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Beyond Access: Predictors Of Unmet Need For Health Care From Adolescence To Young Adulthood

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BEYOND ACCESS: PREDICTORS OF UNMET NEED FOR HEALTH CARE FROM ADOLESCENCE TO YOUNG ADULTHOOD

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ABSTRACT

Background: Unmet need (UN) can be characterized as a person not getting care even when they think they need it. Younger populations are understudied for UN, even though UN can emerge as early as adolescence. The aim of this dissertation is to use the national Longitudinal Study of Adolescent to Adult Health (Add Health) to examine whether adolescent socioeconomic status (SES) or health factors better predict UN over time, and how UN differs by race/ethnicity. Life course perspective, cumulative advantage and disadvantage hypothesis, and fundamental cause theory inform my hypotheses. Primary hypotheses: 1) The likelihood of experiencing UN increases over time as adolescents transition into adulthood; 2) Racial/ethnic minorities are more likely than whites to have UN throughout the transition from adolescence to young adulthood, net of controls; 3) Higher adult SES is associated with lower odds of UN in adulthood, net of controls; 4) The likelihood of poorer SRH increases over time as adolescents transition into adulthood. Results: In longitudinal analysis (N=27,981 person period observations) I found that blacks and Native Americans have higher log odds of UN than whites after controlling for adolescent SES, adult transitions, and health measures. I also found that adolescent SES suppresses the beneficial relationship for excellent SRH for blacks compared to whites. In cross-sectional analysis (N=5,895 respondents) I found that those with the highest SES are most protected from UN, and that adolescent SES has some influence on adult SES attainment. Timing of life events such as college attendance

at traditional age (18-21) is the only education group with lower odds of UN (OR=0.715, $p<0.01$). Discussion: Those with the most advantaged SES accumulate more benefits protecting respondents from UN and poorer SRH compared to those with lower SES, supporting cumulative advantage. Despite blacks having better health measures than whites, they are at greater risk of UN, supporting the weathering hypothesis and fundamental cause of health disparity. Timing of events in the life course also matters for log odds of experiencing UN.

Keywords: unmet need, health disparities, race/ethnicity, health care, socioeconomic status

DEDICATION

This dissertation is dedicated to my mother, Barbara H. Rutland, who has supported me my entire life. This degree would not have been possible without her.

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LIST OF ABBREVIATIONS

ACA	Affordable Care Act
Add Health	The National Longitudinal Survey of Adolescent to Adult Health
BMI	Body Mass Index
CA1	Cross-sectional analysis 1
CAD	Cumulative Advantage/Disadvantage
CAM	Complementary and Alternative Medicine
CDC	Centers for Disease Control
CES-D	Center for Epidemiologic Studies Depression Scale
FCT	Fundamental cause theory
GEE	Generalized Estimating Equation
LA1	Longitudinal analysis 1
LA2	Longitudinal analysis 2
LCP	Life course perspective
OR	Odds ratio
SES	Socioeconomic status
SRH	Self-rated health
UN	Unmet need for health care in past year

INTRODUCTION

Unmet need (UN) can be characterized as a person deciding not getting care even when they think they need it (Harris et al. 2009). The sociological imagination would suggest that the motivation for this thought process is likely supported by societal influences. Yet, current literature on unmet need largely lacks sociological perspective or theory. When UN is studied, it also often focuses on middle-aged adults and above (Ford, Bearman and Moody 1999; Sanders, Donovan and Dieppe 2003). However, UN can start as early as adolescence (Ford et al. 1999). Studying UN earlier in the life course, from adolescence to young adulthood, with sociological theory can increase our knowledge of this subject in two ways: 1) to see the disparities in UN that emerge over time while people are young, and 2) to help us understand why these trends emerge. If researchers understand more about when and why disparities in UN emerge, resources can be allocated to change outcomes for UN. Additionally, researchers could benefit from examining disparities in UN with respect to racial/ethnic and socioeconomic status (SES) disparities, which is the gap this study fills. Integrating sociological theory helps disentangle the mechanisms that support UN and fill a literature gap of who experiences UN and at what points in the life course people experience UN. The elements of this study challenge the assumption that young people do not get care because they do not need it.

Three perspectives and theories can be applied to UN to analyze its origins and evolution in adolescents and young adults over time. Life course perspective (LCP)

considers the historical and personal contexts of people's lives as they intersect with social structures (Elder, Kirkpatrick and Crosnoe 2003). This proves useful for UN as it is likely influenced by shifting mechanisms during the transition to adulthood such as class circumstances, health policy, and employment. Cumulative advantage/disadvantage perspective (CAD) and fundamental cause theory (FCT)
frame UN as an issue that evolves over time due to disparities in statuses like race and class via socioeconomic status (SES) and as a function of life course. CAD perspective posits that SES-related health disparities increase across the life course, building upon each other (Lynch 2008). Fundamental cause posits that defining statuses like race/ethnicity and SES have early, persistent, long-term impacts on health (Link and Phelan 1995). Both theories can help researchers frame the contexts under which UN may occur, such as racial/ethnic status, class status, and health status. These theoretical perspectives can give new contexts to the issue of UN for a population that, while often considered healthy and resilient, is also often economically vulnerable.

This dissertation examines the trends of UN over time and what may be influencing discovered trends. I used data from Waves I-IV of the National Longitudinal Study of Adolescent to Adult Health (Add Health). Add Health is one of the best datasets available to study multiple dimensions of adolescents longitudinally. It is especially well-suited to studying the transition in the life course from adolescence to young adulthood. The method for longitudinal regression models is Generalized Estimating Equations (GEE). The following research aims are addressed in this dissertation:
Aim 1: Examine the trends in UN over time from adolescence to young adulthood.

Hypothesis 1: The likelihood of UN increases over time as adolescents transition into adulthood.

Hypothesis 2: Racial/ethnic minorities are more likely than whites to have UN throughout the transition from adolescence to young adulthood, net of controls.

Hypothesis 2a: Higher adolescent SES is associated with having lower likelihood of UN at baseline and later waves, net of controls.

Hypothesis 2b: For UN, the relationship between racial/ethnic minorities and whites is accounted for by SES, net of controls.

Approach: I performed a longitudinal analysis using GEE to test changes in UN over time. A model build-up approach demonstrates how disparities among racial/ethnic groups change after taking into account key variables such as adolescent SES, adult milestones (e.g., marriage and college attendance), and health measures. The initial model includes demographic variables, the following model includes adolescent SES, and the full model includes adult transitions and health measures. I utilize this model build-up in order to see the effects of adolescent SES on UN relationships before examining less explored theory- and literature-supported measures like adult milestones and health measures.

Aim 2: Examine the association between adolescent SES and adult SES in relation to UN.

Hypothesis 3: Higher adult SES is associated with lower odds of UN in adulthood, net of controls.

Hypothesis 3a: The relationship between adult SES and UN is partially accounted for by adolescent SES, net of controls.

Hypothesis 3b: The relationship between adult SES and UN is partially accounted for by health conditions and biomarkers, net of controls.

Approach: I performed a cross-sectional analysis using logistic regression with Wave IV data to test the relationship between adult SES and UN. Then, a model build-up approach demonstrates the effect of adolescent SES on the relationship between adult SES and UN. Similar to hypotheses in aim 1, I decided to use health measures in the full model for contextual factors. Note that while the models are cross-sectional, I have used longitudinal data from Waves I-IV to constructing "timing" variables. These models allow me to identify when respondents first attended college, started to smoke, etc., and if timing of such events influences the likelihood of experiencing or not experiencing UN. I also include health conditions (asthma, depression, anxiety, and migraines) and biomarkers (blood pressure class and body mass index [BMI] class) which are only available in Wave IV, as potential mediating variables in the full model. I do this because those with long-term conditions may be more likely to be in regular health care regardless of socioeconomic status, while those with less healthy biomarkers may be more likely to have UN because of socioeconomic factors preventing care receipt.

Aim 3: Examine the trends in health over time and UN from adolescence to young adulthood.

Hypothesis 4: The likelihood of poorer SRH increases over time as adolescents transition into adulthood.

Hypothesis 4a: Whites have better SRH in the transition from adolescence to young adulthood compared to racial/ethnic minorities, net of controls.

Hypothesis 4b: For SRH, disparities between racial/ethnic minorities and whites are partially accounted for by adolescent SES, net of controls.

Hypothesis 4c: For SRH, the relationship between racial/ethnic minorities and whites are partially accounted for by UN, net of controls.

Approach: I performed a longitudinal analysis using GEE to test changes in SRH over time, as I dichotomized SRH as excellent health/not excellent health. The model build-up approach demonstrates how racial/ethnic group comparisons change when considering key variables such as adolescent SES. The initial model includes demographic controls, adult milestones, and health measures, as health measures in particular have a connection to SRH well supported by literature. The second model includes adolescent SES, and the full model includes UN. This build-up allows me to assess how much of the relationship between race/ethnicity and SRH is explained by adolescent SES versus UN.

LITERATURE REVIEW

The purpose of this literature review is to understand the scope of and reasons for UN for health care among adolescents and young adults. This also requires a lens toward potential race/ethnicity and class-based disparities. Past research indicates that, depending on the resources and safety nets that individuals have, the transition from adolescence to adulthood can be challenging. Thus, this transition can affect health and health care outcomes. Overall, data suggests that health outcomes for young adults have been worse than health outcomes for adolescents in recent years (Mulye, Park, Nelson et al. 2009; Park et al. 2014). Understanding how unmet need for health care fits into these trends may inform research approaches to improve and better understand health outcomes for this population. I provide an overview of UN conceptually, the health trends that emerge during the transition from adolescence to young adulthood, the recent history with UN and health care for adolescents and young adults and why it matters, and how the cultural and human capital of race/ethnicity and of class support unmet need for care in this population. I also review gaps in the literature to build my argument for what gaps my study fills in this research area.

Describing Unmet Need

Broadly, UN is when a person's health and/or health care needs are not met in some way. UN is used in reference to both health and health care, and has been defined and referenced in many ways. It can be related to the access a person has to health services, or it can be related to the quality of those services once sought (Shi et al. 1999;

Haviland et al. 2005; Park et al. 2006). UN may also refer to undiagnosed or untreated disease burden or under-managed disease burden. For this study, I focus on the definition of unmet need in which a person thought that they needed to get health care recently and they did not get it (Harris et al. 2009). While UN in all its forms is likely to always exist, much like disease itself, it is imperative to understand how UN and supporting mechanisms affect adolescents and young adults so that researchers and policy makers can support ways to decrease incidence of UN. Decreasing UN is important because research suggests that if it begins in earlier stages of life, such as the transition to adulthood, it can support UN in later stages as well. The implications of long term or recurring UN include monetary costs and quality of life costs.

The Transition from Adolescence to Young Adulthood and Health

The transition from adolescence to adulthood is a complicated and unpredictable time in the life course. Past research indicates that the transition from adolescence to adulthood can be challenging depending on the resources and safety nets that individuals have. During these transitional years, gaps in jobs, education, and health care can occur in ways not typical in childhood and early adolescence or middle adulthood and beyond. Social and legal structures that support adolescent participation in the health care system, such as parental oversight and insurance coverage, begin to disintegrate when adolescents reach the legal age of 18 and are then expected to embark on a path to independence and new responsibilities (Park et al. 2006). Thus, health related measures tend to change greatly during this period in life, perhaps defying a common-sense assumption that young people are quite healthy and do not have many health care needs beyond acute infections or injury. However, past literature suggests otherwise.

During young adulthood, the most common reasons for health care visits are for acute and chronic conditions such as trauma-related disorders, asthma, acute bronchitis/upper respiratory infection, skin disorders, and mental health disorders (Park et al. 2006). Other health trends during the transition include substance use, injury, and homicide rates increasing from adolescence to a peak in young adulthood. Young adults also have the highest prevalence of substance use dependency compared to any other age group (Park et al. 2006). For women, need for reproductive health services increases during this transition as well (Henshaw 2000; Park et al. 2006). Young adults have an even higher mortality rate than adolescents. Yet despite these increasing health care needs and health risks, young adults have the highest uninsured rate of any of the age groups under 65 (Collins et al. 2003; Park et al. 2006). These trends indicate clear needs for care that may be going unmet. Additionally, with the literature available, we have a sense that UN cannot be explained fully by common access problems like being uninsured.

Unmet Need from Adolescence to Young Adulthood

Much of UN in young adults is explained by their high rates of being uninsured due to its association with foregone care (Park et al. 2006; Park et al. 2014), but UN disparities go beyond this problem. Being uninsured is one of the biggest risks for UN in young adults, but risk of being uninsured varies by groups such as the poor, Hispanics, those with low educational attainment, and – pre-Affordable Care Act of 2010 (ACA) – not being a student (Park et al. 2006). These groups and others are put at an inherent disadvantage and risk for UN which may also manifest beyond insurance barriers. As of 2014, four years after the ACA was passed and expanded health insurance options,

insurance rates improved for adolescents and young adults. However, the only major change in health care access and utilization for young people was increased dental visits (Park et al. 2014). At the same time the health trends and risks previously described changed little, with young adults still having worse health outcomes than adolescents overall (Park et al. 2014). Thus, the stagnation of health outcomes and health care utilization during a rise in insurance rates may indicate other factors for UN beyond insurance barriers, particularly barriers for pre-ACA populations like that of this study.

Currently, past research has pinpointed some other known risk factors for UN during the transition beyond being uninsured. Early research using the National Longitudinal Study of Adolescent to Adult Health (Add Health), a nationally representative longitudinal survey of youth, found that adolescents who were racial/ethnic minorities, who were older adolescents, who smoked cigarettes daily, and who frequently consumed alcohol had higher UN risk (Ford et al. 1999). This analysis was cross-sectional, so it does not establish if these risk factors promote risk of UN over time or the extent that UN changes as adolescents enter adulthood.

Capital, Unmet Need, and Race/ethnicity and Class

Other contextual explanations for UN include the quality of interaction with and feelings towards the healthcare system. With the transition from adolescence to young adulthood also comes the transition from pediatric to adult health care, which is a developmental period prone to gaps in care (AAP 2011). Possible explanations for these gaps vary. Adolescents, in particular, fear judgment or stigma, fear a lack of privacy, lack information, or have a general lack of access regarding health care (Elliott and Larson 2004; Hargreaves et al. 2015). Adolescents also place great importance on trusting a

doctor's confidentiality, without which they may defer getting health care (Klostermann et al. 2005). Still, we know little about the longitudinal manifestations and associated correlates of UN for adolescents and young adults. Further, beyond personal feelings about care and access through health insurance, there are other potential structural barriers to care such as the human and cultural capital tied to race/ethnicity and to class. The health care setting experiences of adolescents and young adults, and how those experiences vary by racial/ethnic identity and class, provide further context to the issue.

From the view of intersectionality, influences of racial/ethnic identity and class on risk of UN will often have cross-over, particularly for how capital develops for people of different statuses. Lower class people and people of color will have some similarities in the navigation of health care and sometimes those statuses will intersect, but even for lower-class whites their white privilege will afford a substantial amount of capital by itself. For example, research shows doctors can be primed with racist tendencies against patients of color, making whiteness a benefit for quality of care (Stepanikova 2012). To frame the intersection of these identities, I frame the literature in terms of human capital and cultural capital. Human capital is comprised of the individual skills, knowledge, and even health that a person possesses. Human capital is invested in through education, training, and medical care. The benefits of these individual traits cannot be separated from financial and physical assets because they are part of a feedback loop (Flora and Flora 2003). Cultural capital includes values and customs of one's culture, one's tendency to trust versus fear authority, and how one sees the world around them. It shapes what we perceive as knowledge, how knowledge is to be achieved, and what knowledge is considered valid (Flora and Flora 2003). These forms of capital help us

understand how race/ethnicity and class contribute to UN and other disparities in health because, like financial capital found in a bank, human and cultural capital's effects build and multiply over time.

Generally speaking, we know that racial/ethnic minority status is itself a barrier to meeting health care needs (Satcher 2000; Nelson, Smedley and Stith 2002). Past research has made it clear that there are racial/ethnic health disparities for a variety of health problems including obesity, cardiovascular disease, diabetes, and hypertension in favor of whites (Romero et al. 2012). Putting this in terms of human capital we have either failed to invest in people of color's health to a comparable degree to whites, or the payouts of investments in health are not equitable between people of color and whites. In an attempt to offset this dearth in human capital, national strategies have included increasing insurance enrollment for minority populations. Despite these efforts, we see again that increased access to health care (human capital) through insurance has not eliminated disparities as insured people of color still face numerous health disparities compared to whites (Hargraves, Cunningham and Hughes 2001; Romero et al. 2012). Understanding that disparities exist for insured people of color indicates there must be UN for these groups beyond factors like having basic access to health care and other forms of human capital. The role of cultural capital for race and UN provides further clarity.

Cultural capital could be influencing UN for people in racial/ethnic minorities through two main avenues: distrust of authority in health care settings and belief in values and customs that are at odds with participating in the traditional medical setting (e.g., using alternative medicine). Also, compared to whites, people of color trust their physicians less (Beach 2005). Across the board, Hispanics, blacks, and Asians perceive

more disrespect from physicians compared to whites (Johnson et al. 2004). These groups are also more likely to perceive bias and a lack of cultural competency in the healthcare system due to failure to recognize the unique experiences, needs, and perspectives of patients of color (Cook, Kosoko-Lasaki and O'Brien 2005; Eiser and Ellis 2007). The lack of cultural competency may speak to contentious thoughts physicians have about complementary and alternative medicine (CAM). While the use of CAM is integral to multiple ethnic groups, some small samples of physicians have been found to hold reservations about accepting CAM and discredit the validity of Asian and Native American practices, in particular (Berman et al. 1995; Wahner-Roedler et al. 2006). This is problematic because the use of CAM is high in groups like Asians and Hispanics relative to whites (Schoenberg et al. 2004; Gallant, Spitze, and Grove 2010), meaning that physicians who do not regard CAM as legitimate may alienate these people from traditional health care settings.

The evidence in the literature thus points to the importance of cultural competency in understanding potential risks for UN in racial/ethnic minority populations. This may be one explanation for why patients of color have been documented as experiencing overall worse or less satisfying care than whites during first contact visits, and having less trust in their physicians (Shi 1999; Haviland et al. 2005; Stepanikova et al. 2006). Some of these tensions in cultural capital and competency differences could be alleviated with race concordance between patients and physicians (LaVeist and Nuru-Jeter 2002), but this would require better representation from racial/ethnic groups in medicine, particularly blacks and Hispanics.

Since human capital represents the investments and skills gained and utilized to benefit individual lives, talking about human capital in terms of class and UN has an inherent logic. Decades of evidence show the disparate health effects of poverty and low education (Marmot 2005). Educated people, who tend to be of higher class standing, may have a better sense of how to enter and navigate the health care system. They may also have greater economic means to seek and get health care, particularly preventive care, which could decrease risk of UN. These means also reduce barriers to accessing care such as transportation and taking sick leave from work. In this way human capital is multiplicative, where one skill begets another. Quite simply, for the higher educated and middle and upper classes, there are fewer human capital barriers and more facilitators between health needs and health care (Marmot 2005).

Cultural capital is largely shaped by social class and its effects on health care are pervasive. Lareau (2003) explains that lower- and upper-class parents have different child rearing attitudes, which affects how children communicate and interact with the world as adults. Working class parents tend to raise their children under a model of natural growth and a sense of constraint. Working class children spend less time with adults and are raised not to challenge adults and to defer to them all as authority figures. Middle class children grow up in a model of concerted cultivation with a sense of entitlement, where children are encouraged to engage with adults as equals and expect adults to accommodate their individual needs. Lareau (2003) saw these differences first hand in doctor visits, where working class children were silent during doctor visits while middle class children spoke up and communicated their concerns and questions directly. The cultural capital afforded to middle class children in this way sets them up to be

comfortable going to the doctor and engaging in the healthcare system. Thus, cultural capital can buffer some risk of UN, whether through understanding the health care system or being comfortable engaging with the health care system. Since these tendencies are shaped through class early on, class (via SES) in adolescence may have sustaining effects into adulthood for UN.

To conclude, this dissertation project fills several gaps in the literature. First, while there is informative cross-sectional data for youth and UN, there is a dearth of longitudinal studies about unmet health care needs for this age population (Ford et al. 1999; Park et al. 2006; Mulye et al. 2009; Park et al. 2014). Second, studies that examine social determinants of health and their effect on UN in younger populations could benefit from implementing sociological theory (Ford et al. 1999; Hargreaves et al. 2015). This study longitudinally assesses UN from adolescence to adulthood, accounting for social determinants of health with sociological theory rooted in life course perspective and fundamental cause. This study also cross-sectionally accounts for the effect of adolescent SES on the relationship between adulthood SES and UN, as well as the effect of timing of life course statuses on UN. The theories I use propose that statuses like racial/ethnic identity and SES are fundamental causes of UN across the life course, and that the effects of these statuses build upon each other over time (CAD). With this study I show that UN in young people goes beyond traditional notions of lack of access to care as the main barrier to health care needs.

THEORY

While Life Course Perspective (LCP) is not typically considered a stand-alone theory, it is a flexible, comprehensive perspective and framework well-suited for understanding and pursuing longitudinal research questions. LCP can help address one of the key challenges to sociological research which is understanding the interwoven mechanisms that affect people's lives and properly addressing these realities scientifically. Rather than being able to study events in isolation, the timing of an event impacts how other events are experienced and further impacts outcomes associated with that event. For example, the timing of childbearing can impact educational trajectories and vice versa (Elder et al. 2003). Examining people's lives longitudinally further complicates this challenge as structural processes affect individuals differently over time. LCP provides the framework to understand individual lives as trajectories and how these trajectories impact the study of longitudinal relationships. Cumulative advantage-disadvantage perspective folds under LCP to further contextualize statuses and events that contribute to disparities in a manner reminiscent of intersectionality. CAD helps explain the role of race, class, and other differently privileged statuses when using LCP and how these roles compound and build off each other over time. Further, fundamental cause theory can be applied to CAD because scholars have argued that statuses associated with cumulative disadvantages, such as minority status and low socioeconomic status, contribute integrally to health disparities (Link and Phelan 1995; Williams and Collins 2001; Phelan and Link 2015). Using LCP and CAD with fundamental cause allow a more

comprehensive theoretical perspective and examination for how unmet need changes over time and what mechanisms drive this change.

LCP is based on the premise that the study of the trajectory of people's lives should consider the intersection of social structures (e.g., family structure), the social-historical context of their time (e.g., policy), and individual aspects of their lives (e.g., health problems). Previously, Elder et al. (2003) explain that life course research was non-existent in American sociology for decades. This was due to the unavailability of longitudinal data, which was itself due to the social-historical context of the time where cross-sectional data was the norm in budding research into the 1950s. The 1920s brought an interest in child development, and early childhood longitudinal studies eventually evolved into adulthood studies and this started a new area of research across the life course. LCP recognizes that time is connected to ever-shifting histories and experiences. Additionally, it posits that the current social political contexts impact individuals differently based on the age at which they experience this shifting context. Thus, the theory tends to define cohorts by shared or close birth years. For example, major changes to healthcare policy like the ACA affected everyone, but those changes had different impacts for different cohorts. Those who were young adults (i.e., under the age of 26 in 2010) benefited from being able to stay on a parent's insurance policy. However, adults just over the age of 26 missed this benefit. This may have altered these cohorts' health care access trajectories. LCP can frame the context of policies and other social-historical, cultural, and personal statuses as they influence cohort trajectories over time, providing deeper meaning to longitudinal analyses.

Thus, LCP allows the researcher to place the longitudinal outcomes of adolescent cohorts in social-historical and personal contexts as they age into young adults. In addition to LCP, scholars have folded a secondary contextual perspective that accounts for the varied benefits and harms people encounter across the life course. CAD perspective is often used to further contextualize the longitudinal findings of LCP to personal and societal statuses like race and class. Lynch (2008) describes CAD as the argument that SES-related health disparities increase across the life course, building upon each other. This occurs through two pathways. First, different levels of exposure to SES-related risk factors like smoking, diet, and exercise. For example, low-SES individuals are more likely to smoke, have poor diets, and less likely to exercise compared to higher-SES individuals (Cockerham 2013). Second, low-SES individuals also face barriers to resources that protect against poor health such as health care access (Lasser, Himmelstein and Woolhandler 2006). A separate perspective, the double-jeopardy hypothesis, is related to the compounding of SES-related disadvantages, with other marginalized social statuses such as race (Lynch 2008). However, since my study intends to dissect different working mechanisms within SES over time, as well as group statuses like race/ethnicity, I am expanding CAD using fundamental cause theory. Fundamental cause can be applied longitudinally, while double-jeopardy does not emphasize longitudinal perspectives (Phelan and Link 2015). Thus, I will use fundamental cause to include any multitude of statuses that may affect UN in adolescents and young adults.

Fundamental cause theory supports the use of analyzing multiple mechanisms over time, such as race/ethnicity and SES. Though originally Link and Phelan's (1995) theory emphasized that SES predicts health outcomes consistently starting from a young

age, it has also been expanded to include other social statuses like race/ethnicity as fundamental causes to health disparities. They report that those with more economic and social resources have better access to care, treatments, and other factors that support good health. These resources range from liquid monetary assets, to education, to social connections and support, to where you live, as well as many other factors. However, their research using this theory over time prompted a deeper examination into other potential fundamental causes, such as race/ethnicity. Phelan and Link (2015) present an argument that race/ethnicity also determines resources related to health, such as education and employment. Beyond this, they also cite that despite gains for some racial/ethnic minorities such as blacks on education, income, and wealth, the racial gap between blacks and whites has not shrunk appreciably. In fact, as of 2009, the wealth gap between whites and blacks had grown (Taylor et al. 2011). Phelan and Link (2015) conclude by suggesting that the health disparities between racial/ethnic groups in the United States is supported both by racial disparities in SES attainment and the disparities associated with SES and health outcomes. This does not mean that race/ethnicity is synonymous with SES. Rather, it speaks to how the interweaving of multiple statuses affects life course outcomes. Since these relationships form and compound over time, applying fundamental cause under the CAD perspective allows me to frame how these statuses weave together across the life course.

For this study, LCP frames the longitudinal analysis in a general social-historical context and CAD with fundamental cause contextualizes the statuses of advantage and disadvantage in the study population and how these statuses provide perspective to UN over time. LCP is based in 5 paradigmatic principles, which I use to theoretically frame

the study. The framework of each principle also presents a need for CAD perspective and fundamental cause to provide context for proposed status influences.

Principal 1 of LCP is the "Principle of Life-Span Development", that human development and aging are lifelong processes. Elder et al. (2003) make the point that development physically, emotionally, and socially does not end at age 18. Studies that tend to consider the contextual effects of SES, for example, tend to be cross-sectional rather than longitudinal (Ford et al. 1999; Karmakar and Curtis 2008). Additionally, while longitudinal health studies often study populations across long periods of time, only some studies provide contextual data like changes in SES or SES-related outcomes (Link and Phelan 1995; Phelan and Link 2015). This study considers that the population is in a transitional period from adolescence into adulthood, and that irrefutable changes occur within this context that set the course to some degree for the rest of a respondent's life and health.

Additionally, CAD recognizes that educational and class opportunities alone cause ripple effects of advantage and disadvantage across the life course. For example, those adolescents who attend college would be expected to have a higher likelihood of having insurance since some full-time students were able to stay on a parent's insurance longer at the time of data collection. Also, adolescents of higher SES origin at Wave I were more likely to attend college. Racial/ethnic status is also important to contextualize, as this status intersects with SES and evolves across time. It is difficult for researchers to say how early and to what extent race affects educational and class-based outcomes. For example, historic residential segregation of blacks from white spaces has impacted the educational and employment opportunities of black people in ways not seen

systematically in whites, prompting some scholars to argue that residential segregation is a fundamental cause of racial disparities in health (Williams and Collins 2001). Thus, considering the role of race/ethnicity in the life course framework is important. In conclusion, consideration of these statuses is necessary to recognize from the onset of a longitudinal study to ground the data in real-time and real-world influences.

Principle 2 of LCP is that of agency, that "Individuals construct their own life course through the choices and actions they take within the opportunities and constraints of history and social circumstance" (Elder et al 2003:11). Elder et al. (2003) describes the "planful competence" that adolescents are tasked with to plan for their lives in historical and social context. For example, adolescent men in the late 1920s had to make concessions for their life course plan when the Great Depression occurred. The depression pushed men with more resources and high agency back into school to seek advanced degrees for protection from the abysmal job market and low wages. Meanwhile, men without the resources for education had less agency and faced a terrible job market with low wages, which altered their earnings for years after. The less privileged were not able to catch up after these economic events. The cohort of men who transitioned right after the Depression were also able to select from diverse jobs with competitive wages compared to the earlier cohort (Elder et al. 2003). Thus, the after-Depression cohort had better future earning trajectories than the earlier cohort despite having more experience.

These observations parallel to more modern events like the Great Recession of 2008 when the housing market crashed and the passage of the ACA of 2010. During this time, many younger adults lost or could not find stable, well-paying jobs. They also were

vulnerable to not being able to access care due to finances and lack of insurance coverage. Like in the Great Depression, young adults with more resources could pursue college and advanced degrees and waited out the worst of the market. Meanwhile, those without a college education had to suffer through a recessed job market. These factors would have affected key health care access points such as insurance coverage because coverage was not offered for entry level jobs or for people who had to hold multiple part-time jobs. In 2008, policy dictated that children could only stay on as dependents on a parent's insurance plan past age 18 if they were full-time students, with a cap of age 21. Just two years later, however, the passage of ACA into law allowed young adults to stay on a parent's plan until age 26. This legislation increased health insurance coverage and allowed young adults flexibility in employment options and higher education endeavors. These historical-political contexts show that agency is shaped by available resources and environments occurring at various points in the life course, which CAD and fundamental cause can more wholly explain.

CAD and fundamental cause can frame the principle of agency through social flexibility versus constraints. Adolescents who can seek refuge in educational pursuits for their planful competence have high agency and a certain degree of flexibility, as do adolescents whose parents have good insurance benefits. Class advantages frame agency considerably, as wealthier classes have more privileges and protections than less wealthy classes, which build over time. For example, wealthier children have the resources for good educations which, as adults, allows them flexibility in seeking jobs with good benefits and low health risks. Education as a resource also supports delaying childbearing to older ages associated with economic advantages (Heck et al. 1997). In this way,

agency remains flexible and advantages accumulate and build on each other to support good health outcomes, but these mechanisms also show that the resources provided by having higher-SES fundamentally alter the path to good health. Unfortunately, the opposite is often true for poorer children. Poorer children's educational outcomes and job prospects are less consistent. Without the resources for higher education, some poor adults must work lower status, high-health risk jobs (Link and Phelan 1995). In this way agency is constrained and disadvantages accumulate and show that lacking SES-related resources fundamentally alters the path to good health.

Adding race/ethnicity to the equation further alters the role agency has for individuals. Race also affects agency due to social constraints, as people of color are judged against the standard of whiteness from birth. Stereotypes and biases stemming from racism and white imperialism, even unintentionally, impact people of color's life course trajectories and contribute to cumulative disadvantages in health and other areas (Stepanikova 2012). Additionally, arguments about race/ethnicity as a fundamental cause of health disparity display that agency varies in LCP. Even for blacks who are economically comparable to whites, and theoretically have high agency, we see a mortality disparity between blacks and whites (Phelan and Link 2015). Thus, under this principle we see that there are multiple mechanisms impacting an individual's ability to exercise agency as described in LCP.

The third principle of LCP is that of time and place, specifically that "the life course of individuals is embedded and shaped by the historical times and places they experience during their lifetimes" (Elder et al. 2003:12). Government and social institutions are always in flux across the life course, and their changes naturally influence

change in individuals as they experience life. Any number of structural level occurrences in history change some cohorts in ways they did not for others. The invention of the polio vaccine, and subsequent government oversight to ensure vaccination, made such a difference to the baby boomer generation that polio has been eradicated in today's generation of young people in the United States. Government oversight of health via health care policy, for example, thus influences a cohort's health throughout the life course. Again, we see how the ACA may have influenced young adults' health if they met the age extension of 26 at the time the legislation passed. Place matters as well, as more impoverished areas have greater need for government oversight, assistance, and protection, and those needs are not always met, which may contribute to unmet need and health disparities. Conversely, those living in more privileged contexts benefit from living in privileged places and may have less experience of unmet need.

CAD supports that the life course is shaped by historical time and place partly as a function of privilege. Those with more privilege are better protected against structural changes to their environment. Structural changes in historical time and place can contribute to the conditions that contribute to race/ethnicity and SES as fundamental causes of disease. As people at the individual level have little to no power against the structural institutions of society, those of underprivileged statuses are more vulnerable to changes in the historical time and place of society. For example, in funding cycles when federal education grants or Medicaid are cut the underprivileged face more challenges to improving their quality of life. These sorts of structural changes will fluctuate throughout childhood and continue into adolescence and young adulthood, while more privileged individuals are not affected by these changes. Structural changes such as affirmative

action benefited lower class and minority people's education and job prospects in ways older cohorts never experienced, for example. I argue that changes to structural forces affect those with the least power the most. Thus, disparities in UN likely increase as adolescents age out of parents' insurance coverage and parental oversight of health and health care needs. There are countless examples of how changes to policies affect individuals across the life course and while these effects cannot be measured or controlled in a vacuum in the social world, I attempt to recognize them to some degree of efficacy in this study.

The fourth principle of timing suggests that the benefits and consequences of life transitions, behavioral patterns, and events in a person's life are influenced by the timing of these occurrences in the life course (Elder et al. 2003). Common examples of this principle supported by research are the effects of the timing of normal life occurrences, like leaving the parental household, getting married, or child bearing. For example, children who leave the parental household later in life and those who have children while young experience poorer mental health outcomes and a domino effect of other issues that "pile up" (Elder et al. 2003). An example for UN would be that those who have unmet need earlier in life may be more likely to experience UN later in life.

This principle also re-emphasizes how the timing of life events subsequently affect the timing of other life events in advantageous or disadvantageous ways. The example Elder et al. (2003) provide is the difference in life prospects for early Great Depression children compared to late Great Depression children. Those who were born earlier experienced the depression for longer, but also were of an age to be expected to participate in child labor and other avenues of supporting their families. Those born later

in the Depression era experienced it for a shorter amount of time and were too vulnerable and young to be expected to work and try to support the family with labor. Again, a modern example would be that adolescents and young adults pre-Affordable Care Act did not benefit as younger cohorts did for maintaining health insurance. The timing of such policies affects UN in purely circumstantial ways.

The CAD and fundamental cause effects of the timing principle are clear: when disadvantageous events and behaviors develop earlier in life, their effects pile up and lead to worse outcomes on multiple domains. Conversely, when advantageous events and behaviors develop earlier in life, their effects pile up earlier as well. Similarly, the timing of contextual events like policies and of historical periods like recessions and depressions can support piling up effects across the life course. Timing of events and behaviors in adolescence is important because these individuals are in a transitionary phase in their lives from children whose parents are responsible for their basic needs and beyond to young adults trying to support and establish themselves. Class and race circumstances certainly impact the CAD and related fundamental cause mechanisms found in the timing principle, as those who are white and/or of higher SES can transition with more security to adulthood. For example, even if the timing of some events and behaviors, like parenthood or underage drinking behaviors, are less than optimal, privileged statuses can help protect adolescents and young adults from negative outcomes typically associated with these events and behavior. Greater access to resources as we see in fundamental cause theory would explain this phenomenon. High-resource individuals can protect themselves from negative outcomes that low-resource people cannot avoid. This “safety net” afforded to people with greater privilege can protect them from some consequences

of timing. The safety net may perhaps decrease the likelihood of unmet need itself and dampen its potentially negative impact on health.

The fifth and final principle is that of linked lives, meaning that "lives are linked interdependently and socio-historical influences are expressed through this network of shared relationships" (Elder et al. 2003:13). Parents' lives are linked to children's lives and vice versa, and these relationships change over time due to socio-historical influences; as adolescents transition into adulthood, the parent-child relationship naturally transitions as well. Adolescents are expected to take on more responsibility for their health as they leave the household for college or the work force. For example, they are expected to make their own doctor appointments, fill their own prescriptions, and eventually obtain their own insurance plans and care networks. Elder et al. (2003) give an example of how sudden economic hardship changes the relationship of parent and child at least temporarily as parents struggle with poorer mental health and children take on responsibilities to support themselves and the family at earlier ages. With a historical focus on unmet health care need, we can explore how the socioeconomic status of parents, such as their educational attainment, can influence children's safety nets and trajectories for health and healthcare attainment. These influences are part of CAD for this principle and are closely related to the "piling up" effects laid out in the fourth principle of timing. Additionally, as the resources understood in fundamental cause also reproduce, we can expect young adults to continue to benefit from resources existing in adolescence, particularly for those with greater early SES.

Using LCP to examine and frame changes in unmet need over time from adolescence to adulthood enabled me to piece together how time and socio-historical

occurrences influence the statuses and lives of respondents. CAD and fundamental cause further allowed a framework that recognizes the socially constructed differences of race and class, and how this recognition is necessary to examine unmet need. The use of these perspectives and theory provided the longitudinal framework necessary to examine the research questions proposed as fully as sociological research constraints allow.

METHODS

Data

The data for my three analyses are from Waves I-IV of Add Health. All information regarding the sample and survey contents comes from Harris et al. (2009). Add Health's goal was to follow adolescents into young adulthood. It is a nationally representative sample of U.S. adolescents who were in grades 7-12 in 1994-1995 (Wave I). The response rate for Wave I was 79% with a total N=20,745. Wave I participants were originally collected using a clustered sample design via schools, which included an in-home interview. The sample comes from 80 high schools and 52 middle schools which did not have equal probability of selection, but systematic sampling methods and implicit stratification for the study design created a nationally representative sample of U.S. schools including region, urbanicity, size of school, and ethnicity.

In 1996, the Wave II in-home interviews follow-up with 14,738 Wave I respondents (response rate of 88.6%), with the exception of those individuals who were only in the disabled sample at Wave I and those who were in 12th grade at Wave I and not part of the genetic sample. Wave III in-home interviews was conducted five years later from 2001-2002 when the majority of the 15,170 Wave I respondents (response rate of 77.4%) were between 18-26 years old. When Wave IV in-home interviews were conducted in 2007-08, the majority of respondents were between the ages of 24-32. Wave IV had a total N=15,701 with a response rates of 80.3% (Harris et al. 2009). I discuss my sample's missingness in results.

Waves I-IV sought to measure multiple social and behavioral measures as adolescents transitioned to adulthood. Wave IV added biomedical measures beyond the STD and HIV testing in Wave III as well. In all waves, respondents filled out an in-depth survey and in Waves III and IV were asked to give biospecimen samples, for which agreeing respondents received additional compensation. I used the Add Health data to conduct three analyses: longitudinal analysis 1 (LA1), longitudinal analysis 2 (LA2), and cross-sectional analysis 1 (CA1). I describe these analyses in greater detail in the analytic strategy section, but as an overview LA1 examines UN over time from adolescence to young adulthood, LA2 examines self-rated health over time from adolescence to young adulthood, and CA1 examines the association of key events that take place during the transition to adulthood, such as education, with UN.

Measurement

Dependent Variable for Longitudinal Analysis 1 and Cross-sectional Analysis 1

The dependent variable is UN. UN for this dissertation refers to if the respondent did not get health care even when they thought they needed care in the past 12 months. This question is dichotomous: "Has there been a time in the past 12 months when you thought you should get medical care, but you did not?" (Harris et al. 2009). Responses are "no" as in, "No, there *was not* a time when I did not get medical care despite thinking I should" (need care = 0), and "yes" as in "Yes, there *was* a time when I did not get medical care despite thinking I should" (need care = 1). I used this measure to gauge respondents' thoughts on their health care needs just as I and other researchers use self-rated health to gauge respondents' thoughts on their overall health status. Other research have used this unmet need variable as an independent variable assessing its impact on

self-reported health measures longitudinally from adolescence (Wave I of Add Health) to adulthood (Wave IV of Add Health) (Hargreaves et al. 2015). More similar to this study is one that has used the unmet need variable as a dependent variable, seeking to understand cross-sectionally what youth behaviors and statuses are associated with unmet need at Wave I of Add Health (Ford et al. 1999). The measures and methods for the study blend some methodological elements of these two studies together for a more holistic view of UN in these age groups.

Dependent Variable for Longitudinal Analysis 2

SRH is used as the dependent variable of LA2. In Waves I-IV, self-rated health is asked as "In general, how is your health?" The variable has five answer choices: excellent, very good, good, fair, and poor. Answer choices are dichotomously coded as (1=excellent; 0=very good or less). The motivation for this is that young people would be expected to generally feel that they are quite healthy, barring disadvantageous background characteristics (Bauldry et al. 2012).

Main Explanatory Variables and Controls for Longitudinal Analysis 1 and Longitudinal Analysis 2

Race and ethnicity are pulled from Wave I data and merged with the Wave IV dataset via respondent ID. I used a consolidated race/ethnicity variable available from Udry, Li and Hendrickson-Smith (2003) in Add Health data files. Udry et al. (2003) addressed the option for respondents to mark multiple races by a series of rulesets. Hispanic remains a separate ethnicity following Census definitions of race at that time. Most people who do not mark a racial category in the school survey mark Hispanic or Latino as their ethnicity (Udry et al. 2003). The benefit for using this variable is that the

complications from both respondents and interviewers marking racial/ethnic identities are collapsed into manageable categories. The major cons are that many respondents are classified by a "primary" race, such as Native American if they also mark white. Further, marking Hispanic ethnic identity supersedes racial identification. The main Add Health variables that construct the combined race/ethnicity variable are: "What is your race? You may give more than one answer", with white, black or African American, American Indian or Native American, Asian or Pacific Islander, and Other as options; and "Are you of Hispanic or Latino origin?" with no, yes, don't know, and refused as answer choices. I included all separate racial/ethnic categories as identified by Udry et al. (2003). Those who are identified as Hispanic become a separate ethnic category in the race/ethnicity variable. Race/ethnicity was recoded as a series of dummy codes (1=white/Hispanic/black/Asian/Native American/other; 0=n/a).

Adolescent SES is a main explanatory variable in LA1, CA1, and LA2. Add Health asks parents a questionnaire in Wave I of the survey. Note that most parent respondents are mothers. Since adolescents are still under the care of someone else, their independent measures of SES would not fully indicate the resources they benefit from. Thus, these measures of parental resources from Wave I will represent adolescent SES: parent's education and household income at Wave I. In the parent survey, parents are asked, "How far did you go in school?" Response options included less than high school, high school, some college, and college or more. Parent's education levels are recoded as dummy variables (less than high school; high school including vocational training; some college; college or more with less than high school as the reference group). Wave I household income is assessed by parents by asking the following: "About how much total

income, before taxes did your family receive in 1994? Include your own income, the income of everyone else in your household, and income from welfare benefits, dividends, and all other sources." There is an open response range of 0-999, as in $0-$999,000 dollars. I then constructed terciles of income. UN (described above) serves as an explanatory variable in LA2 since SRH is the outcome variable in that analysis.

Controls for longitudinal analyses 1 and 2 include gender, parent nativity, insurance status, adult milestones (respondent ever attended college and respondent ever married), and health measures (self-rated health, depressive symptoms score, smoking habits, and drinking habits). Gender was assessed only in wave I by the interviewer confirming "that R's sex is (male) female. (Ask if necessary.)" Answer choices are "R is male", "R is female", "refused", and "don't know". Gender is dummy coded as (male; female=1, inapplicable=0).). I expected that there would be cultural and social differences between native respondents and children of immigrants. I controlled for parent nativity using the following question from the parent questionnaire: "Were you born in the United States?" (1=yes; 0=no). In Wave I respondent's insurance status must be taken from the parent questionnaire. That question is "What kind of health insurance does {NAME} have (check all that apply)", with Medicare, Medicaid, individual or group private coverage, pre-paid health plan, other, none, and don't know as options. In Wave IV, the insurance variable was asked as "Which of the following best describes your current health insurance situation?" Response choices were: "you have no health insurance", "you get insurance through work", "you get insurance through a union", "you get insurance through school", "you are covered by your husband's or wife's insurance", "you are covered by your parent's insurance", "you are covered because you are active

duty military", "you buy private insurance yourself", "you are on Medicaid", "you are covered through the Indian Health Service", "you do not know what your health insurance is", "refused", and "don't know" (Harris et al. 2009). For Waves I and IV, insurance was recoded as dichotomous such that no insurance=0 and any type of health insurance=1. Since insurance coverage was not measured in Waves II and III, insurance status for Wave I was matched to Wave II and insurance status for Wave IV was matched to Wave III.

Adult milestones are common events that people achieve throughout the life course. For LA1 and LA2 these are ever attending college or ever being married. Respondent's education was measured longitudinally so that I could identify life course timing of college education in CA1, but for the longitudinal analyses the recodes for education indicate ever having attended college at any time in the life course (1=ever attended college; 0=n/a). Similarly, those who ever report being married across waves were put into a dichotomous variable (1=ever married; 0=n/a).

Regarding health measures, SRH (described above) was used as a control for respondent's perceived health in LA1, since this may affect feelings of needing care. Add Health asks respondents a modified Center for Epidemiologic Studies Depression Scale (CES-D) scales in every wave (Lewisohn et al 1997; Harris et al. 2009). Total score ranges from 0 to 3, where 0 is "never or rarely", 1 is "sometimes", 2 is "a lot of the time", and 3 is "most of the time". Scale items that measured positive emotions such as "How often was the following true during the past seven days? You felt happy" were reverse coded. All scales constructed for each wave have a scale reliability alpha score >0.85. Smoking and drinking habits were assessed because their use in adolescence is associated

cross-sectionally with UN (Ford et al. 1999). The chosen smoking variable is "Have you ever smoked cigarettes regularly, that is, at least 1 cigarette every day for 30 days?" It is dichotomous (1=yes; 0=no). Additionally, since light drinking habits are not typically considered risky behaviors for young adults, measures that indicate heavier use were pulled from Waves I-IV for translatability. The drinking variable measured alcohol consumption by asking, "Over the past 12 months, on how many days have you gotten drunk or 'very, very high' on alcohol?" The response options were "never, "once or twice", "once a month or less", "2 or 3 days a month", "once or twice a week", "3 to 5 days a week", or "nearly every day". The variable was recoded using Centers for Disease Control (CDC) guidelines for what constitutes heavy drinking. According to the CDC, heavy drinking is when a woman has 8 or more drinks a week or a man has 15 or more drinks a week (Esser et al. 2014). I used the definition of heavy drinking rather than binge drinking (i.e., about 5 drinks in 2 hours for men and 4 drinks in 2 hours for women) since if someone is drinking enough to get drunk more than once a week they are probably meeting the heavy drinking benchmark regularly. Thus, I recoded drinking as "never drunk" (answered "never"), "infrequently drunk" (answered "once or twice", "once a month or less", "2 or 3 days a month"), and "heavy drinking" ("once or twice a week", "3 to 5 days a week", or "nearly every day").

Main Explanatory Variables and Controls for Cross-sectional Analysis 1

Race/ethnicity and adolescent SES as described above were included in analysis 3. Respondent adult SES at Wave IV was added to this analysis and was measured with the following variables for the cross-sectional analysis: respondent's education, household income, household assets, household debt, and homeownership. Respondent's

education was measured longitudinally so that I could identify life course timing of college education. Through a series of mutually exclusive recodes I identified first age of college enrollment across waves. These recodes allowed me to make a new variable, college, that categorizes timing of first college attendance as "early" (ages 18-20), "late" (ages 21 and older), or "never". The variable income was the respondent's household income in the previous year, asked as "Thinking about your income and the income of everyone who lives in your household and contributes to the household budget, what was the total household income before taxes and deductions in 2006/2007/2008? Include all sources of income, including non-legal sources." There were 12 response categories, in a range from 1="less than $5,000" to 12="$150,000 or more". This variable was then put into terciles. Household assets was used as a measure of wealth. It was asked in the following way: "What is your best estimate of the total value of your assets and the assets of everyone who lives in your household and contributes to the household budget? Include all assets, such as bank accounts, retirement plans and stocks. Do not include equity in your home." Answer choices range from 9 categories, beginning with "less than $5,000" and ending with "$1,000,000 or more". I treated this variable as continuous. Respondent household debt was also examined. It was asked as "Now, think about your debts besides any mortgage on your home. How much do you and others in your household owe altogether? Include all debts, including all types of loans, credit card debt, medical or legal bills, etc." The answer choices range from "less than $2,000" to "$250,000 or more", so I treated this variable as continuous as well. Finally, homeownership was measured dichotomously in Wave IV as "Is your house, apartment,

or residence owned or being bought by {YOU AND/OR YOUR SPOUSE/PARTNER}?" (1=yes; 0=no).

Adolescent SES as described previously is the same in the cross-sectional analysis. Gender, parent nativity, insurance status, and marriage history are the same as well. For health, SRH, depressive symptoms score, timing of becoming a regular smoker, timing of becoming an infrequent or heavy drinker, diagnosis of health conditions (asthma/depression/ anxiety/migraines), and biomarkers (blood pressure classification and BMI classification). I added the diagnosis measures and biomarkers in the cross-sectional analysis because these health measures are only available in Wave IV. I postulated that those with disease burden are more likely to have UN, and people with unhealthy biomarkers may have higher UN. Health conditions were measured in Wave IV by asking, "Has a doctor, nurse or other health care provider ever told you that you have or had: asthma, chronic bronchitis or emphysema; depression; anxiety or panic disorder; migraine headaches?" (0=no; 1=yes). Add Health measured blood pressure and BMI in in-home visits in wave IV. Systolic and diastolic blood pressure were measured with a cuff and reported in the survey. Add Health then put blood pressure results into categories: "normal: systolic <120, diastolic <80, prehypertension: systolic 120-139 or diastolic 80-89, hypertension I: systolic 140-159 or diastolic 90-99, hypertension II: systolic 160+ or diastolic 100+" (Harris et al. 2009). I recoded blood pressure classification to 1=normal, 2=prehypertension, and 3=hypertension I or hypertension II. Add Health measured BMI by measuring respondents' height and weight. They used those calculations to measure BMI, and also constructed a variable that put BMI measurements into classifications: underweight: BMI<18.5, normal: BMI 18.5<25,

overweight: BMI 25<30, obese I: BMI 30<35, obese II: BMI 35<40, obese III: BMI 40+. I recoded BMI classification to 1=underweight, 2=normal weight, 3=overweight, 4=obese I, obese II, or obese III.

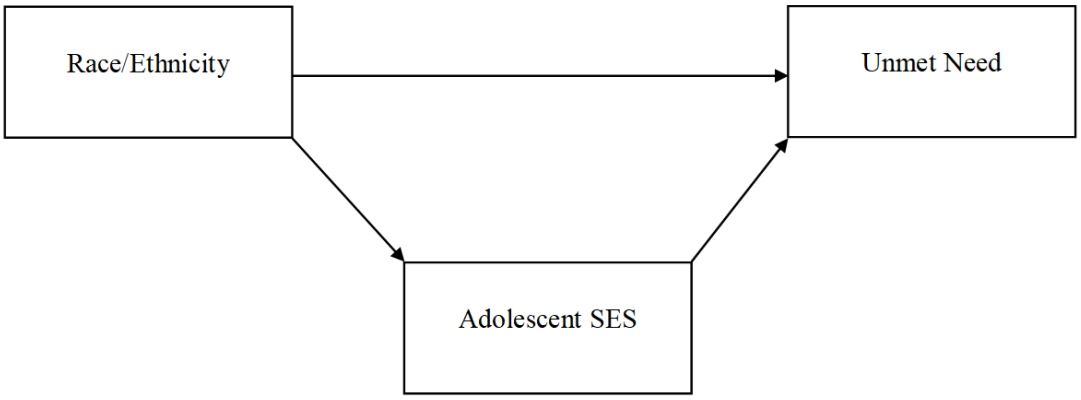
Bivariate Estimations of Reasons for Unmet Need

Reason for UN is only measured in the survey for Waves I-III. However, bivariate estimation of the reasons for UN, via GEE models, provided some context to the larger analyses. The question is "What kept you from seeing a health professional when you really needed to (check all that apply)". Response choices were varied but included lacking transportation, not being able to pay, not knowing who to go see, and not wanting parents to find out.

Analytical Strategy

The analytical strategy encompassed several main components: descriptive statistics, bivariate statistics, and multivariate regression models for two longitudinal analyses and one cross-sectional analysis. Descriptive statistics included means and percentages of the variables used in analyses. Bivariate statistics included regression estimations of the reason for UN over time (measured only in Waves I-III). All analyses were conducted in Stata 15 (StataCorp 2017). All longitudinal analyses considered age non-linearity by adding a quadratic term. The modeling strategy for each hypothesis is described in detail below. Hypotheses 1 and 2 are for the first longitudinal analysis. Hypothesis 3 is for the cross-sectional analysis. Hypothesis 4 is for the second longitudinal analysis.

Figure 1.



Hypothesis 1: The likelihood of UN increases over time as adolescents transition into adulthood.

The following GEE model, adapted from Landerman, Mustillo and Land (2011), tested the effect of the dichotomous dependent variable (UN) longitudinally:

Model 1:

$ln(p(UN)_{ij}/1\text{-}p(UN)_{ij}) = \beta_{0i} + \beta_1 Age_{ij} + e_{ij}$,

where p_{ij} is the probability that the respondent has UN at time j, β_i is a random intercept that can vary across respondents, and Age_{ij} is age at time of survey for respondent i and wave j = 1, 2, 3, or 4, UN_i is unmet need at baseline and e_{ij} is error. Hypothesis 1 will be supported if β_1 is significant.

Hypothesis 2: Racial/ethnic minorities are more likely than whites to have UN throughout the transition from adolescence to young adulthood, net of controls.

The following GEE model tested the relationship between race/ethnicity and UN longitudinally:

Model 2:

$ln(p(UN)_{ij}/1\text{-}p(UN)_{ij}) = \beta_{0i} + \beta_1 Age_{ij} + \beta_2 black_i + \beta_3 Asian_i + \beta_4 Hispanic_i + \beta_5 Native\ American_i + \beta_6 black_i * Age_{ij} + \beta_7 Asian_i * Age_{ij} + \beta_8 Hispanic_i * Age_{ij} + \beta_9 Native\ American_i * Age_{ij} + Z_{ij} + e_{ij}$,

where β_i is a random intercept that can vary across respondents, and Age_{ij} is age at a given wave, UN_i is unmet need at baseline, Z_{ij} are young adult milestone variables and controls, and e_{ij} is error. β_{2i}, β_{3i}, β_{4i} and β_{5i} are dummy variables that represents racial/ethnic minority categories. β_{6ij}, β_{7ij}, β_{8ij}, *and* β_{9ij}, demonstrates whether racial/ethnic disparities between people of color and whites for UN increase over time, controlling for initial disparity. Hypothesis 2 would be accepted if any racial/ethnic groups had higher odds of UN at baseline compared to whites. Additionally, the interactions between race/ethnic groups and time (β_{6ij}, β_{7ij}, β_{8ij}, *and* β_{9ij}) allowed me to examine whether the likelihood of having unmet need increased (positive significant coefficient for β), decreased (negative significant coefficient for β), or remained stable (non-significant coefficient for β) relative to whites.

Hypothesis 2a: Higher adolescent SES is associated with having lower likelihood of UN at baseline and later waves, net of controls.

Hypothesis 2b: For UN, the relationship between racial/ethnic minorities and whites is accounted for by SES, net of controls.

The following GEE model tested the relationship between race, SES and UN longitudinally, with race/ethnicity included in the vector of controls:

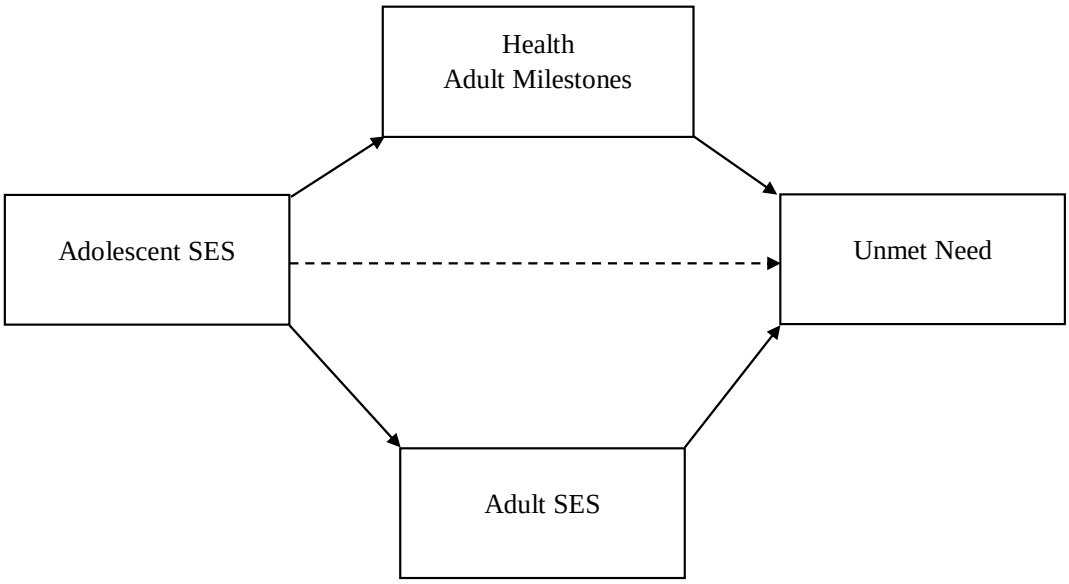
Model 2a for hypotheses 2a and 2b:

$ln(p(UN)_{ij}/1\text{-}p(UN)_{ij}) = \beta_{0i} + \beta_1 Age_{ij} + \beta_2 Adolescent\_SES_i + \beta_3 Adolescent\_SES_i * Age_{ij} + Z_{ij} + e_{ij}$,

Adolescent SES was analyzed in two ways: as a composite of all variables comprising SES and using model buildup of individual SES components. Hypothesis 2a would be accepted if higher SES was associated with lower odds of UN. Hypothesis 2b would be accepted if the odds of UN for racial/ethnic minorities relative to whites decreased with the addition of SES to the models.

The next hypotheses for this study examine the relationship among adolescent SES, adulthood SES, and UN in adulthood cross-sectionally. Von Hippel and Lynch (2014) used an approach that allowed them to test the selection effect of high BMI to higher education. For this study I examined the selection effect of low adult SES to higher risk of UN, with adolescent SES measuring fundamental cause and CAD.

Figure 2.



Hypothesis 3: Higher adult SES is associated with lower odds of UN in adulthood, net of controls.

Hypothesis 3a: The relationship between adult SES and UN is partially accounted for by adolescent SES, net of controls.

Hypothesis 3b: The relationship between adult SES and UN is partially accounted for by health conditions and biomarkers, net of controls.

The following multivariate regression model tested the relationship between SES and UN cross-sectionally in Wave IV of the survey when respondents were young adults:

Model 3:

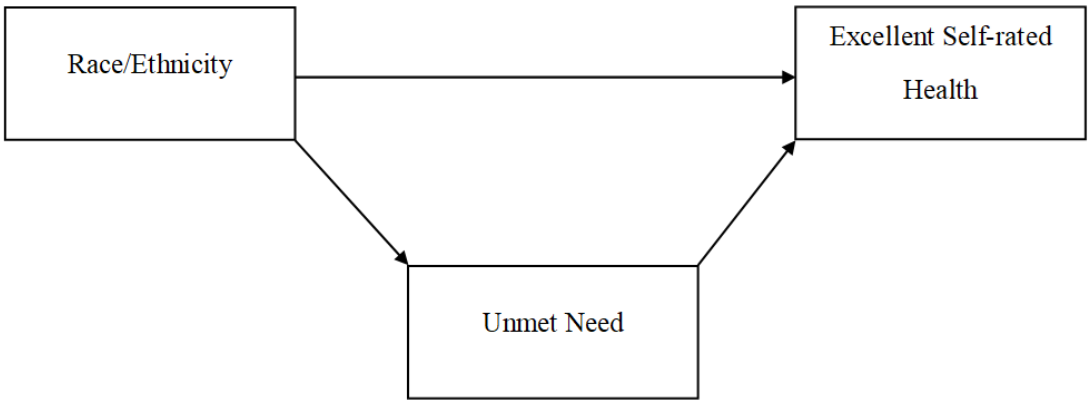
$ln(p(UN)/1\text{-}p(UN))=\beta_0 + \beta_1 Adult\_SES + \beta_2 Adolescent\_SES + Z + e$

where *UN* is the outcome, β_0 is the intercept, *Z* is the full range of controls including race, and *e* is the error term. Adolescent SES and adult SES was analyzed in two ways: as a composite of all variables comprising SES and using model buildup of individual SES

components. Hypothesis 3 would be supported if those with higher adult SES had lower odds of UN. Hypothesis 3a would be supported if adolescent SES significantly decreased the association between adult SES and UN. Hypothesis 3b would be supported if health conditions and biomarkers significantly decreased the association between adult SES and UN.

The last hypotheses examine the second longitudinal analysis, looking at the relationship between health and UN over time where SRH is the outcome.

Figure 3.



Hypothesis 4: The likelihood of poorer SRH increases over time as adolescents transition into adulthood.

Hypothesis 4a: Whites have better SRH in the transition from adolescence to young adulthood compared to racial/ethnic minorities, net of controls.

Hypothesis 4b: For SRH, disparities between racial/ethnic minorities and whites are partially accounted for by adolescent SES, net of controls.

Hypothesis 4c: For SRH, the relationship between racial/ethnic minorities and whites are partially accounted for by UN, net of controls.

The following regression model tested the effect of the dichotomous dependent variable (SRH) longitudinally:

Model 4:

$ln(p(SRH)_{ij}/1\text{-}p(SRH)_{ij}) = \beta_{0i} + \beta_1 Age_{ij} + Z_{ij} + e_{ij}$

where pij is the probability that the respondent has lower self-rated health at time j, β_{0i} is the intercept, Z_{ij} are young adult transition variables and controls, and e_{ij} is the standard error. Age was centered at Wave I and quadratic of age is also in the model. The young adult transition measures were accounted for by adding population averaged model commands with an ar1 correlation structure into Stata. Then, the following model examined the relationship between UN, race, SES, and health.

Model 4a for hypotheses 4a and 4b:

$ln(p(SRH)_{ij}/1\text{-}p(SRH)_{ij}) = \beta_{0i} + \beta_1 Age_{ij} + \beta_2 black_i + \beta_3 Asian_i + \beta_4 Hispanic_i + \beta_5 Native\ American_i\ \beta_6 black_i * Age_{ij} + \beta_7 Asian_i * Age_{ij} + \beta_8 Hispanic_i * Age_{ij} + \beta_9 aNative\ American_{ij} * Age_{ij} + Z_{ij} + e_{ij}$,

where βi is a random intercept that can vary across respondents, and $Ageij$ is age at a given wave, UN_i is unmet need at baseline, Z_{ij} are controls, and e_{ij} is error. β_{2i}, β_{3i}, β_{4i} and β_{5i} are dummy variables that represent racial/ethnic minority categories. β_{6ij}, β_{7ij}, β_{8ij}, *and* β_{9ij}, demonstrate whether racial/ethnic disparities between people of color and whites account for poorer SRH over time, controlling for initial disparity.

Hypothesis 4 would be accepted if the odds of better SRH decreased over time. Hypothesis 4a would be accepted if odds of better SRH were higher for whites than for racial/ethnic minorities. Hypothesis 4b would be accepted if odds of better SRH for racial/ethnic minorities (compared to whites) increased as odds of better adolescent SES increased. Hypothesis 4c would be accepted if UN was associated with a decrease in the effect of race/ethnicity on health.

RESULTS

Most missingness in my final sample is from attrition over waves. As all of my analyses incorporate wave IV data and the data is survey weighted, the weights remove people from analyses who did not answer the survey in waves analyzed. In LA1 and LA2, I used the longitudinal sample weight gswgt4 for analyses, which weights for people who answered Waves I, II, III, and IV of the survey. Dropping respondents who were missing for this weight resulted in 11,353 observations being dropped from analysis. Another necessity to Add Health's survey design is creating subpopulations to handle missing in the final valid sample for invalid responses and legitimate missing for specific variables (Chen and Chantala 2014). Legitimate skips, however, were not dropped but added to "no" or "n/a" categories. The subpopulation for analysis results in a sample size of 27,981 person period observations for LA1 and LA2. In CA1, I used the cross-sectional sample weight gswgt4_2 for analyses, which weights for people who answered Wave I and Wave IV of the survey. Dropping respondents missing for this weight results in 5,974 people being dropped from analysis. The total sample size for gswgt4_2 is 59,200 person period observations. Dropping respondents whose wave does not equal 4 dropped a further 44, 400 person period observations. The subpopulation for analysis results in a sample size of 5,895 person period observations.

The results of bivariate logistic regression estimations of reason for UN are presented for age and racial/ethnic categories in Table 1. There were 10 reasons asked consistently across Waves I-III. While some categories reached significance at $p<0.05$

their standard errors were so large that I chose not to report them in the text. Since respondents to these questions were a smaller subpopulation, sample sizes for some questions may have been too low to calculate meaningful differences. Hispanics, blacks, and Asians were over 2 times more likely than whites to report that their UN was due to not knowing who to go see. Blacks and Asians were at increased log odds of transportation issues contributing to their UN (OR=1.89, $p<0.01$) and OR=1.98, $p<0.05$, respectively). Compared to whites, all other racial/ethnic groups had increased logs of UN due to not wanting their parents to know about their problem. Asian respondents in particular had almost 2.5 ($p<0.001$) the log odds of having this issue compared to whites. Hispanics, blacks, and Asians were more likely than whites to report that being afraid of the doctor was the reason for UN. For blacks, this reason for UN was highly statistically significant (OR=1.81, $p<0.001$).

Descriptive statistics for the total sample of LA1 and LA2 are displayed in Table 2. The descriptive statistics for the cross-sectional study are in Table 3. Additional bivariate analyses were run to examine whether there are any significant unadjusted relationships between the predictor variables and UN in LA1 (Table 4) and CA1 (Table 5) and between the predictor variables and SRH in LA2 (Table 6).

Longitudinal Analysis 1 Results

Descriptive statistics of the total sample of LA1 are detailed in Table 2. The total sample size of the study was 27,981 person period observations. Table 4 presents unadjusted bivariate associations between model variables and UN over time. To ensure results are nationally representative, I used subpopulation measures with the *svy:* command in Stata for descriptive statistics (Chen and Chantala 2014). The percentages in

Table 2 come from results of s*vy, subpop(samp2): prop* or *svy, subpop(samp2): mean* commands.

Select changes in UN by wave are described below (not visualized). In Wave I, UN is reported among 18.2% of respondents. By Wave IV, this increased to 25.1% of respondents. This trend is true for UN by several racial/ethnic groups as well. 16.3% of whites reported UN in Wave I, while 24.2% reported UN by Wave IV. For Hispanics this was an increase from 21.1% to 25.1%; for blacks, an increase from 22% to 29.5%, and for Native Americans, an increase from 31.3to 33.8%. Asians showed little change in UN and those in the other category reported a decrease from Wave I (24.4%) to Wave IV (14.1%). UN by parent's income shows that the highest percentage of UN in Wave I was among those in the lowest parent income group at 37%. This remained true in Wave IV as well, growing to 42%. In comparison, respondents from the highest parent income group started at 32% reporting UN in Wave I and only 27% reporting UN in Wave IV.

Table 7 displays the model build-up of LA1. These models test the following hypotheses: 1) that likelihood of UN increased over time (model 1), 2) that racial/ethnic minorities are more likely than whites to have UN over time (model 1), 2a) adolescent SES is associated with lower likelihood of UN (model 2), and 2b) the relationship between racial/ethnic minorities and whites is accounted for by SES (model 2). Model 1 includes age centered at the mean and the quadratic of age, race/ethnicity, gender, parent nativity, and insurance coverage. Model 2 adds adolescent SES measures (parent's household income wave I and parent's education). Model 3 adds adult milestones (ever reporting marriage across waves I-IV, respondent educational attainment across Waves I-IV, and insurance coverage across waves I-IV) and health measures (excellent self-rated

health, depressive symptomatology score, ever being a regular smoker, and drinking behaviors, across waves I-IV). I ran various model build-ups after models 2 and 3 to determine how each added variable influenced the prior models. I ran post hoc analyses on all time-varying variables to examine whether significant associations were likely due to within group comparison rather than between group comparisons.

Reference Figure 4, constructed from centered age and quadratic age values, which shows the exponentiated effect of age for the trend in UN over time. The time period is over twelve years, the approximate time of Waves I-IV, when respondents are anywhere from 12-34 years old. Observe that odds of experiencing UN increases over a period of 8 years, approximately from WI to WIII, when respondents are aging from their teens to their early and mid-twenties. The rate at which the odds of experiencing UN decreases around this time and somewhat plateaus, but odds of experiencing UN is now higher for young adults than it was for adolescents. I interpret this as support for hypothesis 1, UN increases as adolescents enter adulthood. Examining the race/ethnicity variable I find that Hispanic, black, and Native American respondents have higher log odds of UN than whites, with Native American respondents having log odds almost 2 times higher than that of whites (OR=1.28, $p<0.01$; OR=1.2, $p<0.001$; and OR=1.92, $p<0.001$, respectively). Gender and parent nativity have no discernible relationship with UN in the base model, but insurance coverage is associated with reduced log odds of UN compared to those lacking insurance coverage (OR=0.643, $p<0.001$). Hypothesis 2 is supported in the base model for Hispanics, blacks, and Native Americans as they have significantly higher odds of having UN compared to whites. Hispanics are 1.28 ($p<0.01$) times more likely to have UN than whites, blacks are 1.2 ($p<0.01$) times more likely than

whites, and Native Americans are 1.92 ($p<0.001$) times more likely than whites to have UN.

Model 2 includes adolescent SES (parent income at Wave I and parent educational attainment at Wave I) and I find that the disparities in UN between whites and Hispanics, blacks, or Native Americans are slightly attenuated (OR=1.23, $p<0.05$; OR=1.14, $p<0.05$; and OR=1.87, $p<0.001$, respectively), but retain significance. Thus, hypothesis 2 is still supported. Examining the adolescent SES variables I find that respondents whose parents' household incomes were in the middle or highest income groups in their adolescence had lower log odds of UN compared to those whose parents' household income were in the lowest tercile. Parent education, however, has no significant association to log odds of UN in model 2. Additional analyses indicate that the association between parental education and UN is accounted for by adolescent household income (results not shown). Hypothesis 2a states that higher adolescent SES is associated with lower likelihood of UN and is partially supported by the significant association that adolescent household income has on UN (OR=0.878, $p<0.05$ for the middle tercile and OR=0.847 for the highest tercile). Hypothesis 2b states that the relationship between racial/ethnic minorities and whites for UN will be accounted for by adolescent SES. Hypothesis 2b is partially supported as indicated by the attenuation on racial/ethnic disparities once controls for adolescent SES, specifically parent income, are included in the model. For example, in model 1 the odds ratio for Hispanics is 1.28 ($p<0.01$) and in model 2 the OR is 1.23 ($p<0.05$). See table 7 for OR changes of all groups.

Model 3 includes the measures of adult milestones (college education, marriage, and health insurance coverage) and health measures (SRH, depressive symptomatology,

smoking, and drinking). These variables allow me to address the research question of to what extent adolescent SES influences the relationship between race/ethnicity and UN after accounting for events and health statuses found in literature. Examining the race/ethnicity variables, I find that the racial disparity in UN between blacks and whites still exists with the addition of adult milestones and health measures (OR=1.17, $p<0.05$). The disparity in UN between whites and Native Americans also remains significant despite being partially attenuated by adult milestones and health measures (Native Americans OR=1.79, $p<0.001$), supporting hypothesis 2 for these groups. However, the racial disparity in UN between whites and Hispanics is fully attenuated in model 3, but not by adolescent SES, which loses significance in all categories. The coefficient of the middle parent income group changes from OR=0.878; $p<0.05$ to OR=0.905; $p>0.05$, and the highest parent income group coefficient changes from OR=.847, $p<0.05$ to OR=.896, $p>0.05$. These changes suggest that the effect of adolescent SES on UN is fully accounted for with the addition of adult milestones and health measures. Hypothesis 2a is no longer supported because adolescent SES appears to have no significant relationship to UN. This also means hypothesis 2b is not supported and adult milestones and health measures have greater influence over the disparity between some racial/ethnic groups and whites.

In the final model I find that the control for having insurance coverage compared to not having insurance coverage is still a significant protective factor against UN (OR=0.712, $p<0.001$). The adult milestone variables suggest that marriage is associated with reduced log odds of UN (OR=0.877, $p<0.05$). Respondents ever attending college was not associated with UN in model 3, but a model build-up of adult milestones with the base model suggests that adolescent SES attenuates the effects of educational attainment

to reduce log odds of UN (results now shown). Regarding health measures those with changes in SRH over time are associated with a lower likelihood of having worse UN and a greater likelihood of having better UN relative to SRH staying the same (OR=0.673, $p<0.001$). Those with higher depressive symptomatology scores were over 2.5 times as likely to have UN as those with lower scores (OR=2.65, $p<0.001$). Changing from never being a smoker to ever smoking regularly is associated with higher likelihood of worse UN (OR=1.25, $p<0.001$). Finally, both infrequent drinkers and heavy drinkers were at increased log odds of UN compared to those who never reported being drunk in a previous year (OR=1.22, $p<0.001$ and OR=1.38, $p<0.001$, respectively).

An additional model included interactions of race/ethnicity*age and race/ethnicity*quadratic of age to examine whether racial disparities in the log odds of UN increased or decreased as individuals aged from adolescence to young adulthood. The only significant interaction was black*age, suggesting that the effect of age varies between blacks and whites for UN. See Figure 5 for a visual of this relationship, where I use the total predicted exponential to visualize the interactions. I note that blacks begin with higher log odds of UN than whites in adolescence, and while UN increases over time for both groups, the increase for whites is more rapid than for blacks. I will address this finding in further detail in the discussion. All other racial/ethnic groups showed similar change in UN over time. The conclusions for the model results for LA1 are as follows: hypothesis 1, the increase in UN over time, is supported in all models shown; hypothesis 2, that racial/ethnic disparities in UN would persist across models, was supported for blacks and Native Americans in all models. Adult milestones and health measures fully accounted for the disparity between Hispanics and whites. Hypothesis 2a,

that higher adolescent SES is associated with reduced log odds of UN, was supported in model 2, but in model 3 adult milestones and health behaviors fully account for this association. Similarly, due to the full attenuation of adolescent SES in model 3, hypothesis 2b, that adolescent SES accounts for racial/ethnic disparities in UN, is not supported.

Cross-sectional Analysis 1 Results

Descriptive statistics by UN for CA1 are presented in Table 3. Table 5 presents unadjusted bivariate associations between model variables and UN in Wave IV. The total sample size for the cross-sectional analysis is 5,895. To ensure results are nationally representative, I used subpopulation measures with the *svy:* command in Stata for descriptive statistics (Chen and Chantala 2014). The percentages in the table come from results of s*vy, subpop(samp2): prop* or *svy, subpop(samp2): mean* commands. Roughly 24% of young adults have UN in Wave IV. The average age is about 29. Most respondents are insured (81%), but among those with UN the insurance rate is lower (66%). Table 8 displays the model build-up of CA1. Model 1 includes age, race/ethnicity, gender, foreign-born parent, insurance coverage, marital status and adult SES (household income, household assets, household debts, homeownership, and college education attainment). Model 1 tests hypothesis 3, that higher adult SES is associated with lower log odds of UN in adulthood. Model 2 includes adolescent SES and tests hypothesis 3a, that the relationship between adult SES and UN is partially accounted for by adolescent SES. Finally, model 3 includes health measures (SRH, depressive symptomatology, smoking, and drinking), diagnosed health conditions (asthma, depression, anxiety, and migraines), and biomarkers (blood pressure class and BMI class).

In model 1, the highest adult SES markers are associated with reduced log odds of UN compared to lower adult SES markers. Young adults in the highest income group had reduced log odds of UN compared to those in the lowest income group (OR=0.628, $p<0.01$). For every one unit increase in household assets the log odds of UN decrease by 0.923 ($p<0.01$). Those who attended college in the traditional age range of 18-21 have reduced log odds of UN as well compared to their never college-educated peers (OR=.699, $p<0.001$). These results suggest support for hypothesis 3, that higher adult SES is associated with lower likelihood of UN. The control for insurance coverage is also associated with lower log odds of UN compared to those without insurance coverage (OR=0.46, $p<0.001$).

Model 2 results suggest that there is no statistically significant relationship between adolescent SES and UN. Thus, hypothesis 3a is not supported. However, of note is that adolescent SES slightly attenuates the relationship between insurance coverage and UN, the highest income group and UN, and early college attenders and UN (see Table 8). This suggests that adolescent SES has some influence over adult SES attainment. Finally, model 3 tests the influence of health measures, diagnosed health conditions, and biomarkers on the relationship between adult SES and UN. This is the only model in which gender is significantly associated with UN. Women have reduced log odds of UN compared to men (OR=0.797, $p<0.05$) when health measures, conditions, and biomarkers are considered. With the addition of these variables, the relationships between household assets and UN and respondent college education and UN goes away. The highest adult income group is the only adult SES variable that is still significantly associated with lower log odds of UN when compared to the lowest income group, and it

is partially attenuated by the health variables. The potential meanings for these results will be reviewed in the discussion. These results suggest that hypothesis 3b is supported.

While neither of the biomarkers are significantly associated with UN, respondents who have ever been diagnosed with asthma have 1.33 ($p<0.05$) greater log odds of experiencing UN than those without an asthma diagnosis. Similarly, those who have ever been diagnosed with depression have 1.29 ($p<0.05$) greater log odds of experiencing UN than those without a depression diagnosis. The health measures I use in other analyses have similar associations in the cross-sectional models. Early infrequent drinkers and late heavy drinkers both have higher log odds of UN (OR=1.45, $p<0.01$ and OR=1.32, $p<0.05$, respectively). Those with higher depressive symptomatology have over 2 times the log odds ($p<0.001$) of having UN, while those with excellent SRH have almost half the odds ($p<0.001$) of UN compared to those who see themselves as less healthy. Biomarkers of blood pressure and BMI category were not associated with UN.

In conclusion of the cross-sectional analysis results, higher adult SES is associated with lower odds of UN, supporting hypothesis 3. Hypothesis 3a, that the relationship between adult SES and UN is partially accounted for by adolescent SES, is not supported as no adolescent SES variables have a statistically significant relationship to UN. Finally, the addition of health measures, conditions, and biomarkers showed that the relationship between adult SES and UN is partially accounted for by these measures. Those with the most privileged SES statuses appear to be the most protected from UN, which I will unpack further in the discussion. I will also unpack the relationship of the timing of life events with UN, which I could only assess cross-sectionally.

Longitudinal Analysis 2 Results

Descriptive statistics of the total sample of LA2 are presented in Table 2. Table 6 presents unadjusted bivariate associations between model variables and SRH over time. The total sample sizes for LA2 is also 27,981. To ensure results are nationally representative, I used subpopulation measures with the *svy:* command in Stata for descriptive statistics (Chen and Chantala 2014). The percentages and means in Table 2 come from results of s*vy, subpop(samp2): prop* or *svy, subpop(samp2): mean* commands. Select percentage results of excellent SRH over waves I-IV are described below but not visualized. In Wave I, excellent SRH is reported among 27.6% of respondents. This rating increases to a high of 31.9% in Wave III, but by Wave IV only 19.1% of respondents rate their health as excellent, when they are between the ages of 24-32. This trend is true for SRH by all racial/ethnic groups as well. In Waves I-III, when respondents are aging from adolescence to traditional college age, excellent SRH increases steadily for all racial/ethnic groups. Then, in Wave IV, there is a sharp drop-off of those who rate their health as excellent. For whites, excellent SRH decreases from 31.1% in Wave III to 20% in Wave IV. The percentage of those rating their health as excellent decreases by about half for Hispanics, blacks, Native Americans, and others from Wave III to Wave IV.

Adolescent SES also demonstrates the fluctuation of SRH in the transition from adolescence to young adulthood. Those with the lowest earning parents start out similar to middle income respondents for excellent SRH (30.7% compared to 31.2%, respectively). Both income groups make slow gains in excellent SRH across waves, but by wave IV they diverge. Excellent SRH drops to 27% for the low-income group while it

increases to 35% for the middle-income group. In comparison, the highest income group remains stable for excellent SRH across all waves, starting at 38% in wave I and ending at 38% in wave IV. The disparity for parent education groups is larger. Only 12% of respondents with parents who have less than a high school education report excellent health, compared to 41% of respondents with high school educated parents and 47% of respondents with college educated parents. Finally, those who report UN are less likely to rate their health as excellent compared to those who do not have UN. By wave IV, only 15% of those with UN also report excellent SRH.

Table 9 displays the model build-up of LA2. These models test the following hypotheses: 4) that excellent SRH decreases over time (model 1); 4a) that whites have better SRH than racial/ethnic minorities (model 1); 4b) that the relationship between SRH and racial/ethnic minorities is partially explained by adolescent SES (model 2); and 4c) that the relationship between SRH and racial/ethnic minorities is partially explained by UN (model 3). Model 1 includes of age, race/ethnicity, gender, parent nativity, adult milestones (respondent college education attainment, marriage, and insurance coverage), and health measures (depressive symptomatology, smoking habits, and drinking habits). Model 2 adds adolescent SES (parent income and parent education attainment in Wave I). Model 3 is model 2 plus UN. I ran full models with age interactions, however similar to results from the bivariate analyses, the impact of age on SRH did not vary by race/ethnicity.

Reference Figure 6, constructed from centered age and quadratic age values, which shows the exponentiated effect of age for the trend in excellent SRH over time. Note that odds of reporting excellent SRH slightly increases steadily between the teens

and early twenties, approximately between Waves I-III, then begins to decline. By 2004-05 when respondents are reaching their mid-twenties, respondents have reduced odds (<1.0) of reporting excellent SRH. By Wave IV data collection, when most respondents are in their mid-twenties or later, odds of reporting excellent SRH decreases rapidly compared to adolescence. I interpret these findings as support for hypothesis 4, as excellent SRH has declined by young adulthood/Wave IV. There is no significant association for any other racial/ethnic group compared to whites, so hypothesis 4a is not supported in model 1.Women have lower log odds of excellent SRH than men (OR=0.745, $p<0.001$), as do respondents who have higher depressive symptomatology scores compared to those with lower scores (OR=0.392, $p<0.001$). Ever smoking is associated with reduced log odds of excellent SRH (OR=0.617, $p<0.001$). Heavy drinking decreases log odds of excellent SRH when compared to people who never drink heavily (OR=0.877, $p<0.05$). Those with a foreign-born parent have 1.26 times ($p<0.05$) greater log odds of having excellent SRH health compared to those with native born parents. Respondents who attend college have 1.17 higher log odds of excellent SRH ($p<0.01$). Insurance coverage is associated with higher log odds of SRH (OR=1.16, $p<0.05$) compared to not having insurance coverage.

Model 2 includes adolescent SES (parent education and parent income in Wave I) to test hypothesis 4b, that adolescent SES partially accounts for disparities by race/ethnicity for SRH. Results of model 2 suggest that those with the highest adolescent SES benefit via increased odds of excellent SRH. Blacks now have significantly higher odds of having excellent SRH compared to whites (OR=1.18, $p<0.05$). Those whose parents have a college education have 1.25 times ($p<0.01$) higher log odds of excellent

SRH compared to those whose parents have lower educational attainment. Those whose parents are in the highest income group have increased log odds of excellent SRH compared to those whose parents are in the lowest income group (OR=1.21, $p<0.01$). Hypothesis 4, that the likelihood of excellent SRH decreases over time, is still supported (OR=.097, $p<0.001$). Hypothesis 4a, that whites have better SRH than racial/ethnic minorities, is still not supported and adolescent SES suppresses the relationship for excellent SRH for blacks compared to whites (OR=1.18, $p<0.05$). This indicates that if blacks were not burdened by their lower adolescent SES, they would have even better odds of SRH compared to whites. Adolescent SES attenuates the relationship between depressive symptomatology and SRH (OR=0.399, $p<0.001$) and smoking and SRH (OR=0.624, $p<0.001$). Adolescent SES suppresses the relationship between heavy drinking and excellent SRH, however (OR=0.877, $p<0.05$ compared to OR=0.859, $p<0.01$).

The full model allows me to test hypothesis 4c, that UN partially explains the relationship between race/ethnicity and SRH. UN is associated with lower log odds of excellent SRH (OR=0.7, $p<0.001$), but it does not account for the relationship between race/ethnicity and SRH as odds ratios remain relatively unchanged. Thus, hypothesis 4c is not supported. However, UN does further partially attenuate the relationship of higher depressive symptomatology and ever being a smoker on SRH. UN also further suppresses the relationship between blacks and whites for excellent SRH.

To conclude results for LA2 I will summarize support for hypotheses 4-4c. Hypothesis 4, that the log odds of excellent SRH decreases over time, is supported in the full model. Hypothesis 4a, that whites have better SRH than racial/ethnic minorities, is

not supported. Of note, blacks have better log odds of excellent SRH than whites in all models. Hypothesis 4b, that the relationship between race/ethnicity and SRH is partially accounted for by adolescent SES, and Hypothesis 4c, that the relationship between race/ethnicity and SRH is partially accounted for by UN, are irrelevant as Hypothesis 4a is not supported. While having UN is significantly associated with lower odds of excellent SRH, it does not affect the relationship between race/ethnicity and SRH. Rather, adolescent SES explains the relationship between race/ethnicity and SRH better.

DISCUSSION

This dissertation addressed three research aims:

Aim 1: Examine the trends in UN over time from adolescence to young adulthood.

Aim 2: Examine the association between adolescent SES and adult SES in relation to

UN.

Aim 3: Examine the trends in health over time and UN from adolescence to young adulthood.

Aim 1 is paramount to establishing the role of UN in the transition from adolescence to young adulthood, which has previously not been studied longitudinally with sociological theory. Results of LA1 suggest that log odds of experiencing UN increases during the transition as expected (hypothesis 1). This trend persists despite health insurance coverage, suggesting that common markers of health care access do not explain patterns of UN among younger people. This trend is in line with prior literature showing that insurance coverage alone does not address health care access needs and disparities, though my study did show that insurance coverage was protective against UN to some degree (Park et al. 2014). These results motivate the need to further assess what social determinants of health may influence UN in the transition.

Hypothesis 2, that racial/ethnic minorities would have higher odds of UN compared to whites, assesses this need under CAD. Hispanics, blacks, and Native

Americans begin with higher log odds of UN compared to whites. However, in the full model health measures account for the relationship for Hispanics and UN compared to whites, suggesting that health measures account for the disparity between whites and Hispanics. These results suggest that accumulation of negative health measures like smoking and high depressive symptomatology would support CAD in this study. In future research, the Latinx Health Paradox could provide further context for these results. Researchers have established that even though Hispanic immigrant populations in the U.S. often have disproportionately low SES, their health outcomes outpace other native-born groups including native born Hispanics (Dubowitz, Bates and Acevedo-Garcia 2010). This would support the finding that having a foreign-born parent was not associated with UN in any model in LA1. This could be due to at least two reasons: cultural capital differences in health beliefs or better human capital via health and thus lower UN. Since CAM is prevalent in Hispanic enclaves their health needs may be addressed outside of the traditional health care setting. Further, the paradox suggests that part of the reason foreign-born Latinx immigrants are healthier than natives is due to a selection effect of healthier people being those able to migrate in the first place (Dubowitz et al. 2010). Health is one of the most important types of human capital an immigrant can possess, particularly if they are not highly educated. Healthier people tend to immigrate to the U.S., and that health advantage declines as assimilation occurs for later generations. In future research, a comparison of Hispanics with a foreign-born parent to Hispanics with native-born parents could assess the effects of assimilation. Therefore, future studies with Add Health could further differentiate Hispanic native-

born and Hispanic foreign-born population to test for differences in UN risk and differences in predictors of UN.

The black/white and Native American/white disparities in UN were not explained by health measures or adult milestones like marriage. The disparity between these two groups compared to whites for UN persists in the full model in LA1. The reason the disparity is not attenuated after health measures and adult milestones are considered could be because blacks and Native Americans simply achieved lower levels of human capital (e.g., educational attainment, good health) compared to whites or that human capital achievements do not provide the same benefits for these groups compared to whites regarding UN. Regarding SES relationships, in unpublished results I found that adolescent SES attenuates the effects of educational attainment to reduce log odds of UN, suggesting that adult SES disparities in UN reflect adolescent disparities in UN.

Since this is a longitudinal study of a specific time period in the life course, I have an interest in whether the relationship between race/ethnicity and UN varies by age. Interactions assessing these relationships suggest that, when compared to whites, blacks have higher log odds of UN beginning in adolescence and a stable risk of UN throughout young adulthood. Whites, however, do not see spikes in log odds of UN until post-traditional college years in later waves (see Table 8). The results of this interaction indicate that blacks are less likely to receive the same advantages of being under parent care and responsibility in adolescence as whites for having health care needs met. Further, it suggests the weathering hypothesis manifesting at younger ages for UN. The weathering hypothesis posits that the effects of systemic racism, lifetime discrimination, and less advantage from SES begin to damage blacks' health in various ways at earlier

times in the life course than for whites (Geronimus et al. 2006). For example, black teens birth healthier babies than black women in their late twenties and early thirties, which is not the case for white mothers (Geronimus 1992; Geronimus 1996; Buescher and Mittal 2006). It stands to reason that blacks may experience UN at higher rates than whites at earlier ages as well. The stability of the relationship for blacks as they transition from adolescence to young adulthood shows that the effects of weathering plateau and a standard of UN is attained. For whites, UN may be a new experience post-adolescence and explains the spike that exceeds the log odds of UN for blacks in later waves. Finally, this figure also supports FCT where race/ethnicity and the effects of systemic racism contributes to health disparity in the form of UN.

Adult milestones and health measures are in LA1's later models because of varied information found in theory and literature. I introduce the idea of adult milestones based off LCP to emphasize that there are statuses and achievements common across the life course that impact health, such as educational attainment and marriage. Indeed, results show that both early college attendance (ages 18-21) and having ever been married are associated with lower log odds of UN. Future research could determine if marriage is especially beneficial for men in this area, as women commonly take on responsibility for their spouse's health (Allen, Griffith and Gaines 2013). The relationship between marriage and UN seen in my results suggests that marriage may increase access to health care, perhaps through greater access to insurance coverage or financial resources. In line with Ford et al.'s (1999) cross-sectional work looking at UN in wave I Add Health respondents, I also find that health measures such as smoking and drinking increase the likelihood of experiencing UN. I also assess SRH and depressive symptomatology in

order to address perceived health and mental health status. Not surprisingly, respondents who rate their health as excellent are less likely to have experienced UN, and those with higher depressive symptomatology are more likely to have experienced UN. The relationship of higher depressive symptom scores with higher likelihood of UN is of particular interest as mental health issues are increasing in younger cohorts of adults (Patel, Flisher, Hetrick et al. 2007; Hunt and Eisenberg 2010). It may be that UN is either a symptom or a contributing factor to unaddressed mental health issues.

Aim 2 allows me to assess the role of FCT via adolescent SES and adult SES, and I incorporated a cross-sectional analysis in this dissertation for two reasons. The first reason was to dissect the importance of timing of life course events and statuses in relation to UN. The second reason was to understand the influence of adolescent SES on adult SES in relation to UN. This approach recognizes the timing principle of LCP, which suggests that the timing of events across the life course like education can affect outcomes in different ways, (e.g., income and wealth attainment) which influence health outcomes such as UN. This approach also recognizes that understanding the influence of adolescent SES on adult SES in relation to UN addresses fundamental cause of health disparity (Link and Phelan 1995). As low SES is considered a fundamental cause to health disparity over the life course, it is important to dissect if higher adult SES attainment can make up for low adolescent SES regarding UN experience. Further, there are health variables only available in Wave IV that I thought could provide more context for predictors of UN experience in young adulthood, such as having a diagnosed health condition or unhealthy biomarkers. These measures, though limited to cross-sectional examination, can provide context for how poor health affects UN. People with chronic

conditions, for example, have more reason to be regularly connected to the health care system compared to those without chronic conditions, and thus have more opportunities for needs to go unmet. SES could further prohibit those with poorer health statuses from getting health care when needed, harkening back to FCT and CAD.

The first model in CA1 provided support for hypothesis 3, that higher adult SES is associated with lower log odds of UN. Specifically, those with the highest SES in terms of household income and assets had lower log odds of UN compared to those with the lowest household income and fewer assets. Worth noting is that debt is not associated with UN. This may represent a shift in the social-historical context of this cohort's life course. This age cohort is one of the first to take on considerable debt in student loans to pay for college as tuition costs began to inflate (Draut and Silva 2004). Respondents may have larger debts than previous cohorts but human capital via education and income potential help offset other risks of UN. This also reinforces that those with the highest SES would have the greatest protection against UN. Moreover, I found that those who attended college within the traditional time frame (ages 18-21) were the only education group to have reduced log odds of UN compared to those who never attended college in models that did not include health measures or health conditions. Those who attended college later in the life course did not receive this benefit. Thus, in this study I found that timing of college education is important before health measures and health conditions are considered. This finding supports the timing principle of LCP. The benefit early college attenders receive may be a function of SES, such that those with higher SES do not have to delay college until later in the life course. Further, early college attenders would begin to reap multiplicative benefits and advantages under CAD before people who do

eventually attend college later. The compounding of these benefits and advantages may partially explain why the highest adult SES markers were consistently significant in reducing log odds of UN.

Hypothesis 3a, that the relationship between adult SES and UN will be partially accounted for by adolescent SES, was not supported. However, I found that adolescent SES does have some influence over adult SES. This finding in the cross-sectional analysis supports SES as a fundamental cause of health disparity because the strength of the highest adult SES predictors in relation to UN (i.e., highest income group and early college attendance) are slightly attenuated by adolescent SES. I find that those with the highest adult income continue to have lower log odds of UN compared to those with the lowest income even with addition of health measures, conditions, and biomarkers. Those with more human capital are thus more protected against UN, and this is not due to the greater likelihood of higher-SES individuals having better health. Here I also consider the effects of cumulative advantages at play. Hypothesis 3b, that the relationship between adult SES and UN will be partially accounted for by health conditions and biomarkers, is supported for the health conditions of asthma and depression. These relationships exist despite controls for adult and adolescent SES, which may exhibit other structural or behavioral reasons for UN in these groups related to their health condition. Lack of motivation and isolation are common side effects of depression, and the thought of making and attending a doctor's appointment may prove arduous to someone in a depressive episode, for example (Matthews et al. 2016; Wilson, Rickwood and Deane 2007).

Regarding timing and health, I find that those who become heavy drinkers for the first time later in the transition from adolescence to young adulthood (ages 21 or older) have higher log odds of UN. This tracks with health literature on substance use peaking in young adulthood (Park et al. 2006). The same is true for those who become infrequently drunk over the course of a year early in the transition (ages 12 to 20), though why underage people who only sometimes get drunk are more likely to have UN and those who are underage and heavy drinkers do not is difficult to parse out. In future research it may be more efficient to only compare heavy drinkers to everyone else. The results of CA1 established that there are elements of FCT in the transition from adolescence to young adulthood for UN. Note that this applies only to SES in CA1, not to racial/ethnic arguments of FCT (Williams and Collins 2001).

Aim 3 examined trends in health over time with consideration for UN in the transition from adolescence to young adulthood. As such, the trends I saw for health diverged from some trends that I found in LA1 where UN was the outcome variable. Since LA2 was about health I included the same health measures from LA1 (depressive symptoms score, smoking, and drinking) in the first model of LA2. I also included adult milestones (ever married and ever attended college) to determine what influence they had over SRH at baseline. Negative health measures such as higher depressive symptomatology, ever being a regular smoker, and ever being a heavy drinker are all associated with reduced log odds of excellent SRH in LA2. However, the full model with UN lessens the negative effects of smoking and depressive symptoms on SRH, suggesting that part of the reason that these poor health behaviors result in poor health is

because of their greater association with UN. Odds of excellent SRH health does decline over time, supporting hypothesis 4 (see Figure 6).

I find that women are less likely to rate their health as excellent compared to men, regardless of adolescent SES or UN status. This finding suggests a cultural capital difference between men and women for health evaluation even at younger ages. Boys and girls go through different changes at different rates in adolescence such as more fat gain for girls, which may affect health perception differences (Solorzano and McCartney 2010). Boys are also more likely than girls to be more satisfied with their bodies during puberty while girls are less satisfied with their bodies (Bearman et al. 2006). Girls are also more heavily socialized to critique their bodies beginning at earlier ages than boys (Vander Wal and Thelen 2000).

However, hypotheses 4a, 4b, and 4c are not supported. Blacks are actually more likely than whites to have excellent SRH when adolescent SES and UN are accounted for, while all other racial/ethnic groups have no association with log odds of excellent SRH compared to whites. SES and UN appear to moderate the relationship between blacks and whites and excellent SRH. Future research could more closely examine this relationship. This may indicate a difference in cultural capital and what good versus bad health means for white cultural groups and black cultural groups. While SRH is a good predictor of actual health, it is not a direct substitute. Further, this may show more evidence for the weathering hypothesis since weathering posits that blacks can have better health and health outcomes at younger ages compared to their white counterparts and lose this advantage later. By middle adulthood the relationship I found may change. I also find potential evidence for the Latinx Health Paradox in LA2's results. Those with a

foreign-born parent have greater odds of excellent SRH throughout the transition compared to those with native-born parents, and this effect strengthens when adolescent SES and UN are accounted for. However, I did not analyze whether the protective effect of having a foreign-born parent is specifically true for Hispanics.

Limitations and Future Research

There are several limitations to this study. Missing data and attrition across waves are a limitation to the dataset. However, Add Health is still one of the best datasets for studying adolescents longitudinally as it is a large, nationally representative sample. I chose to use the consolidated race/ethnicity variable from Add Health. Though this simplified my methodological process it removed nuance for multiracial respondents especially. Due to the study design of Add Health some variables could not be assessed longitudinally in all four waves, such as pregnancy and child bearing. I chose not to include these variables in the final study for this reason, despite how insightful they could have been in analyses. Other variables in Add Health, such as marriage, are measured in ways such that identifying the timing of the event would be impossible without an advanced algorithm. While the timing of marriage would have provided interesting context, I decided that the "ever married" recode would suffice as a control in all analyses. Other variables related to UN, such as the type of health problem that contributed to UN and the reasons respondents did not get care, were also difficult to utilize. Health problem type was only asked in Wave III, so I disregarded it. I conducted bivariate analyses for reason for UN in Waves I-III to provide at least some context to why UN occurred in the study population. Many measures of health were only asked in Wave IV, and could only be included in the cross-sectional analysis. Finally, the only

measure of health care access I had was health insurance coverage. There is no way to know using the survey data alone if respondents have geographically accessible health care options.

Future research in this topic can further explore relationships found in this dissertation. Marriage was associated with lower odds of UN in several models, and the gendered differences in marriage may indicate different benefits for men compared to women. This would also be an opportunity to assess the effects of pregnancy and child bearing on UN for women. Women also had worse SRH than men and a suppressed relationship with depressive symptoms (results not shown). Gender stratified models may provide more answers to these results. Other results not shown from this study indicate future research examining gender. I found that women were less likely to experience UN than men when only health variables were added to the first model, which was not the case in the SES-only models. I interpret this as women being more likely to get care when needed, not necessarily that women are healthier than men at this stage in the life course. Longitudinal results (not shown) suggest that women are much more likely to have depressive symptoms than men, for example. The results of these 3 analyses also indicate a need for future research that includes more indicators of adolescent SES and immigration and nativity status to better piece together the effects of these statuses on UN and health. Other indicators of health care access beyond insurance coverage are also necessary for more informed research in this topic. The region where respondents live can serve as a control to at least identify areas known for rural areas with sparse health care options, such as the Midwest. Finally, to truly assess if the Latinx Paradox is

supported for UN, I would need to run models comparing Hispanics with a foreign-born parent to Hispanics with native born parents and the racial groups, particularly whites.

Conclusion

To conclude this project, in LA1 I find that those with the highest and most advantaged SES are most protected for UN. In LA2 I find a similar pattern pointing to the positive effects of CAD for those with greater privilege and human capital. I find that elements of FCT through SES advantage are apparent in CA1, though adolescent SES does not fully explain adult SES. Human capital in the form of higher education and good health outcomes and behaviors protect against UN and poorer SRH. Cultural capital in the form of racial/ethnic differences support the Latinx Health Paradox in LA1 and LA2. I also find evidence for the weathering hypothesis in blacks' greater exposure to UN earlier in life despite better health at younger ages compared to whites. Some of the health measures (e.g., smoking and drinking) found to predict UN cross-sectionally in 1999 (Ford et al. 1999) continue to predict UN across the transition from adolescence to young adulthood regardless of SES. The persistent effects of health measures reinforce the importance of ensuring young adults have access to care and can utilize care when needed. This study is the first to longitudinally assess predictors of UN in the transition from adolescence to adulthood with a nationally representative study. There are many future directions possible in researching unmet need for health care in this population with Add Health data and in future studies.

Policy implications of this project include a need for government entities to better assess what young adults' greatest health care needs are in the transition and how to meet those needs. Literature and this study have indicated that health insurance coverage does

not equate to access and met need, so while the major components of legislation such as the ACA have had some positive impacts on this age population, more needs to be done to encourage health care utilization among young adults. This will become especially important as mental health problems like anxiety and depression continue to grow in younger populations.

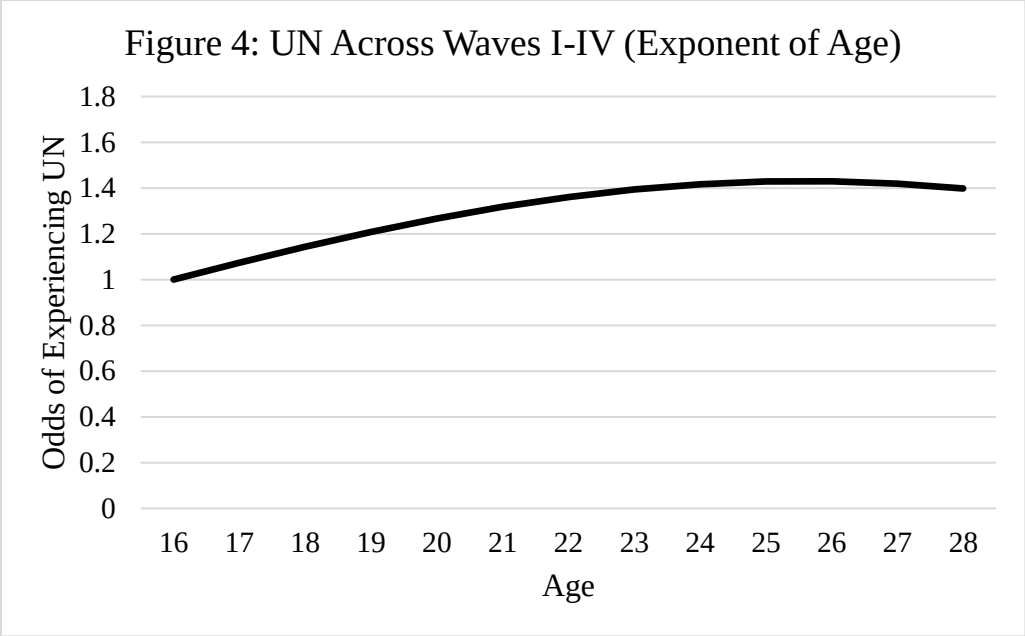
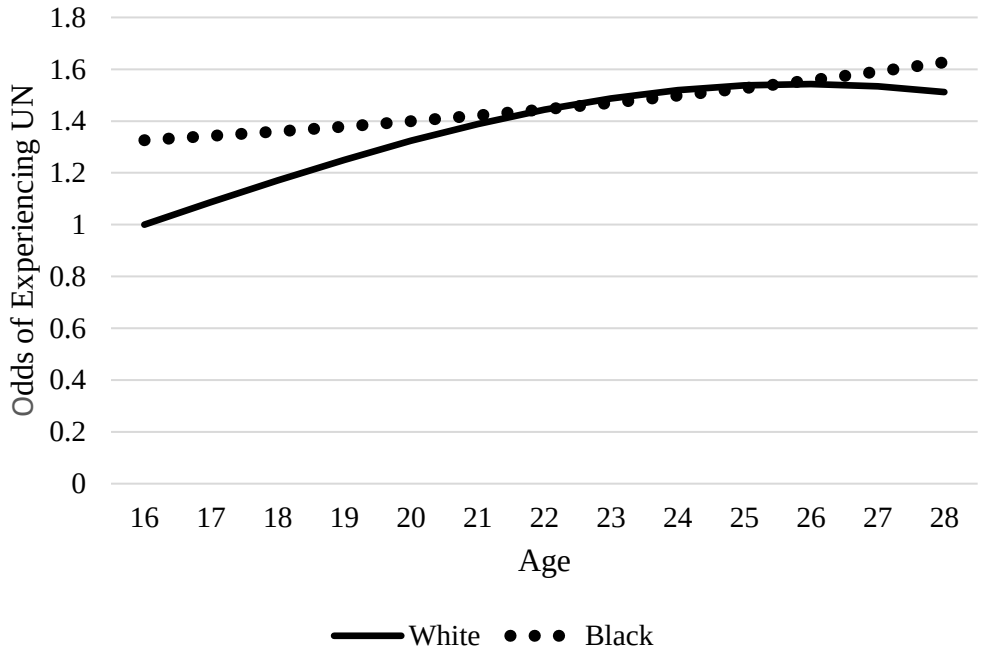


Figure 4: UN Across Waves I-IV (Exponent of Age)
Odds of Experiencing UN
1.8
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
16
17
18
19
20
21
22
23
24
25
26
27
28
Age

Figure 5: Age*Race/Ethnicity for UN Across Waves I-IV



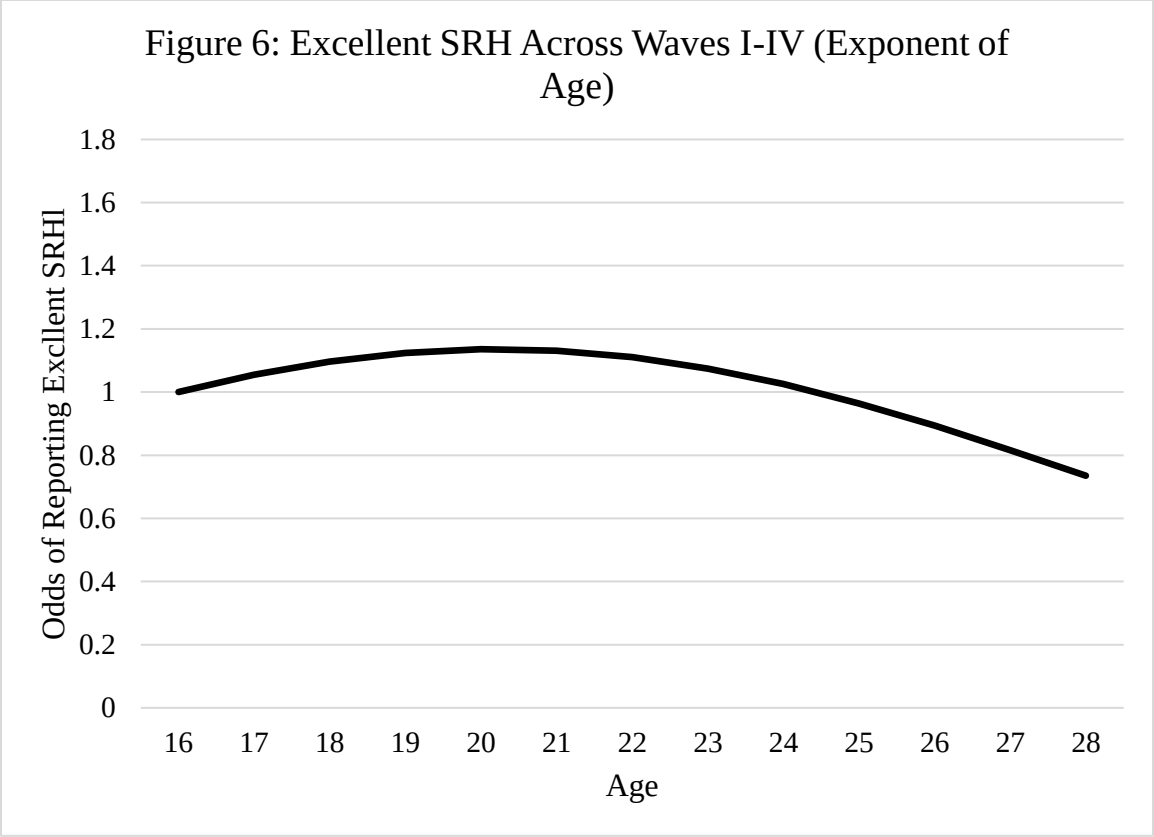


Figure 6: Excellent SRH Across Waves I-IV (Exponent of Age)
Odds of Reporting Exellent SRHI
1.8
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
16
17
18
19
20
21
22
23
24
25
26
27
28
Age

Table 1. Generalized Estimating Equation (GEE) Models Assessing Reason for Unmet Need by Race/Ethnicity in Waves I-III of Add Health (N=5,231)

	Didn't know who to go see	Didn't have transportation	No one available to go along	Parent/Guardian would not go along	Didn't want parents to know	Difficult to make appointment	Afraid of doctor	Thought problem would go away	Couldn't pay	Other reason
	OR	OR	OR	OR	OR	OR	OR	OR	OR	OR
Age (centered)	1.04	1.03	1.0	0.708***	0.936	1.06	0.92*	0.908***	1.39***	1.1
	(0.049)	(0.052)	(0.064)	(0.029)	(0.040)	(0.042)	(0.032)	(0.023)	(0.061)	(0.057)
Age (quadratic)	0.995	0.974**	0.98	0.949*	0.955***	0.997	0.994	0.984***	0.979***	1.0
	(0.007)	(0.009)	(0.011)	(0.021)	(0.100)	(0.006)	(0.006)	(0.004)	(0.005)	(0.007)
Race/Ethnicity (reference =white)										
Hispanic	2.15***	1.38	1.47	0.879*	1.61*	1.26	1.46*	0.905	1.2	1.03
	(0.420)	(0.301)	(0.483)	(0.199)	(0.304)	(0.221)	(0.236)	(0.101)	(0.164)	(0.235)
Black	2.21***	1.89**	1.95*	1.25	1.73**	1.19	1.81***	0.916	0.596***	0.818
	(0.042)	(0.357)	(0.503)	(0.240)	(0.287)	(0.182)	(0.025)	(0.092)	(0.084)	(0.146)
Native American	1.42	0.834	0.97	0.806	2.24*	1.04	1.98**	1.06	0.836	1.35
	(0.534)	(0.349)	(0.750)	(0.305)	(0.695)	(0.337)	(0.506)	(0.224)	(0.239)	(0.470)
Asian	2.85***	1.98*	2.65*	1.59	2.48***	1.39	1.58	0.941	0.747	0.676
	(0.786)	(0.612)	(1.12)	(0.529)	(0.560)	(0.391)	(0.400)	(0.178)	(0.198)	(0.105)
Other	1.49	1.92	6.55**	0.494	2.21	0.996	1.66	0.92	0.864	0.408
	(0.818)	(1.540)	(4.41)	(0.361)	(1.000)	(0.534)	(0.733)	(0.368)	(0.426)	(0.295)

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Table 2. Means and Percentages of the Study Population in Waves I-IV of Add Health (N=27,981)

	Mean or %
Age	20.6
Race/Ethnicity	
White	58.8
Hispanic	14.1
Black	18.3
Native American	2.6
Asian	5.1
Other	1
Female	49.7
Parent foreign born	10.2
Insurance coverage	83.6
Adolescent SES	
Parent Income Wave I	48,000
Parent Education	
Less than high school	15
High school	43.7
Some college or more	41.4
Adult Milestones	
Ever married	46.6
Ever attended college	47.8
Health Measures	
Excellent self-rated health	26.9
Unmet Need	21.2
Depression score (0-3)	0.6
Ever a smoker	33.1
Drinking Habits	
Never drunk in past year WI-WIV	58.3
Infrequently drunk in past year WI-WIV	25.1
Heavy drinking in past year W1-WIV	16.7

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

Table 3. Means and Percentages of the Study Population for Cross-sectional Analysis 1 Using Wave IV of Add Health (N=5,895)

	UN	No UN	Total
	Mean or %	Mean or %	Mean or %
Unmet Need	23.8	76.2	
Age	28.7	28.6	28.7
Race/Ethnicity			
White	69.4	71.7	71.1
Hispanic	10.4	9.8	9.93
Black	13.7	11.8	12.3
Native American	3.4	1.96	2.3
Asian	2.5	3.5	3.3
Other	0.06	1.3	1.1
Female	49.5	52	51.4
Insurance coverage	66.4	85.2	80.7
Ever married	43.6	52.4	50
Adult SES			
Lowest tercile	57.7	39.4	43.8
Middle tercile	22.1	25.3	24.5
Highest tercile	20.2	35.3	31.7
Household assets	3.3	3.9	3.8
Household debts	3.7	3.8	3.8
Owns home	35	48	44.9
College education			
No college	41.1	28.9	31.8
Early college (age 18-21)	38.3	51.8	48.6
Late college (age 22+)	20.6	19.2	19.6
Adolescent SES			
Parent Income Wave I			
Lowest tercile	36.7	27	29.3
Middle tercile	34	34.8	34.6
Highest tercile	29.4	38.2	36.1
Parent Education			
Less than high school	17.5	13.7	14.6
High school	43.5	41.6	52
Some college or more	39.1	44.7	43.3

Table 3. (Continued)

	UN	No UN	Total
	Mean or %	Mean or %	Mean or %
Health Measures			
Excellent self-rated health	11.5	23.2	20.4
Depression score (0-3)	0.762	0.52	0.578
Drinking habits			
Never drunk (WI-WIV)	18.8	23.2	22.2
Early heavy drinking (WI/WII)	4.83	4.8	4.81
Early infrequent drinking (WI/WII)	35.3	31.7	32.5
Late heavy drinking (WIII/WIV)	31.3	31.8	31.6
Late infrequent drinking (WIII/WIV)	9.82	8.54	8.84
Smoking habit onset			
Never a regular smoker (WI-WIV)	40.3	50.9	48.4
Early smoker (WI/WII)	8.46	8.88	8.73
Late smoker (WIII/WIV)	3.85	7.01	6.26
Current smoker (WIV)	47.4	33.2	36.6
Health Conditions			
Asthma diagnosis	20.2	14.6	15.9
Depression diagnosis	24	14	16.4
Anxiety diagnosis	18.7	11.9	13.5
Migraine diagnosis	19	13.4	14.7
Biomarkers			
Blood Pressure Class			
Normal blood pressure	29.4	34	32.9
Pre-hypertensive	50.2	47.2	47.9
Hypertensive	20.3	18.8	19.2
BMI Classification			
Obese	40.1	33.9	35.3
Overweight	27.8	30.3	29.7
Normal weight	30.8	34.4	33.6
Underweight	1.3	1.4	1.37

Note: Percentages may not equal 100 due to rounding

Table 4. Odds Ratios in Bivariate Models Demonstrating Unmet Need Over Time in Waves I-IV of Add Health (N=27,981)

	OR
Age (centered)	
	1.095***
	(0.011)
Age (quadratic)	
	0.995***
	(0.001)
Race/Ethnicity (reference = white)	
Hispanic	
	1.263**
	(0.095)
Black	
	1.265***
	(0.075)
Native American	
	1.959***
	(0.284)
Asian	
	1.177
	(0.145)
Other	
	0.926
	(0.230)
Female	
	0.993
	(0.046)
Parent foreign born	
	1.027
	(0.076)
Insurance coverage	
	0.585***
	(0.031)
Adolescent SES	
Parent Income Wave I (reference = lowest tercile of income)	
Middle tercile	
	0.804***
	(0.045)
Highest tercile	
	0.745***
	(0.042)
Parent Education (reference = less than high school)	
High school	
	0.850*
	(0.059)
Some college or more	
	0.776***
	(0.054)

Table 4. (Continued)

	OR
Adult Milestones	
Ever married	
	1.077
	(0.053)
Ever attended college	
	1.146**
	(0.051)
Health Measures	
Excellent self-rated health	
	0.560***
	(0.027)
Depression score (0-3)	
	2.980***
	(0.136)
Ever a smoker	
	1.697***
	(0.073)
Drinking Habits (reference = never drunk in past year WI-WIV)	
Infrequently drunk in past year WI-WIV	
	1.534***
	(0.067)
Heavy drinking in past year W1-WIV	
	1.577***
	(0.080)

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

Table 5. Odds Ratios in Bivariate Models Demonstrating Unmet Need in Young Adulthood Using Wave IV of Add Health (N=5,895)

	Model 1
	OR
Age (centered)	0.953 (0.024)
Age (quadratic)	0.998 (0.001)
Race/Ethnicity (reference = white)	
Hispanic	1.09 (0.145)
Black	1.19 (0.121)
Native American	1.79* (0.473)
Asian	0.741 (0.187)
Other	0.477 (0.191)
Female	0.905 (0.087)
Insurance coverage	0.344*** (0.034)
Ever married	0.712*** (0.064)
Adult SES	
Household Income Wave I (reference = lowest tercile of income)	
Middle tercile	0.599*** (0.066)
Highest tercile	0.392*** (0.04)
Household assets	0.834*** (0.017)
Household debts	0.963 (0.022)
Owns home	0.583*** (0.053)

Table 5. (Continued)

	OR
College education (reference = no college)	
Early college (age 18-21)	0.52***
	(0.04)
Late college (age 22+)	0.752*
	(0.086)
Adolescent SES	
Parent Income Wave I (reference = lowest tercile of income)	
Middle tercile	0.721***
	(0.065)
Highest tercile	0.568***
	(0.053)
Parent Education (reference = less than high school)	
High school	0.821
	(0.106)
Some college or more	0.687**
	(0.097)
Health Measures	
Excellent self-rated health	0.429***
	(0.057)
Depression score (0-3)	3.04***
	(0.279)
Drinking habits (reference = never drunk in WI-WIV)	
Early heavy drinking (WI/WII)	1.25
	(0.257)
Early infrequent drinking (WI/WII)	1.38**
	(0.141)
Late heavy drinking (WIII/WIV)	1.22*
	(0.117)
Late infrequent drinking (WIII/WIV)	1.42*
	(0.23)

Table 5. (Continued)

	OR
Smoking habit onset (reference = never a regular smoker in WI-WIV)	
Early smoker (WI/WII)	1.2 (0.205)
Late smoker (WIII/WIV)	0.695 (0.158)
Current smoker (WIV)	1.81*** (0.161)
Health Conditions	
Asthma diagnosis	1.48** (0.173)
Depression diagnosis	1.94*** (0.189)
Anxiety diagnosis	1.7*** (0.289)
Migraine diagnosis	1.5*** (0.158)
Biomarkers	
Blood Pressure Class (reference = normal blood pressure)	
Pre-hypertensive	1.23 (0.136)
Hypertensive	1.25 (0.15)
BMI (reference = obese)	
Overweight	0.774** (0.025)
Normal weight	0.754** (0.081)
Underweight	.782 (0.26)

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

Table 6. Odds Ratios in Bivariate Models Demonstrating Excellent Self-rated Health Over Time in Waves I-IV of Add Health (N=27,981)

	OR
Age (centered)	1.040***
	(0.009)
Age (quadratic)	0.994***
	(0.001)
Race/Ethnicity (reference = white)	
Hispanic	0.931
	(0.070)
Black	1.101
	(0.071)
Native American	0.789
	(0.141)
Asian	0.970
	(0.116)
Other	1.210
	(0.280)
Female	0.718***
	(0.034)
Parent foreign born	1.159*
	(0.087)
Insurance coverage	1.283***
	(0.072)

Table 6. (Continued)

	OR
Adult Milestones	
Ever married	0.721*** (0.035)
Ever attended college	0.981 (0.042)
Health Measures	
Depression score	0.366*** (0.021)
Ever a smoker	0.558*** (0.025)
Drinking Habits (reference = never drunk in past year WI-WIV)	
Infrequently drunk in past year	0.833*** (0.035)
Heavy drinking in past year	0.820*** (0.042)

Table 6. (Continued)

	OR
Adolescent SES	
Parent Income Wave I (reference = lowest tercile of income)	
Middle tercile	1.107
	(0.066)
Highest tercile	1.393***
	(0.081)
Parent Education (reference = less than high school)	
High school	1.143
	(0.086)
Some college or more	1.549***
	(0.115)
Unmet Need	0.600***
	(0.027)

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

Table 7. Odds Ratios in Generalized Estimation Equation (GEE) Models Demonstrating Unmet Need Over Time in Waves I-IV of Add Health (N=27,981)

	Model 1	Model 2	Model 3	Model 4
	OR	OR	OR	OR
Age (centered)	1.09*** (0.011)	1.09*** (0.011)	1.08*** (0.013)	1.092*** (0.017)
Age (quadratic)	0.996*** (0.001)	0.996*** (0.001)	0.996*** (0.001)	0.996*** (0.001)
Race/Ethnicity (reference = white)				
Hispanic	1.28** (0.116)	1.23* (0.114)	1.17 (0.104)	1.217 (0.141)
Black	1.2** (0.073)	1.14* (0.071)	1.17* (0.076)	1.326** (0.114)
Native American	1.92*** (0.283)	1.87*** (0.275)	1.79*** (0.267)	2.097*** (0.375)
Asian	1.28 (0.171)	1.31 (0.177)	1.2 (0.129)	1.351 (0.255)
Other	0.955 (0.242)	0.97 (0.244)	0.958 (0.222)	1.437 (0.390)
Female	1.02 (0.047)	1.02 (0.036)	0.914 (0.043)	0.916 (0.043)
Parent foreign born	0.875 (0.084)	0.849 (0.082)	0.945 (0.087)	0.948 (0.087)
Insurance coverage	0.643*** (0.034)	0.663*** (0.036)	0.712*** (0.040)	0.712*** (0.040)

Table 7. (Continued)

	Model 1	Model 2	Model 3	Model 4
	OR	OR	OR	OR
Adolescent SES				
Parent Income Wave I (reference = lowest tercile of income)				
Middle tercile		0.878*	0.905	0.095
		(0.052)	(0.053)	(0.053)
Highest tercile		0.847*	0.896	0.895
		(0.054)	(0.058)	(0.058)
Parent Education (reference = less than high school)				
High school		0.951	1.03	1.029
		(0.070)	(0.076)	(0.076)
Some college or more		0.918	1.06	1.058
		(0.071)	(0.082)	(0.082)
Adult Milestones				
Ever married			0.877	0.867*
			(0.057)	(0.057)
Ever attended college			0.92	0.917
			(0.053)	(0.053)
Health Measures				
Excellent self-rated health			0.673***	0.672***
			(0.034)	(0.034)
Depression score			2.65***	2.647***
			(0.127)	(0.127)

Table 7. (Continued)

	Model 1	Model 2	Model 3	Model 4
	OR	OR	OR	OR
Ever a smoker			1.25*** (0.059)	1.247*** (0.059)
Drinking Habits (reference = never drunk in past year WI-WIV)				
Infrequently drunk in past year WI-WIV			1.22*** (0.064)	1.210*** (0.063)
Heavy drinking in past year W1-WIV			1.38*** (0.076)	1.379*** (0.076)
Age (centered) * Race/ethnicity				
Hispanic				1.002 (0.033)
Black				0.927* (0.028)
Native American				0.991 (0.054)
Asian				0.994 (0.063)
Other				0.912 (0.082)

Table 7. (Continued)

	Model 1	Model 2	Model 3	Model 4
	OR	OR	OR	OR
Age (quadratic) * Race/ethnicity				
Hispanic				0.999
				(0.002)
Black				1.005*
				(0.002)
Native American				0.998
				(0.004)
Asian				0.999
				(0.005)
Other				1.001
				(0.007)

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

Table 8. Odds Ratios in Logistic Regression Models Demonstrating Unmet Need in Young Adulthood Using Wave IV of Add Health (N=5,895)

	Model 1	Model 2	Model 3
	OR	OR	OR
Age (centered)	0.714	0.724	0.729
	(0.284)	(0.286)	(0.286)
Age (quadratic)	1.01	1.01	1.01
	(0.016)	(0.015)	(0.015)
Race/Ethnicity (reference = white)			
Hispanic	0.952	0.939	0.916
	(0.142)	(0.143)	(0.139)
Black	0.944	0.903	.972
	(0.101)	(0.099)	(0.11)
Native American	1.69	1.66	1.67
	(0.521)	(0.515)	(0.552)
Asian	0.774	0.775	0.741
	(0.199)	(0.198)	(0.198)
Other	0.458	0.458	0.456
	(0.186)	(0.185)	(0.187)
Female	0.95	0.949	0.797*
	(0.094)	(0.091)	(0.085)
Parent foreign born	1.21	1.19	1.25
	(0.156)	(0.154)	(0.173)
Insurance coverage	0.46***	0.465***	0.474***
	(0.051)	(0.052)	(0.056)
Ever married	0.876	0.868	0.899
	(0.088)	(0.086)	(0.098)
Adult SES			
Household Income Wave I (reference = lowest tercile of income)			
Middle tercile	0.85	0.856	0.9
	(0.1)	(0.1)	(0.107)
Highest tercile	0.628**	0.637**	0.66**
	(0.082)	(0.085)	(0.09)

Table 8. (Continued)

	Model 1	Model 2	Model 3
	OR	OR	OR
Household assets	0.923**	0.924**	0.949
	(0.023)	(0.023)	(0.024)
Household debts	1.05	1.05	1.03
	(0.028)	(0.028)	(0.028)
Owns home	0.815	0.817 (0.09)	0.888
	(0.09)		(0.098)
College education (reference = no college)			
Early college (age 18-21)	0.699***	0.715**	0.906
	(0.069)	(0.075)	(0.103)
Late college (age 22+)	0.893	0.899	0.995
	(0.107)	(0.112)	(0.124)
Adolescent SES			
Parent Income Wave I (reference = lowest tercile of income)			
Middle tercile		0.883	0.895
		(0.084)	(0.089)
Highest tercile		0.853	0.863
		(0.167	(0.094)
Parent Education (reference = less than high school)			
High school		1.02	1.02
		(0.137)	(0.142)
Some college or more		1.06	1.12
		(0.167)	(0.18)
Health Measures			
Excellent self-rated health			0.583***
			(0.083)

Table 8. (Continued)

	Model 1	Model 2	Model 3
	OR	OR	OR
Depression score (0-3)			2.34***
			(0.241)
Drinking habits (reference = never drunk in WI-WIV)			
Early heavy drinking (WI/WII)			0.935
			(0.232)
Early infrequent drinking (WI/WII)			1.45**
			(0.165)
Late heavy drinking (WIII/WIV)			1.32*
			(0.142)
Late infrequent drinking (WIII/WIV)			1.31
			(0.223)
Smoking habit onset (reference = never a regular smoker in WI-WIV)			
Early smoker (WI/WII)			1.06
			(0.201)
Late smoker (WIII/WIV)			0.583*
			(0.138)
Current smoker (WIV)			1.13
			(0.125)
Health Conditions			
Asthma diagnosis			1.32*
			(0.162)
Depression diagnosis			1.29*
			(0.152)
Anxiety diagnosis			0.99
			(0.137)
Migraine diagnosis			1.21
			(0.149)

Table 8. (Continued)

	Model 1	Model 2	Model 3
	OR	OR	OR
Biomarkers			
Blood Pressure Class (reference = normal blood pressure)			
Pre-hypertensive			1.07 (0.119)
Hypertensive			1.01 (0.136)
BMI (reference = obese)			
Overweight			0.909 (0.086)
Normal weight			0.864 (0.099)
Underweight			0.794 (0.267)

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

Table 9. Odds Ratios in Generalized Estimation Equation (GEE) Models Demonstrating Self-rated Health Over Time in Waves I-IV of Add Health (N=27,981)

	Model 1	Model 2	Model 3
	OR	OR	OR
Age (centered)	1.055*** (0.012)	1.058*** (0.012)	1.062*** (0.012)
Age (quadratic)	0.993*** (0.001)	0.993*** (0.001)	0.993*** (0.001)
Race/Ethnicity (reference = white)			
Hispanic	0.882 (0.075)	0.940 (0.082)	0.948 (0.083)
Black	1.110 (0.075)	1.181* (0.082)	1.191* (0.083)
Native American	0.844 (0.146)	0.881 (0.154)	0.909 (0.159)
Asian	0.847 (0.123)	0.800 (0.115)	0.811 (0.118)
Other	1.097 (0.269)	1.078 (0.266)	1.070 (0.265)
Female	0.745*** (0.035)	0.745*** (0.035)	0.742*** (0.035)
Parent foreign born	1.259* (0.116)	1.308** (0.122)	1.305** (0.122)
Insurance coverage	1.157* (0.007)	1.116 (0.068)	1.099 (0.067)

Table 9. (Continued)

	Model 1	Model 2	Model 3
	OR	OR	OR
Adult Milestones			
Ever married	1.027	1.041	1.031
	(0.065)	(0.066)	(0.066)
Ever attended college	1.166**	1.116*	1.111
	(0.064)	(0.062)	(0.062)
Health Measures			
Depression score	0.392***	0.399***	0.423***
	(0.023)	(0.023)	(0.025)
Ever a smoker	0.617***	0.624***	0.632***
	(0.030)	(0.031)	(0.031)
Drinking Habits (reference = never drunk in past year WI-WIV)			
Infrequently drunk in past year WI-WIV	0.984	0.973	0.980
	(0.050)	(0.049)	(0.050)
Heavy drinking in past year W1-WIV	0.878*	0.859**	0.871*
	(0.048)	(0.047)	(0.048)

Table 9. (Continued)

	Model 1	Model 2	Model 3
	OR	OR	OR
Adolescent SES			
Parent Income Wave I (reference = lowest tercile of income)			
Middle tercile		1.025	1.022
		(0.063)	(0.063)
Highest tercile		1.213**	1.209**
		(0.078)	(0.078)
Parent Education (reference = less than high school)			
High school		1.031	1.037
		(0.082)	(0.083)
Some college or more		1.254**	1.262**
		(0.104)	(0.105)
Unmet Need			0.700***
			(0.034)

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

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APPENDIX

IRB APPROVAL

THE UNIVERSITY OF ALABAMA AT BIRMINGHAM
Office of the Institutional Review Board for Human Use

470 Administration Building
701 20th Street South
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205.934.3789 | Fax 205.934.1301 | irb@uab.edu

APPROVAL LETTER

TO: Rutland, Sarah

FROM: University of Alabama at Birmingham Institutional Review Board
Federalwide Assurance # FWA00005960
IORG Registration # IRB00000196 (IRB 01)
IORG Registration # IRB00000726 (IRB 02)

DATE: 20-Nov-2017

RE: IRB-300000939
Beyond Access: Predictors of Unmet Need for Health Care from Adolescence to Young Adulthood

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The IRB reviewed and approved the Initial Application submitted on 06-Nov-2017 for the above referenced project. The review was conducted in accordance with UAB's Assurance of Compliance approved by the Department of Health and Human Services.

Type of Review: Exempt (Category 4)
Determination: Approved
Approval Date: 20-Nov-2017
Approval Period: No Continuing Review