the findings of previous research and the literature on health lifestyle theory that motivated my specific hypotheses.

**Mother–daughter communication and vaccine uptake.** My first set of hypotheses focused on (a) whether SES and racial-ethnic inequalities existed in mother–daughter communication about meningitis, tetanus, and HPV vaccines, and (b) whether mother–daughter communication about these vaccines led to vaccine uptake.

**SES and racial-ethnic inequalities in mother–daughter communication.** My findings were inconsistent with my hypotheses that lower (versus higher) SES and Black and Hispanic (versus White) mothers would be less likely to communicate with their daughters about meningitis, tetanus, and HPV vaccines. For SES and Black race-ethnicity parents, there were no observed differences in having conversed with one’s daughter about meningitis, tetanus, or HPV vaccines. However, Hispanics were more likely than Whites to have engaged in mother–daughter conversations about meningitis and tetanus (but not HPV) vaccines. The lack of significant negative associations between SES or Black race-ethnicity and vaccine communication may be
the result of structural forces that facilitate vaccination within these traditionally marginalized groups, which are detailed later in this chapter in relation to the absence of identifiable social inequalities in the uptake of these three vaccines.

The positive association between Hispanic race-ethnicity and communication about tetanus and meningitis vaccines is somewhat perplexing. While the CDC do not collect surveillance data on meningitis incidence by race-ethnicity, incidence rates of tetanus among Hispanics are nearly double that of the non-Hispanic population (Tiwari, 2011). Consequently, one might conjecture that this leads to (a) increased interfacing with health provider’s due to injuries that require tetanus shots to be given, and/or (b) health provider’s perceptions of Hispanics as a high-risk group for vaccine-preventable diseases, and, potentially, a target for provider-initiated conversations about vaccines. Moreover, given that in the 2000s approximately 40% of Hispanics were immigrants (Pew Research Centre, 2017), it is also plausible that (a) the requirement that immigrants be vaccinated against both meningitis and tetanus (but not HPV) before entering the United States, and (b) personal experience with vaccine-preventable diseases in their home countries (Perkins, Pierre-Joseph, Marquez, Iloka, & Clark, 2010) act as cues for mother–daughter conversations about these vaccines among Hispanics. In contrast to meningitis and tetanus, conversations about HPV vaccination may not have been more likely for Hispanics due to a lack of knowledge about this relatively new vaccine (Allen et al., 2010; Burdette, Gordon-Jokinen, & Hill, 2014; Kepka et al., 2012; Molokwu, Fernandez, & Martin, 2014). Given research has shown that Hispanic parents tend to be accepting of HPV vaccination for their daughters once they have acquired knowledge about this vaccine and few believe that HPV vaccination will be misinterpreted by adolescents as permission to engage in sexual activity (Jeudin et al., 2014), it is unlikely that the sexually transmitted nature of HPV would
significantly deter conversations about HPV vaccination between Hispanic mothers and daughters.

_The importance of mother–daughter communication for vaccine uptake._ As hypothesized, daughters whose mothers communicated with them about meningitis, tetanus, and HPV vaccines (versus daughters whose mothers did not communicate with them) were more likely to be vaccinated against each of these diseases. The positive relationship between mother–daughter communication and vaccine uptake across these three vaccines suggests that parents play an active role in shaping the vaccine-related behavior of their children in adolescence. This finding is consistent with earlier investigations of parent–child decision-making about HPV vaccines that found many adolescents were actively involved in the decision-making process with their parents (Mathur et al., 2010; McRee et al., 2010). Research concerning mother–daughter communication about HPV vaccination has found that the majority of conversations focus on the benefits and disadvantages of getting vaccinated (McRee, Reiter, Gottlieb, & Brewer, 2011). Coupled with this research, my findings highlight the importance of the family as a collectivity for sharing norms, values, and knowledge (in this case, medical knowledge) with their children, which may shape adolescents’ worldviews and influence their present and future preferences and behaviors (Cockerham, 2013).

_Conversation initiator and vaccine uptake._ My second set of hypotheses considered (a) whether SES and racial-ethnic inequalities existed in adolescent girls initiation of conversations with their mothers about HPV vaccines, and (b) whether conversation initiator was relevant for determining HPV vaccine uptake.

_SES and racial-ethnic inequalities in conversation initiator._ I hypothesized that lower (versus higher) SES and Black and Hispanic (versus White) adolescent girls would be less likely
to initiate conversations about HPV vaccination with their mothers. While I did not observe the predicted SES or racial-ethnic differences in daughters’ initiation of vaccine-related conversations with their mothers, I did identify a negative association between Black race-ethnicity and parents’ likelihood of initiating conversations about HPV vaccination with their daughters, which mediated the relationship between Black race-ethnicity and HPV vaccine uptake.

The lack of any observed inequalities in adolescent conversation initiation may have been due to the very small percentage (4%) of adolescent girls who had initiated conversations with their mothers about HPV vaccination overall. Conversely, the observed inequalities in HPV vaccine conversation initiation among Black (versus White) parents may reflect (a) differential patterns of information diffusion about HPV vaccines and (b) a general lack of knowledge about HPV vaccines among Black parents, which may limit their ability to initiate conversations with their daughters on this topic. Below, I expand upon the logic for this reasoning.

HPV vaccines only became available in 2007, and are thus presently subject to period effects in terms of the diffusion of vaccine-related information via media coverage, health providers, and other sources (Polonijo & Carpiano, 2013). Black race-ethnicity may be particularly relevant for vaccine-related information diffusion—even more so than SES—due to the concentrated disadvantage that exists within many African American communities (Williams, 2012). While socioeconomically disadvantaged Whites tend to be geographically dispersed across the United States, Blacks are disproportionately concentrated in disadvantaged communities with limited access to social and economic resources, as well as healthcare (Williams, 2012). Given that individuals of higher social position tend to be the first to have knowledge about and adopt beneficial health practices (Cockerham, 2005), it is reasonable to
propose that information about HPV vaccination (as well as other new health promoting technologies) may be relatively slow to diffuse within predominantly Black communities and social networks, creating a structural barrier to parents’ vaccine-related knowledge acquisition and ability to initiate conversations with their daughters.

Previous research has found a (a) lower likelihood of Black parents knowing about HPV vaccines in the years immediately following their approval (Polonijo & Carpiano, 2013; Burdette et al., 2014) and (b) profound lack of awareness about HPV vaccination among low-SES, Black adolescents (Miller et al., 2014). These previous findings provide support for the proposition that the observed racial-ethnic inequalities in mothers’ initiation of conversations with their daughters about HPV vaccination may be attributed to a slow rate of HPV vaccine-related information diffusion among Black parents. This observed racial-ethnic inequality in parental conversation initiation also provides some evidence to support health lifestyle theory’s proposition that health promoting messages are disproportionately transmitted from parents to children within families of more privileged social position and may contribute to the reproduction of health lifestyles across generations (Cockerham, 2014).

*The importance of conversation initiator for vaccine uptake.* I hypothesized that adolescent girls who initiated conversations about HPV vaccination with their mothers (compared to adolescent girls who had not conversed with their mothers) would be more likely to be vaccinated, and found a positive association in support of this prediction. Positive associations between conversation initiator and HPV vaccine uptake were also observed when parents and someone else (versus no one) initiated conversations. The finding that mother–daughter conversations about HPV vaccination increased the likelihood of HPV vaccine uptake—regardless of conversation initiator—suggests that parents, adolescents, and others may
all have an influential role in shaping vaccine uptake at this point of the life course. This finding has three further important implications when considered in conjunction with the statistics describing the proportion of mother–daughter conversations initiated by each of these three groups.

First, the observation that parents were the most likely figures to initiate mother–daughter conversations about HPV vaccines suggests that, while health decision-making may begin to shift from the parent to child in adolescence (Umberson et al., 2000), parents are likely the primary decision makers for influencing adolescent HPV vaccine uptake at the routinely recommended age of 11 or 12. This conjecture is consistent with research by McRee and colleagues (2010) that found parents were primary decision-makers regarding HPV vaccination for their adolescent daughters.

Second, although only a small percentage of daughters had initiated conversations about HPV with their mothers, these daughter-initiated conversations were positively and significantly associated with HPV vaccine uptake. This corroborates with additional findings from the aforementioned study by McRee et al. (2010), which identified adolescents as the primary drivers of HPV vaccine-related decisions within a minority of families, and suggests adolescents can possess a significant degree of personal agency in making health-related decisions and may thus be a useful focus for initiatives aiming to increase vaccine uptake.

Third, the findings that (a) one quarter of mothers in the sample reported having a conversation with their daughter about HPV vaccination that was initiated by someone else outside of the parent–daughter dyad (a group that was comprised largely of healthcare providers; 88%) and (b) conversations initiated by someone else were also significant for predicting adolescent vaccine uptake, suggest that adolescent health decision-making is not only shaped by
one’s family, but also influenced by others—especially healthcare professionals—within in their community. This finding is consistent with a body of adolescent vaccination literature that finds provider recommendation to be one of the strongest and most consistent correlates vaccine uptake (Dorell, Yankey, Kennedy, & Stokley, 2013; Small, Sampselle, Martyn, & Dempsey, 2014; Moss, Reiter, Rimer, & Brewer, 2016). The influential role of these others for shaping adolescent decisions may help to partially explain the lack of SES and racial-ethnic inequalities observed in the uptake of HPV—as well as meningitis and tetanus—vaccines, as well as the tendency for Blacks to have higher odds of tetanus vaccine uptake after controlling for communication about this vaccine. These inequalities in uptake are further discussed below.

**SES and racial-ethnic inequalities in vaccine uptake.** Though I did not specifically hypothesize about racial-ethnic or SES inequalities in vaccine uptake in this chapter, comparisons of (a) racial-ethnic differences observed in parents’ initiation of HPV vaccine-related conversations with (b) patterns of meningitis, tetanus, and HPV vaccine communication and uptake are useful for highlighting the classic interplay between structure and agency that are central to health lifestyle theory and the discipline of sociology overall. While Black parents were less likely to have initiated conversations about HPV vaccination with their daughters, they were not less likely to have conversed with their daughter about HPV vaccination or to have a daughter who had received at least one HPV shot. Moreover, neither marginalized socioeconomic nor racial-ethnic status was associated with a lower likelihood of being vaccinated against meningitis, tetanus, or HPV. Collectively, these findings may reflect both (a) the current structural conditions that facilitate vaccine uptake among disadvantaged or marginalized groups and (b) the individual agency that is required to opt-out of adolescent vaccines in many jurisdictions. These factors are discussed below.
As of 2015, initiation rates for meningitis and tetanus vaccines (among all adolescents), and HPV vaccines (among female adolescents) by the age of 13 (i.e., just after the recommended 11-/12-year-old age of inoculation) were 87%, 79%, and 56%, respectively (Reagan-Steiner et al., 2016). In 2009, at the time that these survey data were collected, tetanus vaccination was mandated as a requirement for school attendance in 45 states and DC, meningitis vaccination was mandated for school attendance in 22 states and DC, and HPV vaccination was mandated only in Virginia and DC (Immunization Action Coalition, 2018). All three vaccines were also included in the VFC program and were accessible in safety-net health clinics that typically are found in underserved areas (Tsui et al., 2013). As Giddens (1984) contended, the social structure is not only a restricting force: the resources afforded by structure can also help individuals realize their choices. In the case of adolescent vaccines, school mandates, VFC coverage, and vaccine availability in safety-net clinics may be examples of such resources afforded by the social structure that help to mitigate knowledge-, financial-, and accessibility-based barriers to vaccine uptake for typically vulnerable populations. With regard to health lifestyle theory, the aforementioned current structural conditions may make vaccination a “realistic choice” (Cockerham, 2005) for most adolescents, by providing disadvantaged social groups some of the resources required to participate in this health-promoting behavior at rates similar to their more advantaged counterparts.

The reverse inequalities identified in tetanus vaccine uptake by race-ethnicity for Blacks—once controlling for mother–daughter communication—may also highlight the role of agency for participating in or opting out of health behaviors. Vaccination is a unique health behavior in that, despite being widely proven to be beneficial for one’s health, it has become a topic of significant media controversy and safety concern for some parents (Reich, 2014). In this
case, the observed reverse disparities in vaccine uptake may reflect the greater likelihood of those of higher social position to have access to anti-vaccination information, question scientific and medical authority, oppose mandated vaccination on the basis that it infringes on their parental autonomy, and ultimately exercise their agency to refuse vaccines on behalf of their children (Reich, 2016; Perkins et al., 2010).

Implications for future research. This chapter has relevant implications for medical sociology in terms of understanding the role of health lifestyles for shaping health inequalities. First, this chapter highlights the important role of parent-child communication, and the potential for parents, adolescents, and others (including health providers) to share responsibility for health decision-making at this point in the life course. Future research on health lifestyles in adolescent populations should thus not only aim to evaluate the preferences and intentions of adolescents in relation to health behaviors, but also consider the congruence between the dispositions of adolescents, their parents, and other influential individuals (including healthcare providers) in the adolescent’s life.

Second, this chapter identifies the existence of a racial-ethnic inequality in parents initiating communication with their daughters about a new medical technology, and emphasizes the role that information diffusion may play in shaping health lifestyles for different racial-ethnic groups. In doing so, it highlights the relevance of race-ethnicity for shaping health lifestyles—an area that has received relatively little attention in health lifestyles research and that will likely prove fruitful for future investigation. It also identifies the important role of others—including health providers—for engaging parents and adolescents in conversations about health-promoting interventions, especially within populations where new health-relevant knowledge may be less likely to circulate.
Third, in identifying null associations and reverse disparities in vaccine-related conversations and uptake, this chapter acknowledges the potential of resources afforded by the social structure for facilitating equitable participation in health promoting interventions. From a policy perspective, structural-level interventions such as the VFC program, safety-net clinics, and school-based vaccination mandates may be beneficial for encouraging vaccination and participation in other health-promoting behaviors among typically marginalized groups, who may otherwise face resource-based barriers to participation. Such interventions may also help to overcome inequalities in the initiation of health-promoting communication within families, which may influence patterns of adolescent health behavior.

**Limitations and strengths.** In this chapter, I analyzed national data from a survey that focused specifically on mother–daughter communication. In doing so, I explicitly addressed several gaps in the research literature including the (a) consideration of racial-ethnic inequalities in the reproduction of health lifestyles across generations, (b) application of health lifestyle theory to help understand the uptake and utilization of health-promoting medical interventions, and (c) identification of the significance of conversation initiator—and relative agency of adolescents—for influencing adolescent health behavior. Nonetheless, some limitations must be noted.

The survey data were cross-sectional, and thus I was unable to establish causal relationships. The survey also relied on mother’s recall of their daughter’s vaccination status (rather than immunization records or provider verified data), which leaves estimates of vaccine uptake subject to both social desirability and recall bias (Dorell, Jain, & Yankey, 2011). Substantial variation in reported uptake across the three vaccines of interest (ranging from 29% to 79%), however, suggests that social desirability was not driving the reported vaccination uptake.
uptake rates. The potential for recall bias is highlighted by the fact that 28% of mothers reported being “not sure” of whether their daughter had received the meningitis vaccine. Nevertheless, the primary focus of this dissertation is on HPV vaccination (a vaccine for which less than 3% of parents reported being “not sure”), and parent recall has been found to be a valid measure of adolescent HPV vaccine uptake in other US national surveys (Dorell et al., 2011). As noted in chapter 3, the wide publicity that the HPV vaccines have received may make them easier to recall than other vaccines administered in adolescence, while the fact that HPV is sexually transmitted makes it a potentially sensitive and memorable topic of conversation for parents (Dorell et al., 2011).

This data contained a number of variables that were ideal for testing my theoretically-motivated hypotheses with regard to SES and racial-ethnic inequalities in vaccine decision-making within the family context. Yet, it did not include variables related to community-level demographics or the content of mother–daughter conversations about vaccines, which may be useful for more fully understanding the observed relationships. In addition, given the survey was only administered in English, the results cannot be generalized to adolescents with non-English-speaking mothers. Consideration of the abovementioned factors as well as analyses that include adolescents whose parents do not speak English (and as a result, who may be exposed to different sources of information about specific vaccines) may be worthwhile directions for future research.

Despite these limitations, this chapter makes a unique contribution to research on vaccine-related communication and uptake. It may also be useful for understanding inequalities in the uptake of other health promoting medical interventions among adolescents, which can have long-term impacts on health and longevity. I will discuss the broader implications of my
findings from this chapter, with respect to understanding the mechanisms underlying health inequalities, in chapter 6. In the next chapter, I turn my attention to the HPV vaccine-related attitudes of adolescent boys and their parents, and consider the potential role of prosocial attitudes for shaping health inequalities.
Chapter 5: Socioeconomic and Racial-Ethnic Inequalities in Prosocial Health Attitudes

In this chapter, I examine SES and racial-ethnic differences in parents’ and adolescent boys’ attitudes toward HPV vaccination as a prosocial method of disease prevention. As outlined in chapter 2, this inquiry is informed by research on prosocial behavior, social networks, social capital, and social stratification. This body of empirical literature suggests that individuals of marginalized social position—including lower SES, Hispanic, and non-White individuals—may possess more prosocial attitudes, and thus be more willing to engage in behaviors that benefit others. I apply these conjectures to the case of HPV vaccination for adolescent boys, testing two sets of related hypotheses.

The first set of hypotheses considers whether SES and race-ethnicity are associated with parents’ and adolescent boys’ attitudes toward HPV vaccination. Specifically, I predict that, compared to their non-Hispanic White and higher SES counterparts:

3a. Black and Hispanic and lower SES parents will place greater importance on HPV vaccination to prevent genital warts and cancers in their sons’ (a) future sex partners, (b) future girlfriends/wives, and (c) the community; and
3b. Black and Hispanic and lower SES adolescent boys will place greater importance on HPV vaccination to prevent genital warts and cancers in (a) their future girlfriends/wives and (b) the community.

The second set of hypotheses considers whether attitudes mediate adolescent boys’ and parents’ willingness to vaccinate themselves or their sons against HPV. I predict:
3c. among parents, possessing more positive prosocial attitudes toward HPV vaccination will be positively associated with willingness to vaccinate one’s son against HPV;

3d. among adolescent boys, possessing more positive prosocial attitudes toward HPV vaccination will be positively associated with willingness to be vaccinated against HPV; and

3e. prosocial attitudes will serve as an indirect pathway between SES and race-ethnicity and willingness to vaccinate.

I use a unique US national dataset that focused on the HPV vaccine-related attitudes of adolescent boys and their parents to test these hypotheses.

Following the structure of the previous two empirical chapters, in this chapter I outline my methodology and analytic strategy, present the results of my analyses, and discuss my findings in relation to the theory that motivated my hypotheses.

Methods

Dataset and sample. I tested my hypotheses using data from the UNC HPV Immunization in Sons Study, which examined attitudes and beliefs about HPV vaccination for adolescent boys (Reiter et al., 2011). Participants were (1) a national US sample of parents with adolescent sons ages 11 to 17 years (response rate = 73% among parents who responded to their e-mail invitation), and (2) adolescent boys ages 11 to 17 whose parents completed the survey (response rate = 56% among those whose parents responded to their e-mail invitation; Reiter et al., 2011). Parents and adolescent boys completed an English-language survey online in August and September 2010, at which point only about 2% of male adolescents had received any doses of the HPV vaccine (Reiter et al., 2010).

Parents belonged to a pre-existing panel of US households maintained by Knowledge
Networks, a survey company that recruited a probability sample of participants for the panel using a dual-frame approach (list-assisted, random-digit dialing with cell-phone only household supplementation, and address-based random sampling; Reiter et al., 2013). For households without pre-existing Internet access, the survey company provided a laptop computer and internet access in exchange for completing multiple surveys each month (Reiter et al., 2013). Participating parents were asked to allow their 11- to 17-year-old son with the most recent birthday to participate in the study, and sons provided assent before beginning the survey. Respondents who completed the survey using existing computer and Internet access received standard incentive points from the survey panel that they could accumulate and redeem for small cash payments. As noted in chapter 4, evidence suggests that the Knowledge Networks panel is generally free of common biases found in Internet survey methodology and is similar to the US population on several demographic features, including race-ethnicity, education, and income (Baker et al., 2003; Dennis et al., 2009).

My analytic sample consisted of 401 adolescent boys and 518 parents of sons who were not yet vaccinated against HPV and had complete data on all variables required for my analyses (97% of valid cases for adolescent boys and parents). The demographic backgrounds of responding adolescent boys and parents were similar, an exception being that adolescents were more likely to be of “other” race-ethnicity and less likely to be non-Hispanic White than parents.

**Measures and variables.** Details of the survey instrument design and pre-testing are reported elsewhere (see Reiter et al., 2011; 2013) and both parent and son survey instruments are available to view online (www.unc.edu/~ntbrewer/hpv.htm). Table 5.1 details the descriptive statistics for all dependent, independent, and control variables.
Table 5.1 Descriptive statistics for study variables.

<table>
<thead>
<tr>
<th></th>
<th>Parents (n = 518)</th>
<th>Adolescent Boys (n = 401)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. (%)</td>
<td>Mean (SD)</td>
</tr>
<tr>
<td><strong>Dependent Variables</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Individualistic attitudes</td>
<td>3.66 (1.29)</td>
<td>3.57 (1.29)</td>
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<tr>
<td>Prosocial girlfriend/wife attitudes</td>
<td>3.64 (1.29)</td>
<td>3.66 (1.26)</td>
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<tr>
<td>Prosocial sex partner attitudes</td>
<td>3.57 (1.27)</td>
<td></td>
</tr>
<tr>
<td>Prosocial community attitudes</td>
<td>3.52 (1.29)</td>
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<tr>
<td>Adolescent willingness to get HPV vaccine</td>
<td>2.97 (1.14)</td>
<td></td>
</tr>
<tr>
<td>Parent willingness to get son HPV vaccine</td>
<td>3.38 (1.21)</td>
<td></td>
</tr>
<tr>
<td><strong>Independent Variables</strong></td>
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<td></td>
</tr>
<tr>
<td>Parent’s education</td>
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<td></td>
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<tr>
<td>College degree+</td>
<td>133 (25.68)</td>
<td>93 (23.19)</td>
</tr>
<tr>
<td>Some college</td>
<td>160 (30.89)</td>
<td>131 (32.67)</td>
</tr>
<tr>
<td>High school diploma</td>
<td>158 (30.50)</td>
<td>127 (31.67)</td>
</tr>
<tr>
<td>&lt; 12 Years</td>
<td>67 (12.93)</td>
<td>50 (12.47)</td>
</tr>
<tr>
<td>Household income</td>
<td>67,457 (42,986)</td>
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<tr>
<td><strong>Respondent’s race-ethnicity</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>355 (68.53)</td>
<td>251 (62.59)</td>
</tr>
<tr>
<td>Black</td>
<td>61 (11.78)</td>
<td>48 (11.97)</td>
</tr>
<tr>
<td>Hispanic</td>
<td>76 (14.67)</td>
<td>61 (15.21)</td>
</tr>
<tr>
<td>Other race-ethnicity</td>
<td>26 (5.02)</td>
<td>41 (10.22)</td>
</tr>
<tr>
<td><strong>Control Variables</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Parent’s religiosity</td>
<td>3.58 (1.35)</td>
<td>3.63 (1.34)</td>
</tr>
<tr>
<td>Adolescent male’s age</td>
<td>14.03 (2.11)</td>
<td>14.04 (2.10)</td>
</tr>
<tr>
<td>Parent aged 44 or younger (vs. 45+)</td>
<td>313 (60.42)</td>
<td></td>
</tr>
<tr>
<td>Parents married or cohabitating (vs. single)</td>
<td>425 (82.05)</td>
<td>334 (83.29)</td>
</tr>
<tr>
<td>Female responding parent (vs. male)</td>
<td>274 (52.90)</td>
<td></td>
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<tr>
<td>1 or 2 children in household (vs. 3 or more)</td>
<td>371 (71.62)</td>
<td>282 (70.32)</td>
</tr>
<tr>
<td>Urban dwelling (vs. rural)</td>
<td>429 (82.82)</td>
<td>330 (82.29)</td>
</tr>
<tr>
<td><strong>Region of residence</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>South</td>
<td>171 (33.01)</td>
<td>139 (34.66)</td>
</tr>
<tr>
<td>Midwest</td>
<td>131 (25.29)</td>
<td>102 (25.44)</td>
</tr>
<tr>
<td>Northeast</td>
<td>109 (21.04)</td>
<td>78 (19.45)</td>
</tr>
<tr>
<td>West</td>
<td>107 (20.66)</td>
<td>82 (20.45)</td>
</tr>
</tbody>
</table>

*Source: UNC HPV Immunization in Sons Study, 2010.*

**Willingness to vaccinate.** Parents’ and adolescent boys’ willingness to vaccinate against HPV were the dependent variables of interest. Adolescent male willingness to be vaccinated was derived from the question: “How willing would you be to get the HPV vaccine?” Parent willingness to vaccinate their sons was derived from the question: “How willing would you be to get the HPV vaccine for [son’s name] if it was free?”, which is consistent with current HPV vaccine accessibility for most adolescent boys in the United States (Markowitz et al., 2014).
Both items had a five-point response scale ranging from definitely not willing (coded as 1) to definitely willing (coded as 5).

**HPV vaccine attitudes.** Parents’ and adolescent boys’ attitudes toward HPV vaccination were the mediating variables of interest. Though my hypotheses relate to prosocial attitudes, I also included a variable for individualistic attitudes (assessing the importance of HPV vaccination to protect one’s son or oneself against genital warts and some cancers) to test whether differences in prosocial attitudes are consistent with (or vary from) differences in individualistic attitudes toward vaccination. The survey assessed parents and adolescent boys (separately) about their attitudes with respect to three foci:

1. **individualistic attitudes** (“The HPV vaccine protects guys from getting some kinds of HPV that can cause genital warts and some cancers. How important is it to you that [son’s name/your] getting the HPV vaccine could protect [him/you] against genital warts and maybe some cancers?”),

2. **prosocial girlfriend or wife attitudes** (“A guy who gets the HPV vaccine may be less likely to pass HPV to his future girlfriend or wife. How important is it to you that [son’s name/you] getting the HPV vaccine could protect [your son’s/your] future girlfriend or wife against genital warts and maybe some cancers?”), and

3. **prosocial community attitudes** (“If most people get the HPV vaccine, it may reduce the spread of HPV in communities. How important is it to you that [son’s name/your] getting the HPV vaccine could reduce genital warts and some cancers in the community?”). This item corresponds to the idea of herd immunity.

In addition to these three items, the parents’ survey assessed their **prosocial sex partner attitudes** (“A guy who gets the HPV vaccine may be less likely to pass HPV to his future sex partners.
How important is it to you that [son’s name] getting the HPV vaccine could protect his future sex partners against genital warts and maybe some cancers?”). For these questions, the five-point response scale was not at all important (coded as 1), slightly important, fairly important, very important, and extremely important (coded as 5). As shown in Table 5.2, the Pearson $r$ correlations between these attitude variables ranged from .83 to .90 (mean $r = .85$) for parents and from .72 to .80 (mean $r = .76$) for adolescents. Hence, while these variables are correlated, the extent of their unshared variance enables their use for assessing distinctions between the abovementioned focal attitudes.

Table 5.2 Correlations ($r$) between attitude variables for parents and adolescent boys.

<table>
<thead>
<tr>
<th>Attitudes of Parents ($n = 518$)</th>
<th>Attitudes of Adolescent Boys ($n = 401$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Individualistic</td>
<td>Individualistic</td>
</tr>
<tr>
<td>Prosocial girlfriend/wife</td>
<td>Prosocial girlfriend/wife</td>
</tr>
<tr>
<td>Prosocial sex partner</td>
<td>Prosocial sex partner</td>
</tr>
<tr>
<td>Prosocial community</td>
<td>Prosocial community</td>
</tr>
<tr>
<td>Individualistic</td>
<td>1.00</td>
</tr>
<tr>
<td>Prosocial girlfriend/wife</td>
<td>.84</td>
</tr>
<tr>
<td>Prosocial sex partner</td>
<td>.90</td>
</tr>
<tr>
<td>Prosocial community</td>
<td>1.00</td>
</tr>
<tr>
<td>Individualistic</td>
<td>.84</td>
</tr>
<tr>
<td>Prosocial girlfriend/wife</td>
<td>.83</td>
</tr>
<tr>
<td>Prosocial sex partner</td>
<td>.86</td>
</tr>
<tr>
<td>Prosocial community</td>
<td>1.00</td>
</tr>
<tr>
<td>Individualistic</td>
<td>N/A</td>
</tr>
<tr>
<td>Prosocial girlfriend/wife</td>
<td>N/A</td>
</tr>
<tr>
<td>Prosocial sex partner</td>
<td>N/A</td>
</tr>
<tr>
<td>Prosocial community</td>
<td>N/A</td>
</tr>
</tbody>
</table>


**Independent variables.** Independent variables included SES and race-ethnicity. SES was measured using the responding parent’s education and household income, modeled as separate variables. I categorized education as: college or university degree (hereafter “college degree”, the referent category), some college or university, high school diploma, and less than high school. The survey classified household income using 19 categories, ranging from < $5,000 to $175,000 or more. I took the midpoint of each income interval to approximate a continuous household income variable. Sensitivity analyses revealed similar results when I modeled income as a four-category scale variable.
Respondent race-ethnicity was coded as: non-Hispanic White (hereafter “White,” the referent category), non-Hispanic Black (hereafter “Black”), Hispanic, and non-Hispanic other or multiple races-ethnicities (hereafter “other race-ethnicity”).

**Control variables.** My analyses controlled for several potential confounding factors. Parent’s religiosity was based on a single item asking parent respondents: “How important is religion to you?” The five-point response scale ranged from not at all important (coded as 1) to extremely important (coded as 5). I imputed the sample’s mean religiosity score (3.6) for five participants (< 1% of the total sample) who did not answer this question, the only independent or control variable for which I had missing data. This allowed me to modestly increase my sample size, but it did not change the findings of my analyses.

The survey assessed the adolescent male’s age (11 to 17) on the date that his parent was screened for the survey, which I treated as a continuous variable. I used dichotomous variables to model the responding parent’s age (younger than 45 years = 0; 45 years and older = 1), marital status (married or cohabitating = 0; other = 1), and sex (female = 0; male = 1), as well as the number of children in the household (1 or 2 = 0; 3 or more = 1), and urbanicity of their location (urban = 0; rural = 1). Lastly, geographic region of residence, the US census region in which the respondent resided, was modeled categorically as South (the referent category), Midwest, Northeast, or West.

**Analytic strategy.** To test my hypotheses, I used seemingly unrelated (linear) regression (UCLA, 2015) to simultaneously model two sets of equations—for both parents and adolescents. Results obtained via this method and ordinary least squares regression were nearly identical.

First, I tested SES and racial-ethnic differences in HPV vaccination attitudes. For the parent sample, I specified four separate equations regressing parents’ (1) individualistic, (2)
prosocial son’s girlfriend/wife, (3) prosocial son’s sex partner, and (4) prosocial community attitudes on their household income, education, and race-ethnicity. For the adolescent male sample, I specified three separate equations regressing adolescent boys’ (1) individualistic, (2) prosocial girlfriend/wife, and (3) prosocial community attitudes on their household income, parent’s education, and race-ethnicity.

Second, I tested the extent to which individualistic and prosocial HPV vaccination attitudes were associated with willingness to vaccinate by first regressing willingness on income, education, and race-ethnicity, separately for parents and adolescent boys, and then adding the respective attitude variables to each model.

From these two sets of results for each sample, I used the product of coefficients method (Preacher & Hayes, 2008) to determine which SES and race-ethnicity variables had statistically significant, indirect associations with willingness to vaccinate via prosocial attitudes. Each indirect effect is the product of slope estimates observed for two indirect paths between SES or race-ethnicity, attitudes, and willingness: path a, the association between a SES or racial-ethnic category and an individualistic or prosocial attitude (as detailed in step 1 above); and path b, the association between an attitude variable and willingness to vaccinate (as detailed in step 2). Then, I determined the statistical significance of each indirect effect (reported as a*b) using bootstrapping procedures with 500 replications to generate bias-corrected 95% confidence intervals.

I conducted all analyses using Stata/SE 13. For regression results, I report unstandardized regression coefficients (b) with p values < .05 as statistically significant. Parent models control for the respondent’s age, gender, religiosity, and marital status, as well as their son’s age and household-level urbanicity, region, and number of children. Adolescent male models control for
the parent’s religiosity and marital status, and household-level urbanicity, region, and number of children.

**Results**

Before presenting the results of my hypothesis tests, examination of my dependent variables is informative for understanding the importance that parents’ and adolescent boys’ attributed to HPV vaccination as a means to prevent genital warts and some cancers for the vaccinated individual and others. Parents and adolescents felt that potential benefits associated with HPV vaccination were fairly to somewhat important regardless of whether they were to be conferred on the vaccinated adolescent, his future girlfriend/wife, his sex partner, or his community (for parents, mean = 3.52–3.66 and median = 4; for adolescents, mean = 3.34–3.66 and median = 4; Table 5.1). However, both parents and adolescents were generally “not sure” how willing they would be to get the vaccine for their sons or themselves (for parents, mean = 3.38 and median = 3; for adolescents, mean = 2.97 and median = 3).

Next, I outline the results of the analyses that test SES and racial-ethnic differences in individualistic and prosocial attitudes and willingness to vaccinate for parents and adolescent boys.

**Hypothesis 3a: Parents’ attitudes.** Parents’ perceived importance of HPV vaccination to protect their sons against genital warts and some cancers differed by education and race-ethnicity, but not income (Table 5.3). Parents with less than high school ($b = .45$) or a high school ($b = .32$) education (versus a college degree) and Black ($b = .60$) and Hispanic ($b = .32$; versus White) parents possessed more positive individualistic attitudes toward HPV vaccination.
For parental prosocial attitudes, significant associations consistent with my hypotheses existed for education and race-ethnicity, but not income. For education, parents with high school diplomas ($b = .37$) or less ($b = .48$; versus a college degree) placed greater importance on HPV vaccination to benefit their son’s future sex partners, while parents in each education level less than a college degree (from $b = .33$ for some college to $b = .63$ for less than high school) placed greater importance on HPV vaccination to benefit the community. For race-ethnicity, Black (versus White) parents possessed placed greater importance on HPV vaccination to benefit their son’s future girlfriends or wives ($b = .52$), sex partners ($b = .64$), and communities ($b = .63$).
Hispanic (versus White) parents also placed greater importance on HPV vaccination to benefit their son’s future sex partners \((b = .38)\).

**Hypothesis 3b: Adolescent boys’ attitudes.** For adolescent boys, individualistic attitudes toward HPV vaccination differed by race-ethnicity, but not SES. Black \((b = .64)\) and Hispanic \((b = .67; \text{versus White})\) males reported higher individualistic vaccination importance. For the two prosocial attitude measures, I observed no SES differences, while racial-ethnic differences only existed for prosocial community attitudes. As hypothesized, Black \((b = .48)\) and Hispanic \((b = .46; \text{versus White})\) males had more positive prosocial community attitudes.

**Hypothesis 3c: Parents’ willingness to vaccinate.** SES and race-ethnicity were not associated initially with parents’ willingness, as shown in Table 5.4, Model 1. However, introducing the attitude variables in Model 2 indicated suppressor effects for Black and Hispanic race-ethnicity, whereby their respective small, non-significant estimates in Model 1 \((b = -.01\) and \(b = -.04)\) were of larger magnitude and significant in Model 2 \((b = -.40\) and \(b = -.24)\). Of the four parent-specific vaccine attitudes in Model 2, only more positive individualistic \((b = .24)\) and prosocial girlfriend/wife attitudes \((b = .27)\) were associated with greater willingness to vaccinate one’s son. Sex partner- and community-specific attitudes were not associated with parents’ willingness, likely due to correlation with the other prosocial attitudes. Supplementary analyses that omitted other prosocial attitude variables found that sex partner- and community-specific attitudes were statistically significant predictors (data not shown).
<table>
<thead>
<tr>
<th></th>
<th>Parents (n = 518)</th>
<th></th>
<th>Adolescent Boys (n = 401)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Income</td>
<td>.00</td>
<td>−.00</td>
<td>.00</td>
<td>.01</td>
</tr>
<tr>
<td>( .07)</td>
<td>(.41)</td>
<td>(.04)</td>
<td>(.15)</td>
<td></td>
</tr>
<tr>
<td>Education</td>
<td></td>
<td></td>
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<td></td>
</tr>
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<td>College degree+</td>
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<td>Referent</td>
<td>Referent</td>
<td>Referent</td>
</tr>
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<td>.24</td>
<td>.11</td>
<td>.40*</td>
<td>.34*</td>
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<td>(.11)</td>
<td>(.17)</td>
<td>(.14)</td>
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<td>.10</td>
</tr>
<tr>
<td>( .15)</td>
<td>(.11)</td>
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<tr>
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<td>.28</td>
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<tr>
<td>( .19)</td>
<td>(.15)</td>
<td>(.22)</td>
<td>(.18)</td>
<td></td>
</tr>
<tr>
<td>Race-ethnicity</td>
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<td></td>
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<tr>
<td>White</td>
<td>Referent</td>
<td>Referent</td>
<td>Referent</td>
<td>Referent</td>
</tr>
<tr>
<td>Black</td>
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<td>−.40**</td>
<td>.14</td>
<td>−.14</td>
</tr>
<tr>
<td>( .17)</td>
<td>(.13)</td>
<td>(.18)</td>
<td>(.15)</td>
<td></td>
</tr>
<tr>
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<tr>
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<td>(.12)</td>
<td>(.17)</td>
<td>(.14)</td>
<td></td>
</tr>
<tr>
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<td>−.03</td>
<td>−.19</td>
</tr>
<tr>
<td>(.24)</td>
<td>(.18)</td>
<td>(.19)</td>
<td>(.15)</td>
<td></td>
</tr>
<tr>
<td>Attitudes toward HPV vaccination</td>
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</tr>
<tr>
<td>Individualistic</td>
<td></td>
<td>.24***</td>
<td>.16*</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>(.06)</td>
<td>(.06)</td>
<td></td>
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<tr>
<td>Prosocial girlfriend/wife</td>
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<td>.27***</td>
<td>.18**</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>(.07)</td>
<td>(.06)</td>
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<td></td>
<td></td>
<td>(.08)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prosocial community</td>
<td>.01</td>
<td></td>
<td>.23***</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(.06)</td>
<td></td>
<td>(.05)</td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
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<td>.45</td>
<td>2.05***</td>
<td>.57</td>
</tr>
<tr>
<td></td>
<td>(.44)</td>
<td>(.35)</td>
<td>(.47)</td>
<td>(.39)</td>
</tr>
<tr>
<td>R²</td>
<td>.10</td>
<td>.49</td>
<td>.08</td>
<td>.41</td>
</tr>
</tbody>
</table>


Note: Estimates are unstandardized slopes (standard errors). For the parent and adolescent male samples, Model 1 results were estimated via ordinary least squares regression and Model 2 results were estimated via seemingly unrelated regression. Parent and adolescent models respectively control for all variables listed in parent and adolescent columns of Table 5.1.

* p < .05, ** p < .01, *** p < .001

**Hypothesis 3d: Adolescent boys’ willingness to vaccinate.** Adolescent boys’ willingness to vaccinate was positively associated with having a parent who completed some college (versus a college degree; b = .40), but not with income or race-ethnicity, as shown in Model 1 (Table 5.4). Possessing more individualistic (b = .16), prosocial girlfriend/wife (b = .18), and prosocial community attitudes (b = .23)—as well as having a parent with some college
education \( (b = .34) \) were each positively associated with willingness, as shown in Model 2 (Table 5.4).

**Hypothesis 3e: Indirect effects of attitudes on willingness to vaccinate (mediation).**

Finally, I identified reliable indirect pathways from SES or race-ethnicity to vaccination willingness via prosocial attitudes. For parents, having a high school diploma or less (versus a college degree) was associated only indirectly with willingness to vaccinate via individualistic attitudes (indirect effect \( a*b = .08 \) and \( a*b = .11 \)); and Black (versus White) race-ethnicity was associated with willingness to vaccinate via individualistic \( (a*b = .14) \) and prosocial girlfriend/wife attitudes \( (a*b = .14) \). For adolescent boys, Hispanic and Black (versus White) race-ethnicity was associated with willingness to vaccinate via both individualistic \( (a*b = .10 \) and \( a*b = .11 \) and prosocial community attitudes \( (a*b = .11 \) for both groups). All reported indirect pathways \( (a*b) \) were statistically significant \( (p < .05) \).

**Discussion**

In this chapter I investigated SES and racial-ethnic differences in prosocial attitudes—attitudes that were associated with willingness to participate in HPV vaccination and that may impact health behaviors (Donahue et al., 2014). This prosocial focus offers potential for improving understanding of the mechanisms underlying SES and racial-ethnic disparities in health. My analyses of US national data support some of my prosocial hypotheses, which I discuss below with regard to the literatures that motivated them.

**SES and racial-ethnic differences in prosocial attitudes.** I predicted that lower (versus higher) SES and Black and Hispanic (versus non-Hispanic White) parents would place greater importance on HPV vaccination to help protect their sons’ (a) sex partners (b) girlfriends/wives, and (c) the community, and that lower SES and Black and Hispanic adolescent boys would place
greater importance on HPV vaccination to help protect their (a) girlfriends/wives and (b) the community. I observed educational (but not income) differences in two of the prosocial attitudes (sex partner and community)—as well as in individualistic attitudes—among parents, though no SES differences were observed for adolescent boys. I also observed racial-ethnic differences in all three prosocial attitudes among parents, and in prosocial community attitudes among males, in addition to racial-ethnic differences in individualistic attitudes within both groups.

The observed educational differences in parents’ prosocial attitudes may reflect numerous underlying social processes. Parents with higher education may have greater access to healthcare information and resources, possess greater health literacy, and be better able to take advantage of health-promoting treatments for themselves and their children (Lutfey & Freese, 2005; Ross & Mirowsky, 2005). However, they may also be those most likely to question scientific and medical authority and possess the agency needed to refuse vaccines on behalf of their children, which may result in individual-focused choices that undermine community health (Reich, 2014). Hence, while higher educated parents can prioritize their own child’s interest when making healthcare decisions, lower educated parents may be more reliant on their social bonds for health information, more socially aware, and, thus, possess more prosocially-oriented attitudes toward health behaviors that benefit others (Piff et al., 2010).

In contrast to education, the lack of association between income and parents’ prosocial attitudes suggests that lower income does not facilitate sharing of knowledge and resources among similarly positioned others—at least for HPV vaccination. Such findings may reflect that lower income adolescents are likely eligible for free vaccination through the VFC program (Gilkey et al., 2012), and thus less reliant on others for HPV prevention. Hence, income alone is likely insufficient for shaping parents’ prosocial attitudes toward vaccination.
The racial-ethnic differences in prosocial attitudes observed for parents and sons may be explained, in part, by neighborhood segregation, concentrated disadvantage, and the strong network ties that characterize some Hispanic and Black communities in the United States. As previously noted, Hispanic and Black populations are disproportionately concentrated in communities with limited resource access, while socioeconomically disadvantaged Whites tend to be geographically dispersed (Williams & Mohammed, 2009). The homophilous racial-ethnic and SES profile of these communities may strengthen informal social ties, promote a sense of bounded solidarity, and encourage resource sharing (Portes, 1998; Stack, 1974). Hence, the observed racial-ethnic differences in prosocial attitudes may be driven by compassion toward the needs of similarly disadvantaged community members, and norms and expectations that prosocial actions will be reciprocated (Dominguez & Watkins, 2003; Nelson, 2000). Also, disadvantaged neighborhoods are typically targets for safety-net health services, which traditionally focus on adolescent preventive care, and may facilitate sharing of information about and acceptability of HPV vaccination among community members and their social networks (Tsui et al., 2013).

Furthermore, given that cervical cancer incidence and mortality rates are, respectively, highest among Blacks and Hispanics (Jeudin et al., 2014), members of these racial-ethnic groups may be more likely to have been directly or indirectly affected by the disease and perceive HPV as a serious concern. In contrast, Whites may be less likely to have been affected by cervical cancer, and thus less likely to conceptualize HPV as a high-risk infection—an established barrier to HPV vaccine uptake (Holman et al., 2014)—or to identify with the prosocial benefits of HPV vaccination.
Differences between my findings for parents versus adolescent boys may be explained by life course stage. The lack of SES disparities in adolescent boys’ prosocial attitudes may be due to regular interactions with peers of varying SES in school and social settings, whose norms and values may contradict those of their families and moderate the effect of familial SES (Cockerham, 2013; Umberson et al., 2000). This effect may be especially relevant within socioeconomically disadvantaged communities where various competing and contradictory cultural frames and scripts—including those around sexual behavior—exist that may impact adolescents’ attitudes and behaviors (Harding, 2007). Regarding the lack of racial-ethnic differences in sons’ attitudes toward girlfriends or wives, despite Black and Hispanic adolescents being more likely to exist in communities that encourage adherence to prosocial community norms, these adolescents may never have had a serious girlfriend, and are unlikely to be thinking about their future spouse. Hence, in contrast to community members, girlfriends or future spouses may not resonate as strongly with adolescents as figures who will benefit from their actions.

**Prosocial attitudes and willingness to vaccinate.** I predicted that the possession of more positive prosocial attitudes toward HPV vaccination would be positively associated with willingness to vaccinate one’s son or oneself. For parents, despite the multiple associations found between (low) education and Black and Hispanic race-ethnicity and all three prosocial attitudes, only prosocial girlfriend/wife attitudes were associated with willingness to vaccinate one’s son—and this was only a significant indirect pathway for Blacks. For adolescent boys, I observed positive associations between both prosocial girlfriend/wife and prosocial community attitudes and willingness to be vaccinated, while only prosocial community attitudes served as an indirect pathway between Hispanic and Black race-ethnicity and willingness. Individualistic attitudes
were also positively associated with willingness to vaccinate for both parents and adolescent boys, and were a significant indirect pathway between (a) lower education and Black race-ethnicity among parents and (b) Hispanic and Black race-ethnicity among adolescent boys.

Attitudes shape intentions, which may affect behaviors (Bohner & Dickel, 2011). My findings suggest that when parents make health decisions for their children, prosocial attitudes may be overshadowed by individualistic concerns for their own child’s (or in the case of a future girlfriend or wife, a potential future family member’s) well-being. This finding is consistent with research that finds, though some parents recognize the importance of herd immunity, parents’ vaccination decisions are primarily driven by the benefits conferred to their own child (Quadri-Sheriff et al., 2012). Hence, despite the educational and racial-ethnic patterns I observed across parents’ prosocial attitudes, these attitudes alone are likely insufficient for motivating parents’ willingness to have their child participate in a health intervention.

My findings for adolescent boys, however, suggest that, in addition to individualistic considerations, the community benefits of prosocial health behaviors may be salient for motivating their willingness to participate. This finding echoes results of previous studies that demonstrate, when making vaccination decisions for oneself, individuals take into account the amount of good they can do for others and are not solely driven by self-interest (Vietri et al., 2012; Shim et al., 2012).

Also of note are educational and racial-ethnic disparities in willingness to vaccinate (Table 5.4), which suggest that other, unexplained pathways are not captured by my attitude measures. Adolescents whose parents had some college education (versus a college degree) reported greater willingness to vaccinate, even after controlling for attitudes. This finding may be driven by highly educated parents who may have been more likely to be anti-vaccine (Reich,
— and potentially shared negative vaccination viewpoints with their children. It is also consistent with research demonstrating that parents with college degrees are less willing to vaccinate their children against HPV (Jeudin et al., 2014). Additionally, when controlling for attitudes, Black and Hispanic parents are less willing to vaccinate their sons than non-Hispanic Whites. Barriers including a lack of information and concerns that the vaccine may be experimental, unsafe, or have long term side effects—similar to the barriers identified by Black and Hispanic parents shortly after HPV vaccines were approved for females (Jeudin et al., 2014)—may explain this initial lack of willingness to vaccinate sons. However, surveillance data indicate these barriers have not resulted in lower HPV vaccine initiation among these racial-ethnic groups (Curtis et al., 2014).

In terms of informing health-promoting initiatives that target adolescent health behaviors—a time when individuals and their parents may both play a role in health decision-making (Umberson et al., 2000)—these findings suggest that emphasizing the individual benefits that an intervention can confer is key for motivating adolescents’ willingness to participate, while communicating the prosocial benefits of interventions may also be helpful. Emphasizing prosocial benefits may be particularly useful for Black and Hispanic adolescents, as such a strategy may capitalize on community-focused norms (Dominguez & Watkins, 2003) and help mitigate the negative health disparities typically observed within these groups (Williams & Mohammed, 2009).

**Implications for future research.** This chapter has important implications for medical sociology in terms of studying and addressing health inequalities. First, it draws attention to the need to consider prosocial attitudes and vaccination as sociological phenomena. Research largely conceptualizes prosocial attitudes as a psychological trait (Penner, Dovidio, Piliavin, &
Schroeder, 2005), rather than a sociological phenomenon. Also, much previous research has treated vaccination (like many other health behaviors) as an individual, self-interested act or, in the case of parents of minors, as being driven solely by parents’ desires to act in the best interests of their child (Allen et al., 2010). However, my finding that (among parents) SES and (among parents and adolescent boys) race-ethnicity influenced willingness to vaccinate via both individualistic and prosocial attitudes illustrates a classic sociological focus: the interplay between the self and society (or community; e.g., Durkheim, 1893/2014; Simmel, 1950). An individual can be self- or child-interested and (a) also possess prosocial concern that motivates them to behave in a way that benefits others, or (b) lack prosocial concern, yet still undertake actions that have positive spillover effects for others. Thus, in considering prosocial attitudes and vaccination as sociological phenomena, the findings from this chapter demonstrate the utility of the prosocial attitudes concept for medical sociology, particularly for understanding the role of SES- and race-ethnicity-based attitudes for shaping willingness to participate in health behaviors that pose a collective health benefit.

Second, this chapter raises the salience of the differential role that SES and race-ethnicity may play at different stages of the life course. Existing research suggests that adolescents play an active role in the negotiation of their own vaccine-related decisions (Mathur et al., 2010; McRee et al., 2010), but also reveals that their preferences may be different from their parents (Vietri et al., 2011). This chapter adds to this knowledge base by suggesting that parent’s education—a commonly used marker of SES—may be a salient predictor of prosocial community attitudes in parents, but an insignificant predictor in adolescents. This finding suggests intergenerational differences in the role of education for shaping prosocial attitudes.
Third, the findings of this chapter have relevance for informing health disparities research. Well-documented SES and racial-ethnic disparities exist in the uptake of numerous medical innovations (Phelan et al., 2010). While prosocial attitudes might be helpful for motivating certain health behaviors, in many cases they may be insufficient for reducing health disparities due to limited knowledge about an innovation (including its individual and prosocial benefits), inadequate material resources, or a lack of willingness to adopt a particular treatment. Hence, prosocial attitudes may act as a countervailing mechanism for understanding health disparities—that is, a mechanism that is counter to and cumulatively smaller than other effects that collectively contribute to observed relationships between SES, race-ethnicity, and health (Lutfey & Freese, 2005). From a policy perspective, interventions solely targeting community attitudes may be limited in communities where socioeconomic resources are scarce.

As noted in chapter 1, SES and racial-ethnic inequalities in HPV vaccination are complex and vary across stages of uptake (Polonijo & Carpiano, 2013). Although reverse disparities in initiation exist that favor lower (versus higher) SES and Black and Hispanic (versus non-Hispanic White) adolescents, lower SES and Black (but not Hispanic) adolescents are less likely to complete the vaccine series and be fully-protected against HPV (Curtis et al., 2014). The reverse disparities identified in initiation may reflect the relative success of the VFC program and HPV vaccine accessibility at safety-net clinics in underserved areas (Tsui et al., 2013), as structural-level interventions that have weakened the association between social position and the uptake of a new health-promoting technology. A recent study also found that Black and Hispanic adolescent males with private and Medicaid insurance are more likely than non-Hispanic Whites to initiate HPV vaccination both at their first eligible visit with a healthcare provider and overall (Agawu et al., 2015). When considered with respect to my findings, this study suggests more
favorable patterns of vaccine acceptance within these groups. However, unequal vaccine series completion among lower SES and Black adolescents may reflect the persistence of additional barriers (e.g., lack of awareness that multiple vaccine doses are needed, lack of flexible work hours to schedule doctor appointments, or the need to travel far distances to obtain healthcare) that trump the potential of prosocial attitudes to reduce overall levels of HPV-related cancers in vulnerable communities.

Fourth, this chapter has implications for knowledge surrounding social ties and social capital, and their application to understanding inequalities in prosocial health behaviors. In terms of social connections, greater prosocial attitudes identified among lower educated parents and racial-ethnic minority parents and adolescent boys may be a product of their close ties to local community and kin, which facilitates a sense of community cohesion. However, the homophilous nature of their similarly disadvantaged social networks may undermine the greater propensity for prosocial attitudes toward health behaviors within some lower educated and racial-ethnic minority groups. These groups may also be insulated from knowledge (including knowledge pertaining to health and medical innovations) circulating outside of their immediate social circles due to the fact that their networks are characterized by a lack of less-intimate ties to other social networks (Granovetter, 1983). In contrast, higher status groups may have less dense networks that consist of a broader range of ties to other networks (Campbell, Marsden, & Hurlbert, 1986; Granovetter, 1983; Song & Chang, 2012), that privies them to a greater range of health-related knowledge yet results in a weaker sense of community altruism.

**Limitations and strengths.** This chapter tested theoretically-motivated *a priori* hypotheses by analyzing data from a unique national survey focused on parental and adolescent male attitudes toward HPV vaccination. Nonetheless, some limitations must be noted.
Given the cross-sectional survey design, I could not establish causal relationships. I was also limited by the time of survey administration—only six months after the permissive approval of HPV vaccination for males, when knowledge about and uptake of the HPV vaccine for boys was low (Reiter et al., 2013). Ideally, I would test how attitudinal differences relate not only to willingness to vaccinate, but also to vaccine uptake, and I would encourage future studies to consider this relationship.

Though this data had a unique range of variables that were useful for testing my conjectures, it did not include more in-depth SES- and race-ethnicity-related factors such as language proficiency, immigration status, duration of time in the United States, cultural norms, and community ties—factors that may be relevant for shaping one’s social capital, social networks, and, ultimately, prosocial attitudes and behaviors. Moreover, the sample limited my ability to investigate how SES and race-ethnicity may interact to shape prosocial attitudes and behaviors. Future research should consider these factors.

Nevertheless, this chapter makes a novel contribution to the literature and illustrates the relevance of prosocial attitudes for understanding health inequalities. Moreover, unlike much prior health disparities research, which focuses primarily on the deficits experienced by disadvantaged groups as explanations for health inequalities, this chapter study suggests potential social strengths that may encourage health-promoting behaviors among these groups.

In the proceeding final chapter, I discuss conclusions that can be drawn from the findings of this chapter, as well as my two preceding empirical chapters, in relation to the broader aims of this dissertation.
Chapter 6: Conclusion

I began this dissertation concerned with the complexities of how SES and race-ethnicity may shape health outcomes and health inequalities among adolescents. In aiming to better understand the pathways linking social conditions to health outcomes at this stage of the life course, I formulated a number of predictions from fundamental cause theory, health lifestyle theory, and prosocial health attitude perspectives. I empirically tested these predictions with respect to HPV vaccination, a relatively new and highly important health intervention that, when administered in adolescence, has the potential to significantly reduce the personal risk and population incidence of cervical, vulvar, vaginal, penile, and anal cancers (and pre-cancers) and genital warts. Through analyzing data from three, large, US nationally representative surveys on vaccination, my dissertation yielded three key sets of findings.

First, in chapter 3, I employed ideas from fundamental cause theory and examined the role of policy for shaping the equal diffusion of HPV vaccines (specifically, via provider recommendation and uptake) across various SES and racial-ethnic groups. I found that mandates requiring HPV vaccination for adolescent girls in DC and Virginia facilitated provider recommendations for some SES and racial-ethnic groups—but that patterns of provider recommendation did not correspond with patterns of vaccine uptake. These findings support the fundamental cause contention (e.g., Link, 2008; Phelan et al., 2010) that implementing policies that aim to distribute health-promoting interventions equally across populations can help to weaken the link between social position and health in terms of supporting the equal diffusion of information about new health technologies. However, mandates were also negatively associated with vaccine initiation and completion. Overall, this illustrates that when policy-level interventions are implemented in a manner that (a) does not facilitate universal uptake or
compliance and (b) leaves room for personal decision-making (such as opting out; e.g., advising individuals to purchase fluoride supplements rather than fluoridating the public water supply or requiring that landlords keep homes free of lead paint rather than warning parents to keep children away from chipped paint; see Phelan et al., 2010), they may be ineffective for reducing social inequalities in the uptake of health interventions and/or have the unintended effect of discouraging the overall uptake of such interventions, which undermines disease prevention at the population level.

Second, in chapter 4, I drew on health lifestyle theory and considered mother–daughter communication as a key pathway by which health inequalities may be reproduced across generations for SES and racial-ethnic groups. I found that mother–daughter communication about vaccines was positively associated with vaccine uptake and that parents were the most likely persons to initiate conversations about HPV vaccines. These findings identify parents as playing a significant role in shaping the vaccine-related health behavior of their daughters in adolescence and pre-adolescence. Also, Black mothers were less likely than their White counterparts to initiate conversations about HPV vaccines with their daughters; yet, Black adolescent girls were no less likely to have talked with their mothers about HPV vaccines or to ultimately be vaccinated. This suggests that the impact of race-ethnicity-based differences in mother–daughter communication on health behavior may be buffered by important figures (e.g., health providers) and forces (e.g., health policy) that operate outside of the parent–child dyad.

Third, in chapter 5, I integrated sociological and psychological ideas to examine the potential of SES- and race-ethnicity-based prosocial attitudes to motivate willingness to participate in health behaviors that have positive spillovers for others. With regard to HPV vaccination for adolescent boys, I found that (a) lower educated parents and (b) Black and
Hispanic parents and adolescent boys report greater prosocial vaccination attitudes—some of which were associated with greater willingness to vaccinate. This finding highlights the potential of prosocial attitudes to motivate members of typically marginalized communities to engage in health-promoting behaviors that benefit others. In doing so, it identifies an asset of these groups, who are more typically studied for their deficits.

Together, these key findings offer insights into several areas of scholarship—with respect to HPV vaccination specifically and medical sociology and population health more broadly. In this concluding chapter, I summarize these key research contributions and consider the larger theoretical and policy-oriented implications of my findings, while acknowledging the strengths and weaknesses of my approach, and identifying directions for future research.

**Research Contributions**

This dissertation makes contributions to the research literature on the pathways that shape health inequalities in three overarching areas: (1) the identification of multiple mechanisms and metamechanisms underlying fundamental causes, (2) the evaluation of adolescent agency and the intergenerational reproduction of health inequalities, and (3) the salience of prosocial attitudes for motivating health behaviors.

**Mechanisms and metamechanisms underlying fundamental causes.** Prior to this study, scant research had considered how (a) SES or racial-ethnic inequalities manifest in distinct stages of the uptake of new medical innovations or (b) early-life health interventions may shape disparities in specific diseases in adulthood (for an exception, see Polonijo & Carpiano, 2013). By investigating these phenomena, my research expands upon understandings of a number of mechanisms and metamechanisms underlying the fundamental cause relationship between social
position and health, including the role of flexible resources for creating latent health disparities and the significance of institutions and spillovers.

Chapter 3’s documentation of the existence of SES and racial-ethnic inequalities in provider recommendation and vaccine uptake—some of which were mitigated by mandates at the policy-level—contributes to the literature by identifying how flexible resources (e.g., money, knowledge, beneficial social connections) contribute to the production of health inequalities at various stages in the uptake of new health-promoting innovations. For example, adolescents in families with greater monetary resources may exercise (or have parents that exercise) their means to access vaccines in instances when private insurers do not automatically pay for all costs associated with them, while a lack of discretionary funds may exclude other adolescents from uptake. Money may also afford adolescents access to regular and trusted health providers who act as a beneficial social connection for sharing knowledge about and encouraging uptake of HPV vaccines, even when office visits are not for the purpose of vaccination (see Moghtaderi & Adams, 2016). As observed, flexible resources may be less significant for shaping future health outcomes when policies—such as vaccine mandates or the VFC program—remove financial barriers to access and mitigate inequalities in provider recommendations. In the absence of such policies, differences in flexible resources in adolescence may act to create latent disparities in disease at later points in adulthood (Polonijo & Carpiano, 2013). In this case, the observed differentials in vaccination by adolescents from various social groups are likely to translate into socially patterned inequalities in HPV-related disease incidence in adulthood.

By identifying distinct SES and racial-ethnic inequalities in provider recommendations of HPV vaccines, chapter 3 also highlights how the metamechanism of institutions—that is, the ways in which institutions’ differential treatment better facilitates health gains for certain groups
(Freese & Lutfey, 2011)—perpetuate social inequalities in health. A major finding from chapter 3 was the positive association between vaccine mandates and shrinking inequalities in provider recommendation for some lower SES and marginalized racial-ethnic groups. These findings indicate that, to some extent, inequalities in receiving provider recommendations are likely caused by institutions via providers varied interactions with patients based on their SES and race-ethnicity—interactions that may be leveled following policy changes, such as mandated vaccination.

By drawing attention to HPV vaccination as an early life course intervention, my research also contributes to better understanding the metamechanism of spillovers; in this case, how the attitudes and actions of others contribute to shaping health inequalities among adolescents. Chapter 5’s analysis of prosocial health attitudes provokes consideration of spillovers as it demonstrates how—even if a teen is not vaccinated—their health may be influenced by the willingness of their peers (and their peers’ parents) to vaccinate for either an individual- or prosocially-oriented benefit. If peers are willing to vaccinate, possess sufficient flexible resources to acquire vaccines, and exercise their means to vaccinate, their actions will ultimately have positive spillovers for the health of unvaccinated teens by reducing opportunities for disease transmission.

**Agency, the intergenerational transmission of health lifestyles, and adolescence.** The process by which families reproduce SES- and race-ethnicity-based health lifestyles across generations remains an important area for theoretical testing and expansion. My research adds to the existing literature by enhancing understandings of the extent to which (a) adolescents possess agency over their own health behavior and/or are influenced by their parents and other significant individuals in their lives, (b) SES- and race-ethnicity-based health lifestyles are
reproduced across generations, and (c) health lifestyle theory can be applied to understanding the uptake of one-time interventions.

Chapter 4’s findings, in particular, suggest that parents remain important figures for shaping adolescent’s vaccine-related behaviors via communicating with their children. By conversing with their children about vaccines (and, presumably, other health behaviors) parents may continue to shape adolescents norms, preferences, and lifestyles—that is, their habitus—in adolescence, which may potentially remain with them throughout adulthood. Given the small percentage of adolescents who were found to initiate conversations about HPV vaccines with their parents (as opposed to parents or other individuals, including health providers, initiating such conversations), adolescents overall do not appear to be exercising considerable agency with regard to vaccination—or, if they are, they are not doing so via the initiation of conversations with their parents. Hence, parents and health providers may play influential roles in shaping health behavior—or at least vaccination behavior—at this point in the life course.

While health lifestyle theory has most often been tested with regard to the reproduction of social-class- or SES-based behaviors (Cockerham, 2013), chapter 4 identified important racial-ethnic differences in communication-related aspects of health lifestyles—inequalities that were not observed for SES. Reverse disparities were identified for Hispanics in communication about meningitis and tetanus vaccines, which—despite running counter to the predicted direction—nonetheless suggest that race-ethnicity is a salient factor in shaping vaccine-related communication and, potentially, adolescent health lifestyles at large. Similarly, disparities in HPV vaccine conversation initiation between Black and White parents emphasize the salience of race for shaping adolescents’ access to health-related information within the family unit. Overall, these findings add to what is known about the intergenerational transmission of health lifestyles.
by suggesting (as other recent research on adolescent health lifestyles has also proposed; see Burdette, Needham, et al., 2017) that race-ethnicity is a particularly relevant factor for shaping health behaviors in adolescence. This finding is echoed by chapter 5’s identification of race-ethnicity-based—but not SES-based—differences in adolescent’s prosocial attitudes. One might conjecture that this is due to differences in the diffusion of information about vaccines (and other health technologies) within more insular racial-ethnic communities (see Williams, 2012).

In addition, this study also made a unique contribution in applying health lifestyle theory to understanding the uptake of a new health promoting medical intervention—rather than a sustained health behavior, such as diet, exercise, or substance use. Overall, my explicit tests of health lifestyle theory did not find significant SES or racial-ethnic differences in vaccine uptake (with the exception of a higher likelihood of tetanus vaccination among Black adolescents). This may indicate that health lifestyle theory is less applicable to understanding participation in short-term interventions (such as vaccination) and more relevant to explaining behaviors that are repeated over time (such as sleep habits or physical activity). This may be due to the fact that structural-level interventions, such as the vaccine mandates identified in chapter 3 or the implementation of safety-net clinics in underserved areas, may facilitate participation in short-term health behaviors such as vaccination, and do not require the individual to engage in sustained long-term behavior-related choices and modifications (e.g., as would be the case with changing one’s physical activity routine).

The salience of prosocial attitudes. Finally, this dissertation makes theoretical and empirical contributions to understanding health disparities at large by identifying the significance of prosocial attitudes as a pathway that may motivate willingness to participate in health behaviors that confer benefits to others. Specifically, chapter 5 represents one of the first studies
to explicitly examine how social position shapes vaccine-related intentions via prosocial attitudes. The positive relationships that I identify between marginalized racial-ethnic status (for parents and adolescents boys) and lower education (for parents) and prosocial attitudes—some of which mediate willingness to vaccinate—echo findings from research on social networks and social capital (e.g., Dominguez & Watkins, 2003; Portes, 1998) that suggest that marginalized social position strengthens reliance on social bonds and promotes prosocially-oriented behaviors. In doing so, I identify a potential countervailing mechanism that shapes the relationship between social position and health—that is, a mechanism that runs counter to and has cumulatively smaller effects on the relationship between social position and health than other pathways (such as SES- or race-ethnicity-based differences in the diffusion of health information or access to new medical innovations). The countervailing nature of this mechanism is emphasized by the findings of chapters 3 and 4, in which lower education and marginalized racial-ethnic status showed some negative associations with HPV vaccine-related outcomes, including parents initiation of conversations about vaccines as well as provider recommendations and uptake (especially, in the absence of vaccine mandates). Hence, it should be noted that there may be conflict between one’s prosocial attitudes and their ability to participate in prosocial health behaviors, as constrained by their knowledge and resources.

**Implications for Health Disparities-Related Practice and Research**

The broader implications of this research can be divided into two categories: (1) practical implications for the mitigating health disparities and (2) theoretical implications for understanding health disparities.
Practical implications for mitigating health inequalities. Health disparities constitute significant and challenging public health problems. My findings point to three strategic recommendations for mitigating these disparities.

First, my findings for HPV vaccination suggest that health providers play a key role in the generation of health disparities via differential recommendation patterns. They also suggest that providers have a particularly important role to play in initiating conversations about HPV vaccines in groups where parents are less likely to do so. In light of my findings and previous research that suggests provider’s recommendations are the strongest and most consistent correlate of vaccine uptake (e.g., Small et al., 2013; Moss, Reiter, Rimer, et al., 2016), policies and other health-promotion interventions that seek to target and equalize provider communication patterns may be effective strategies for targeting the reduction of social inequalities in the uptake of HPV vaccines and other preventive or diagnostic tools.

Second, my findings emphasize that policy makers aiming to reduce disparities in health must also carefully consider their potential impact on populations-at-large. Eradicating the diseases that HPV vaccines protect against requires high levels of compliance with national vaccination recommendations so that herd immunity can be achieved (Jeudin et al., 2014; Lee & Garland, 2017). My findings suggest that not only have vaccine mandates been ineffective for reducing relative differences in levels of HPV vaccine series completion (e.g., for Black adolescent girls) but also (in agreement with other recently published studies; e.g., Perkins et al., 2016; Moghtaderi & Adams, 2016) that vaccine mandates have led to declining levels of HPV vaccine uptake in the overall populations of the jurisdictions in which they were implemented. While mandates have reduced some key inequalities in provider recommendation and HPV vaccine initiation, their implementation has not been strong enough to target disparities in
completion and may have led many to opt-out of uptake—undermining the goals of disease prevention at large. Hence, policy makers must be sensitive to the contexts that they are dealing with and be prepared to address potentially negative public reactions, to ensure that well-meaning structural-level interventions are not met with significant public resistance.

Third, considering the role of prosocial attitudes as a motivational tool may offer a useful strategy for public health interventions and other efforts aimed at encouraging participation in activities that benefit the well-being of others (e.g., volunteering, donating) and even oneself (Thoits & Hewitt, 2001). However, in many cases they may be insufficient for reducing health disparities due to limited knowledge about an innovation (including its individual and prosocial benefits), inadequate material resources, or a lack of willingness to adopt a particular treatment. The unequal vaccine series completion (documented here, and in other surveillance studies; e.g., Curtis et al., 2014) among lower SES and Black adolescents may reflect the persistence of additional barriers (e.g., lack of a regular health provider for follow-up, lack of awareness that more than one shot is needed) that trump the potential of prosocial attitudes to reduce overall levels of HPV-related cancers in vulnerable communities.

**Theoretical implications for understanding health inequalities.** This dissertation has a number of theoretical implications for understanding health inequalities. First, race-ethnicity may be an especially relevant factor for understanding the diffusion of information about and uptake of new medical innovations in the adolescent life course stage. Compared to examinations of health lifestyles in adulthood, relatively few studies have examined health lifestyles in adolescence, and among these only a limited number have explicitly examined race-ethnicity (e.g., Blum et al., 2000; Burdette, Needham, et al., 2017). Hence, attempts at future theorizing
about and empirical testing of the role of health lifestyles for shaping health inequalities among adolescents should specifically take race-ethnicity into account.

Second, my findings suggest that the policy context is crucial to understanding the relationship between social position and health. While I demonstrated that vaccine mandates mitigated some of the pathways to health disparities, other policies such as the VFC program are likely to have explained many of the findings that ran counter to my hypotheses (e.g., higher initiation of vaccination among low-SES adolescent girls, even in non-mandated jurisdictions). Hence any consideration of the relationship between social position and health must be cognizant of and account for the various policies that may shape specific health outcomes. That is, to better understand health inequalities, one must not only consider the limitations of the social structure, but also the resources that it affords.

Third, prosocial attitudes toward one’s community may be a particularly salient motivator of willingness to participate in vaccination and other prosocial health behaviors. Hence, prosocial attitudes may be relevant for medical sociologists looking to understand personal-, family-, and community-level mechanisms underlying health disparities, including health spillovers, the uptake or adoption of health-promoting treatments, and the disease risks that such treatments may prevent throughout the life course. Future research in this area, however, must be careful to acknowledge that structural limitations may prevent some parents and adolescents from acting in accordance with such attitudes.

Finally, in light of my findings, it is important to acknowledge that efforts to address inequalities at the level of a specific health intervention, like HPV vaccination, will not necessarily contribute to lessening social inequalities in health overall. A key feature of the flexible resources discussed in this dissertation is their ability to be transported from one
situation to another under conditions of change (Link & Phelan, 1995). Diseases, treatments, and risk knowledge are ever evolving. Even if HPV-related diseases were to be eradicated due to the widespread uptake of HPV vaccines, we would expect to see persistent inequalities in health outcomes and mortality overall due to the changing importance and even emergence of other diseases (e.g., re-emergence of pertussis and syphilis in the United States; Chiappini, Stival, Galli, & Martino, 2013; Miller & Karras, 2010) and risk factors (e.g., youth vaping as a still-harmful substitute for tobacco use, opioid addiction; Kozlowski & Abrams, 2016), that those with greater access to flexible resources will better able to prevent and treat (Clouston, Rubin, Phelan, & Link 2016).

**Strengths and Limitations**

My research approach had a number of strengths. A key strength is the application of fundamental cause theory and health lifestyle theory—two leading theories on health inequalities—as well as emergent psychological and sociological literature, to generate and test theoretically motivated hypotheses about the pathways underlying SES and racial-ethnic health inequalities. Adolescent HPV vaccination in the United States was an ideal case to examine my specific hypotheses, as it is a health innovation that helps to protect against cancers for which there are well-established SES and racial-ethnic disparities (Jeudin et al., 2014). Differences in jurisdictional policy surrounding HPV vaccination offered a natural experiment to examine the role of health policy in relation to health inequalities. Moreover, given HPV vaccination necessitates parent–adolescent decision making (McRee et al., 2010) and has benefits that extend beyond the self to others (Hechter et al., 2013), it allowed me to study the reproduction of health inequalities across generations and examine the seldom-considered role of prosocial attitudes. Nonetheless, as with all research, there were limitations associated with this approach.
My analyses focused only on the case of adolescent HPV (and, to a lesser extent, meningitis and tetanus) vaccination. Consequently, my findings cannot be generalized to other vaccines or to vaccination behavior at alternate points in the life course (e.g., Hepatitis B vaccination in infancy, shingles vaccination in adulthood). These findings also cannot be generalized to other adolescent health interventions or behaviors (e.g., condom use, diet, initiation of pap testing). Similar analyses using comparative cases would thus be insightful for shedding light on the application of my findings to other behaviors and contexts.

It is also crucial to acknowledge the geographical limitations of my approach. While I provided an in-depth understanding of how disparities in adolescent HPV vaccination may be generated and reproduced within the United States, these findings are not representative of other geographic contexts that have distinct histories of social inequality and different health and social policies in place. For example, we would expect racial-ethnic inequalities in HPV vaccination to vary based on (a) the racial-ethnic composition of the society and (b) how the society distributes health-promoting resources that ways that either minimize or exacerbate inequalities between social groups (e.g., different welfare state regimes). Based on the theoretical approach used to frame this dissertation, we would expect that countries with stronger welfare-state policies as well as universal policies concerning vaccination and healthcare would have a more equal diffusion of HPV vaccines (and, by extension, have smaller inequalities in HPV-related cancers). For instance, we would expect to see more equal uptake of HPV vaccines in both Australia and Scotland, countries that have implemented fully-funded school-based HPV vaccination programs which have resulted in high-levels of uptake in the overall population and reported declines in HPV-related infections—even among unvaccinated individuals—suggestive
of herd immunity (Lee & Garland, 2017). Hence, cross-national comparisons of inequalities in HPV vaccination would provide complimentary insights to my findings.

Another strength of my research is its methodological approach. I leveraged three large US nationally representative datasets, focusing specifically on vaccine-related attitudes, communication, and behaviors. While I analyzed some of the best available data on HPV vaccines, these data and the methods I used also posed some limitations.

Overall, my data spanned the six-year period between 2008 through 2013, and some analyses were conducted using data from only one of these years (e.g., for mother–daughter communication and prosocial attitudes). Given HPV vaccines entered the market in 2007 and were still relatively new at the time of data collection, they are subject to period and cohort effects in terms of the diffusion of vaccine-related information. In other words, the longer that HPV vaccines are on the market, the more routine HPV vaccination will become. While my analyses are useful for documenting inequalities that typically emerge when new medical technologies become available, as HPV vaccination becomes more routine, it is reasonable to expect that patterns of HPV vaccine uptake and attitudes toward HPV vaccination will change. Continuing to follow social inequalities in HPV vaccination over time will thus be necessary for documenting long-term trends.

It is also important to acknowledge the overarching limitations of my choice to rely solely on quantitative methods to answer my research questions. While a quantitative approach is useful for documenting large-scale health inequalities, it does so at the expense of more in-depth understandings of why such inequalities may occur. For example, while I was able to document some inequalities in mother–daughter communication about vaccines, I was unable to assess the accuracy of information discussed, determine whether such conversations focused on the benefits
and/or perceived risks of vaccination, or establish whether adolescents actively engaged in these conversations (versus being passive recipients of knowledge). Qualitative inquiry investigating inequalities in HPV vaccination will undoubtedly complement my findings by adding greater understanding.

Despite the abovementioned overarching limitations, this dissertation makes a novel contribution to the sociological literature and explores a relatively understudied area for future expansion in medical sociology: vaccination. Moreover, it acknowledges not only the deficits experienced by disadvantaged groups as explanations for health inequalities, but also suggests potential social strengths (i.e., prosocial attitudes) that may encourage health-promoting behaviors among these groups.

**Future Research and Conclusion**

This dissertation points to three key directions for future research. First, future research should aim to further articulate the role of policy on the fundamental cause relationship between social position and health. My analyses suggest that, indeed, policy can interrupt some of the pathways by which social position shapes participation in vaccination. Additional analyses focusing on documenting (a) changes in the uptake of other health interventions following policy changes and (b) long-term inequalities in morbidity and mortality (e.g., actual incidence of HPV-related cancers and related mortality) following the introduction of such interventions will be important for further understanding the importance of policy for shaping health inequalities. As noted above, cross-national policy comparisons may also be useful in this endeavor.

Second, future research on health lifestyles in adolescent populations should aim to assess the congruence between the dispositions of adolescents, their parents, and other influential individuals (e.g., healthcare providers, teachers, peers). Qualitative research documenting
adolescent health decision-making may be particularly fruitful for this endeavor. Additionally, given the differences in conversation initiation patterns that I identified for Black adolescents, and the relative lack of research on race-ethnicity-based health lifestyles, further analyses of race-ethnicity-based health lifestyles in adolescence is certainly warranted.

Third, given the significant educational- and racial-ethnic differences in prosocial health attitudes and their identification as promising for motivating participation in prosocial health behavior, future research should focus on how to best leverage these attitudes into behaviors. For example, research could test whether intentions to participate in health behaviors and/or actual participation increases when communication materials about such interventions correspond to the prosocial- or individual-focused attitudes of the audience.

In conclusion, this dissertation identifies several pathways underlying the relationship between social position and health in adolescence via an examination of SES and racial-ethnic inequalities in HPV vaccination from fundamental cause, health lifestyle, and prosocial attitude perspectives. Specifically, it identifies how multiple mechanisms and metamechanisms—including flexible resources, means, institutions, and spillovers—shape adolescent vaccination behavior and highlights both the strengths and limitations of existing vaccination policy for supporting the equal diffusion of HPV vaccines across various SES and racial-ethnic groups. It contributes to understanding differences in the relative agency of adolescents and their parents (particularly, adolescent girls and their mothers) in decision-making about vaccines and emphasizes the significant role that parents and health providers play in shaping adolescents’ vaccine-related behavior. Finally, it identifies prosocial attitudes as being a pathway that may motivate vaccination—and possibly engagement in other health behaviors that offer a social benefit—among marginalized groups who may have the most to gain from participation. Overall,
these findings inform important knowledge gaps for understanding health inequalities, including: the potential impact of adolescent-focused policy for mitigating health inequalities, the intergenerational reproduction of health inequalities within families, and the relative importance of prosocial attitudes for motivating health behaviors among SES and racial-ethnic groups.
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