

THE RELATIONSHIP BETWEEN ADVERSE CHILDHOOD EXPERIENCES, PRETERM
DELIVERY, AND POTENTIAL PROTECTIVE FACTORS:
A LIFECOURSE AND HEALTH EQUITY APPROACH

By

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A DISSERTATION

Submitted to
Michigan State University
in partial fulfillment of the requirements
for the degree of

Epidemiology – Doctor of Philosophy

2022

ABSTRACT

Background & Specific Aims: Preterm delivery (PTD), the birth of an infant occurring prior to 37 weeks of gestation, is one of the leading causes of infant mortality in the United States and of significant concern for child and maternal well-being. Moreover, significant racial and socioeconomic disparities in PTD have persisted for over 30 years. Life stressors are hypothesized as one pathway through which PTD disparities may be influenced. Specifically, adverse childhood experiences (ACEs), adverse life events occurring in the first 18 years of life, are a type of life stressor with repercussions for pregnancy and reproductive health. While foundational research has demonstrated that ACEs can negatively impact pregnancy health and birth outcomes, few studies have **1)** assessed the influence of ACEs on PTD by race and socioeconomic status (SES) subgroups using diverse, nationally representative longitudinal datasets **2)** assessed whether specific ACEs, or co-occurring patterns of certain ACEs, exert greater detrimental effects on PTD risk than others or **3)** examined the role that potential protective factors play to buffer against the risk of ACEs on PTD. These gaps prevent a complete understanding of how the interplay between adverse life stressors and protective factors may influence disparities in PTD. Acknowledging these gaps, this dissertation aims to 1) Determine the association between specific ACEs and PTD and evaluate whether this relationship differs across race and SES; 2) Identify subgroups of women characterized by early life patterns of ACEs and determine the association between subgroup membership and PTD; 3) Examine the role that potential protective factors (i.e., religiosity (R) and spirituality (S)) play in the association between ACEs and PTD, and whether these factors operate differently by race and SES. **Methods:** I used data on n=3,884 and n=3,767 women from the National Longitudinal Study of Adolescent to Adult Health (Add Health; 1994-2018), a nationally representative study

which examines the influences on adolescent health and their development into adulthood. Data on six ACEs (sexual, physical, and emotional abuse; neglect; family member suicide or death; foster care placement), race, SES, R, S, and a composite variable of R and S (R/S), were collected via self-report. I used 1) logistic regression to assess the association between specific ACEs and PTSD and 2) latent class methods to identify underlying classes based on patterns of ACEs, assess the relationship between latent class membership and PTSD, and determine whether R, S, and R/S modified the relationship between latent class membership and PTSD. Results: There were no statistically significant associations between any of the six ACEs and PTSD among women overall. Two latent classes of ACEs were identified (high ACEs and low ACEs) but neither were associated with PTSD. While R, S, and R/S modified the association between latent class membership and PTSD, these factors were not protective against PTSD amongst women in the high ACEs class. Finally, race and SES did not play an overall significant role in the relationships between ACEs, PTSD, R, S, and R/S. Conclusion: This body of work suggests that ACEs may not impact PTSD in hypothesized ways in this sample of women. This work also contributes to an improved understanding of how religiosity and spirituality may not operate equally for all women in all contexts of adversity, highlighting the need to consider adversity thresholds in clinical interventions. Future research should assess how factors such as maternal age and prevalence of ACEs among women who do not have live birth pregnancies may influence the relationship between ACEs and PTSD and disparities by race and socioeconomic status.

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To the brave women and schoolgirls of Iran, and to the men fighting alongside them, in their ongoing battle to liberate themselves from over 40 years of oppression, discrimination, and gender apartheid.

ACKNOWLEDGEMENTS

I was once told that the hallmark of good mentorship is exhibiting selflessness. I am privileged to have mentors and teachers who have been truly selfless with their time, energy, and patience. First and foremost, I would like to thank my dissertation chair and primary advisor, Dr. Claire Margerison, without whom I would not be here today. Her extraordinary mentorship, expertise, and advocacy over these past five years has taught me how to become a better thinker, writer, and researcher, and I am grateful to have developed under her wing. I appreciate how Dr. Margerison genuinely cares not only about her students' academic lives, but their personal lives as well. I encountered multiple obstacles during this PhD journey, but her patience, encouragement, and kindness always saw me through it. Dr. Margerison has shown me the importance of being a good mentor, and I hope to carry this on in the next step of my career.

Furthermore, I would like to thank Dr. Ahnalee Brincks for all the time, expertise, and mentorship she has provided me as I started this long journey of learning about latent class analysis. She always made the time out of her busy schedule to help me troubleshoot, and I appreciate her patiently answering my long emails and the wonderful way in which she explains difficult statistical concepts with such clarity.

Thank you to Dr. Nicole Talge, who has graciously provided me with her mentorship and expertise in developmental psychology and took the time to guide me through an independent study to learn more about the theoretical frameworks of resilience and biological embedding of stress, much of which provided me with the framework to complete this dissertation.

Thank you to Dr. Jaime Slaughter-Acey for her willingness to be a mentor on my committee and provide me with her guidance and expertise. Her foundational work on stress,

resilience, and pregnancy outcomes has been a source of motivation for this research and I am grateful to be able to build upon this work.

I would like to thank Dr. David Barondess for all his mentorship, advocacy, and support over these past five years. I especially appreciate his willingness to guide me through an independent study on the theoretical and historical frameworks of race, providing his wealth of knowledge as a biological anthropologist to help guide my understanding of how the meaning of race has evolved over human history, and which motivated me to think deeply about how best to conceptualize race in this dissertation.

In addition, I would like to thank all the members of the Margerison research group, especially Dr. Colleen Macallum-Bridges, for all their support and constructive feedback on my research. Thank you to all my peers in the program, especially the GEMS/BEAMS group, for their friendship and support.

Last but not least, I would like to thank my family for all their help, patience, and support over these past five years. I want to especially thank my wonderful and loving husband, Jeff, and my mom and dad for all their support and time spent caring for our son over these past few years so I could reach the finish line. Thank you to my brother for his camaraderie, sense of humor, and for always looking at the bright side of things. Thank you to my mom for all the time and energy she has spent throughout these 32 years supporting my academic, professional, and personal endeavors. Thank you to my dad for paving the way as the first person in his family to pursue a PhD and for always emphasizing the importance of education.

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CHAPTER 1: INTRODUCTION

Racial and socioeconomic disparities in preterm delivery (PTD), a birth occurring less than 37 weeks of gestation, have persisted for decades in the United States. The PTD rate for Black women has consistently hovered around 14% since 2008, compared to around 9.0% for White women.^{1,2} Moreover, women with lower socioeconomic status (SES) are at greater risk of delivering preterm.^{3,4}

Preterm birth poses an array of adverse health and socio-emotional consequences for infants, mothers, and their families.^{5–8} Infants born preterm are at increased risk of developing a host of physical, developmental, and neurocognitive conditions that may often be lifelong.^{5,8} Mothers who deliver preterm are at increased risk of suffering postpartum depression.^{9,10} Moreover, PTD represents a significant psychosocial stressor for parents.^{6,7}

An increased burden of life stressors, particularly during the preconception period, is hypothesized as one pathway through which disparities in PTD may be perpetuated.^{11–15} Specifically, adverse childhood experiences (ACEs), defined as adverse life events occurring before the age of 18, have been associated with numerous adverse health outcomes in adulthood.^{16,17} While foundational research has demonstrated that ACEs can negatively impact pregnancy health and birth outcomes,^{14,16,18–21} few studies have **1)** assessed whether specific ACEs, or co-occurring patterns of certain ACEs, may exert greater detrimental effects on PTD risk than others; **2)** assessed racial and SES impacts on the relationship between ACEs and PTD using a health equity framework and diverse, nationally representative datasets; or **3)** analyzed the role that potential protective factors may play to buffer against the risk of ACEs on PTD. These gaps prevent a comprehensive understanding of how the interplay between adverse life stressors and protective factors may influence disparities in PTD by race and SES.

1.1 Study Aims & Hypotheses

Acknowledging these gaps in the literature while building upon the important contributions of previous research, **the overall goal** of this dissertation is to investigate the relationship between adverse childhood experiences, preterm delivery, and two types of potential protective factors (religiosity and spirituality) in a national, longitudinal sample of women using a health equity framework and lifecourse approach. Specifically, I will address this goal through three specific dissertation aims:

- 1) Determine the association between specific adverse childhood experiences and odds of PTD and evaluate whether this relationship differs across race and SES subgroups.**
- 2) Identify subgroups of women characterized by early life patterns of ACEs and determine the association between subgroup membership and odds of PTD.**
- 3) Examine the role that potential protective factors (i.e., religiosity and spirituality) play in the association between adverse childhood experiences and odds of PTD, and whether these factors operate differently by race and SES subgroups.**

1.2 Public Health Significance

There is a growing body of literature examining the impacts of ACEs on adverse birth outcomes, including PTD.^{18,20} Preterm birth, defined as a birth occurring earlier than 37 weeks of gestation, poses a significant public health problem in the United States with a prevalence of 10.5% as of 2021.²² Infants born preterm are at risk for multiple adverse health consequences, which can often be lifelong, including death, cerebral palsy, vision and hearing impairments, gastrointestinal disturbances, infections, developmental delays, behavioral problems such as ADHD, and complications across the cardiovascular, respiratory, and neurocognitive systems.^{5,8}

Moreover, PTSD poses a high burden to society, as evidenced by the estimated \$26.2 billion associated with PTSD-related health care, early intervention, special education, and impacts on labor productivity.⁵

In addition to constituting one of the highest rates globally, the prevalence of PTSD in the United States differs substantially by race, and these disparities have not changed substantially in over 30 years.^{2,23,24} Moreover, PTSD disparities by SES in the United States have been documented since the 1990s.²⁵

Life stressors, including adverse childhood experiences, may contribute to racial and socioeconomic disparities in PTSD.^{11,13,26,27} Research suggests that more than 50% of adults in the United States have experienced at least one ACE, while over 20% have experienced more than three ACEs.²⁸ Importantly, ACE prevalence differs significantly by race/ethnicity and socioeconomic status, whereby multiracial individuals, black individuals, American Indian or Alaska Native, and Hispanic individuals experience higher levels of ACEs compared to white individuals, and individuals with both lower childhood and adulthood SES report higher levels of ACEs.^{28–31}

The detrimental impacts of early childhood adversity on human health and development can be pervasive throughout the lifecourse.^{32–34} Early life stress is hypothesized to “get under the skin,” or become biologically embedded, through disruptions to the structure and functioning of neuroendocrine and immunological systems, increasing an individual’s vulnerability to the development of pathologies.^{32,33,35,36} As the role of preconception health is becoming increasingly recognized in healthy pregnancy and perinatal outcomes, adverse events that occur long before pregnancy have the propensity to influence women’s reproductive health and

subsequent pregnancy outcomes.^{14,19,21,37} This contextualizes the important implications that ACEs may have for women's reproductive, preconception, and pregnancy health.

Indeed, while the literature is in its early stages, ACEs have been found to increase the risk for poor mental health during pregnancy, unintended pregnancies, adverse birth outcomes, pregnancy loss, risky sexual behaviors (i.e., higher number of sexual partners), sexually transmitted diseases, cortisol dysregulation during pregnancy, as well as negative health behaviors during pregnancy such as drinking.^{16,18,20,21,38–42} While ACEs have also been linked to PTSD, the literature is not unanimous^{18,19} and entails methodological limitations that restrict generalizability of findings.

Despite this foundational research, few studies in the literature have **1)** assessed whether specific ACEs, or co-occurring patterns of certain ACEs, may exert greater detrimental effects on PTSD risk than others; **2)** assessed potential differences in the relationship between ACEs and PTSD by race and SES using diverse, national datasets; or **3)** analyzed the role that potential protective factors may play to buffer against the risk of ACEs on PTSD.

Therefore, research that aims to understand the relationship between patterns of ACEs, PTSD, and potential protective factors using a racial and SES health equity lens is important for identification of subgroups of women who may be at high risk for adverse birth outcomes to better inform targeted prevention and development of effective clinical interventions. The **impact of this research** will be to enhance understanding of the role of potential protective factors which may buffer against the effects of ACEs on adverse birth outcomes to lend useful insight in clinical settings and help women “identify, affirm, and build” (Walsh, 2016, p. 140)⁴³ their repertoire of protective factors that can be mobilized to promote healthy pregnancy outcomes, despite experiences of significant life adversity.

CHAPTER 2: BACKGROUND AND LITERATURE REVIEW

2.1 Preterm Delivery

Preterm delivery (PTD), defined as a birth occurring before 37 weeks of gestation, poses a significant public health problem in the United States with a prevalence of 10.5% as of 2021.²² Indeed, the United States is one of the 10 countries with the highest prevalence of PTD in the world, trailing behind Bangladesh, Philippines, Democratic Republic of Congo, and Brazil, a trend that sets it apart from other Western countries.⁴⁴

Preterm birth can result in multiple adverse short and long-term health consequences for mothers, babies, and their families. Specifically, preterm infants have a higher risk of death and developing respiratory, cardiovascular, and neurological complications, gastrointestinal problems, cerebral palsy, vision and hearing impairments, behavioral disorders like ADHD, and developmental delays.^{5,8} These health impacts can often be lifelong. PTD poses a significant psychosocial stressor for parents,^{6,7} and mothers who deliver preterm are at greater risk of experiencing postpartum depression and anxiety.^{9,10} Moreover, the socioeconomic burden associated with PTD, including medical health care costs, early intervention services, and special education services, is estimated to be \$26.2 billion annually in the United States.⁵

While approximately 30% of preterm cases are medically indicated (i.e., as a result of known medical conditions such as preeclampsia), the etiology of the remaining 70% of cases is unknown (i.e., spontaneous), making the prediction of its occurrence, and therefore its prevention, difficult.¹⁵

To date, research has identified multiple factors which may increase a woman's risk of delivery preterm, including a history of delivering preterm, having a short cervix, young maternal age (<16 years old), advanced maternal age (>35 years old), low socioeconomic status, adverse health behaviors including smoking, drinking, and illicit drug use, short interpregnancy

interval, low prepregnancy BMI or obesity, and predisposing medical conditions, including preeclampsia, diabetes, high blood pressure, blood clotting conditions, vaginal infections, sexually transmitted infections.^{5,45}

2.1.1 Racial and Socioeconomic disparities in PTD

Significant disparities in PTD prevalence in the United States exist by race and socioeconomic status (SES).²⁻⁴ Specifically, Black women experience the highest prevalence of preterm birth, at 14.4% of all live births as of 2020, compared to 11.6% for American Indian and Alaskan Native (AIAN) women, 9.8% for Hispanic women, 8.5% for Asian women, and 9.1% for white women.² These racial disparities in PTD have not changed substantially in over 30 years.^{1,2,24}

Furthermore, studies have documented that women with lower socioeconomic status are at greater risk of delivering preterm.^{3,4,46} However, while individual-level occupation and aggregate measures based on census (i.e., poverty, income, education, unemployment, and wealth) are consistently associated with PTD risk, the findings for individual-level socioeconomic measures like education and income are more mixed.³

Moreover, the relationship between socioeconomic factors and risk of PTD varies within race subgroups. For example, census-based socioeconomic measures including neighborhood poverty and unemployment are more commonly associated with higher PTD risk among Black women, but not among White women.³ In terms of individual-level socioeconomic measures, Pickett et al. (2022) found that compared to Black women who had a high school education, Black women with less than a high school education had 2.34 times the odds of spontaneous PTD (OR 2.34, 95% CI 1.15-4.74).⁴⁷ However, this pattern was not found among White

women.⁴⁷ Collectively, these findings indicate that race and socioeconomic status play a prominent role in PTD disparities.

2.2 Hypothesized Explanations for Disparities in Preterm Delivery

While the exact cause of these racial and socioeconomic disparities is unclear, there are multiple hypothesized explanations for the higher rates of PTD seen among Black women and women with low SES, including a disproportionate exposure to life stressors (e.g., adverse life events and racial discrimination), mental health conditions like anxiety and depression, health behaviors (e.g., smoking, alcohol and drug use, diet), & biophysiological conditions (e.g., inflammation and infections).^{11,15,27,48} However, the majority of research on these risk factors has been limited to the period right before, during, or after pregnancy.

A growing body of perinatal research is recognizing the important role of the preconception period as part of a lifespan approach to understanding racial and SES disparities in adverse birth outcomes.^{11,14,37} This approach acknowledges that, as stated by Misra et al. (2003), “...some of the most powerful influences on pregnancy outcomes are related to influences on women’s health that occur long before pregnancy begins” (p. 65).³⁷ While the application of a lifespan approach to perinatal disparities research on adverse birth outcomes has grown in recent decades,^{11,37} developmental psychologists have recognized the effects of early life experiences on developmental outcomes since the 1960s.⁴⁹ Specifically, adverse childhood experiences are a unique type of early life stressor with significant repercussions for biological, psychological, and physical health.^{32–36,50,51} In this body of work, I will be applying this lifespan approach to assess how adverse childhood experiences occurring during the preconception period may contribute to racial and SES disparities in PTD among women of reproductive age.

2.3 Adverse Childhood Experiences: A Unique Life Stressor

Adverse childhood experiences (ACEs), defined as adverse life events occurring before the age of 18, constitute a unique life stressor in that they have been associated with a diverse range of adverse health and societal consequences.^{16,17,52,53} Research suggests that more than 50% of adults in the United States have experienced at least one ACE, while over 20% have experienced more than three ACEs.²⁸

One of the first major studies on ACEs was conducted by the CDC and Kaiser Permanente (1995-1997), which was groundbreaking in discovering that adverse experiences in childhood were associated with a plethora of negative health outcomes in adulthood, including injuries, depression, anxiety, substance use, unintended pregnancy and adverse pregnancy outcomes, infectious diseases, and chronic diseases, underscoring the long-term consequences of ACEs across the lifecourse.^{17,54}

The CDC/Kaiser ACE study identified key domains of ACEs including abuse (e.g., physical, emotional, sexual), neglect (e.g., physical and emotional), and household dysfunction (e.g., substance abuse, mental illness, parental separation, and incarceration) which are known as “traditional ACEs” in the literature.^{16,17} However, these categories of ACEs have been expanded in recent years to include factors such as witnessing violence, experience of discrimination, living in an unsafe neighborhood, experiencing bullying, and living in foster care, in recognition that a diverse array of negative experiences during childhood can be considered adverse.⁵⁵

2.3.1 Sociodemographic Disparities in ACEs

The prevalence of ACEs differs by gender, race, and socioeconomic status. Using BRFSS data on $n = 211,376$ adults from 34 states, Giano et al. (2020) found that women exhibited significantly higher mean ACE scores compared to men (1.64, 95% CI 1.62-1.67 vs. 1.46, 95%

CI 1.44-1.49, respectively).²⁸ They also documented that the mean ACE score for multiracial individuals (2.39, 95% CI 2.26-2.51), Black individuals (1.66, 95% CI 1.60-1.71), and Hispanic individuals (1.63, 95% CI 1.58-1.69) were significantly higher than the mean ACE score for White individuals (1.53, 95% CI 1.51-1.54).²⁸

In terms of socioeconomic status indicators, Giano et al. (2020) found that individuals with less than a high school education had a mean ACE score (1.71, 95% CI 1.66-1.77) that was significantly higher compared to those with a high school diploma or GED (1.57, 95% CI 1.54-1.61) and those with a college degree (1.26, 95% CI 1.24-1.29), but not compared to those with some college (1.70, 95% CI 1.67-1.73).²⁸ Furthermore, unemployed individuals had a mean ACE score (2.05, 95% CI 1.97-2.14) that was significantly higher compared to employed individuals (1.61, 95% CI), whereas those individuals who are unable to work (i.e., due to a disability) had a significantly higher mean ACE score (2.24, 95% CI 2.17-2.31) compared to both unemployed (2.05, 95% CI 1.97-2.14) and employed individuals (1.61, 95% CI 1.59-1.64).²⁸

These socioeconomic patterns also existed by household income. Individuals with a household income of less than \$15,000 had a significantly higher mean ACE score (2.00, 95% CI 1.97-2.06) compared to all higher income categories (\$15,000-\$24,999: 1.73, 95% CI 1.68-1.78; \$25,000-\$34,999: 1.59, 95% CI 1.54-1.65; \$35,000-\$49,999: 1.62, 95% CI 1.56-1.67; >\$50,000: 1.44, 95% CI 1.42-1.47, respectively).²⁸ Confirming these disparities in ACE prevalence by socioeconomic factors during adulthood, a systematic review assessing the relationship between childhood socioeconomic position and adverse childhood experiences (ACEs) worldwide found that lower childhood socioeconomic position (SEP) is associated with a higher risk of experiencing ACEs and maltreatment.²⁹ Together, these studies indicate that vulnerability to ACEs differs importantly across dimensions of race, gender, and SES.

The findings of Giano et al. (2020) are consistent with research using BRFSS (2015-2017) data which also found racial differences in the number of ACEs reported by adults.³⁰ Indeed, Merrick et al. (2019) found that 28.3% of American Indian and Alaska Native adults reported experiencing 4 or more ACEs, compared to 15% of White adults, 17.7% of Black adults, and 8.6% of Asian adults.³⁰ A significantly higher percentage of Black adults, American Indian and Alaska Native adults, and Hispanic adults reported experiencing 4 or more ACEs compared to White adults and Asian adults. Interestingly, a significantly higher percentage of Asian adults reported 0 ACEs (56.3%), compared to all other racial groups.³⁰

While these two studies incorporated large datasets and sample sizes, the data were not from all 50 states (i.e., a sub-selection of states), which may limit generalizability. Furthermore, the data on ACEs were self-reported by adults retrospectively. Although retrospective recall (i.e., self-report) is a common method of collecting data on ACEs, there is always a possibility for potential recall bias compared to assessment via medical records (e.g., provider notes from counseling sessions). Indeed, individuals may underreport adverse experiences in childhood,⁵⁶ especially women,⁵⁷ possibility due to stigma or denial.

Using nationally representative data from the National Survey of Children's Health to assess ACEs experienced by children under 18 as reported by a parent or guardian, Sacks & Murphey (2018) found similar disparities by race, whereby 33% of NH Black children experienced 2 or more ACEs, compared to 19% of white children, 21% Hispanic children, 5% NH Asian children, and 26% of children classified as Other NH.³¹ Together, these findings from the literature provide important evidence that disparities in ACE prevalence exist by sociodemographic factors in the United States.

2.4 Literature on ACEs and Health Outcomes

The impacts of ACEs on human health and development can be pervasive throughout the lifecourse, increasing the risk of mental illness, premature mortality, cardiometabolic diseases, suicide, cancer, and substance abuse during adulthood.^{16,17,32,52} Indeed, ACEs have been linked to 5 of the 10 primary causes of death.⁵² It is estimated that prevention of ACEs would decrease cases of depression by up to 21 million, heart disease by up to 1.9 million, and cases of overweight and obesity by up to 2.5 million.⁵² Furthermore, prevention of ACEs is estimated to reduce the occurrence of coronary heart disease by 13%, cancer by 6%, smoking by 33%, and heavy drinking by 24%.⁵²

Further emphasizing the wide reach that ACEs have on a diverse array of health outcomes, a systematic review by Petruccelli et al. (2019) which assessed the association between ACEs and various health outcomes found statistically significant increased odds ratios for the majority of 22 examined health outcomes across all number of ACEs (1, 2, 3, 4+, highest number of ACEs reported in each study).¹⁶

While these findings underscore that the health impacts of ACEs occur across diverse dimensions of medical, psychosocial, and behavioral conditions, it is important to highlight a notable gap: pregnancy-related outcomes were not assessed. As exposure to ACEs in childhood and adolescence occurs during women's preconception period, this carries important implications for the potential effects of ACEs on women's reproductive health.

2.4.1 ACEs and Reproductive Health Outcomes

There is a growing body of perinatal literature recognizing the impacts of early life experiences on women's reproductive health outcomes as part of a lifecourse approach to studying factors which may influence preconception health.^{11,14,18,21,37} Indeed, exposure to

adverse life events during the preconception period, not just during pregnancy, has the potential to impact pregnancy health and birth outcomes.^{11,14,18,21,37}

In an integrative review of 17 studies from the literature that assessed ACEs and pregnancy-related outcomes, Olsen (2018) found that ACEs were positively associated with prenatal alcohol use, preterm birth, fetal death, health problems during pregnancy, and prenatal depression and anxiety.²⁰

Among n = 1848 low-income women enrolled in home visiting programs in Wisconsin who self-reported 10 types of ACEs using the Childhood Experiences Survey, Mersky & Lee (2019) found that a higher ACE score was associated with increased odds of experiencing pregnancy loss (OR 1.12, 95% CI 1.08, 1.17), preterm birth (OR 1.07, 95% CI 1.01, 1.12), and low birth weight (OR 1.08, 95% CI 1.03, 1.15).⁵⁸ Moreover, while the mean of observing a pregnancy loss increased continuously as the number of ACEs increased, this relationship did not increase substantially for premature birth or low birthweight until extreme levels of adversity (9-10 ACEs). These findings suggest a potential non-linear threshold effect whereby certain adverse outcomes may not “manifest” (Mersky & Lee, 2019, p. 5) until extremely high levels of adversity are encountered.⁵⁸ This threshold risk model has been proposed by experts in developmental psychology³² and similarly observed in other ACE studies.^{17,59}

However, due to the small sample size used by Mersky & Lee (2019) which was not nationally representative and only included select racial subgroups of women, these results may not be generalizable to other populations.⁵⁸ Nonetheless, findings from larger samples seem to indicate similar magnitude of effects for other reproductive health outcomes. In a sample of n = 8810 women from the National Longitudinal Study of Adolescent to Adult Health, Hall et al. (2019) found that an increase in adverse life experiences (ALE) score by one standard deviation

was associated with an increased risk of unintended first pregnancy (adjusted Hazard Ratio aHR = 1.11, 95% CI 1.04- 1.17) among women overall.³⁹ Higher ALE scores also increased the risk of unintended first pregnancy for Black women (aHR = 1.12, 95% CI 1.01-1.25), Asian women aHR 1.69, 95% CI 1.26-2.26), White women (aHR = 1.12, 95% CI 1.03-1.22), low-income women (aHR 1.21, 95% CI 1.03-1.23), as well as high income women (aHR=1.36, 95% CI 1.12-1.66).³⁹ While this study examined diverse types of adverse life experiences among a large sample of female adolescents, they did not focus exclusively on adverse childhood experiences, nor did they examine impacts on adverse birth outcomes.

In a study of n = 111,330 postpartum women from PRAMS, Koning & Ehrenthal (2019) assessed the impacts of adverse maternal life events one year prior to childbirth (including financial difficulties, loss of job, divorce or separation, unwanted pregnancy by partner, incarceration, and homelessness, as well as intimate partner violence one year prior to conception) on birth weight and premature birth using clustering methods to find groups of women based on similar patterns of life experiences.⁶⁰ Women who were classified under the “toxic/cumulative” subgroup, meaning they experienced frequent and acute adverse life events, had babies who were on average 107 grams lower in birth weight (BW) compared to women in the “protected” subgroup, meaning they experienced few adverse life events. Furthermore, women in the toxic subgroup experienced a 27% increase in preterm birth (PTB) risk, 49% increased risk of low birth weight (LBW), and 57% increased risk of very low birth weight (VLBW).⁶⁰ Disparities were also apparent by race/ethnicity. For example, across all three subgroups of adverse maternal life events (protected, illness/isolated, and toxic), non-Hispanic Black (NHB) women experienced significantly poorer birth outcomes (lower BW, LBW, PTB, & VLBW) compared to non-Hispanic White (NHW) women (p<0.05).⁶⁰

Koning & Ehrental (2019) also calculated the “probability of being born in the toxic stressor landscape,” p(TSL), based on race/ethnicity and income, controlling for sociodemographic factors such as age, education, marital status, and state of residence (p. 6).⁶⁰ While NHW women experienced a precipitous decline in p(TSL) at higher income quartiles, the decline for NHB women and Hispanic women at higher incomes was smaller.⁶⁰ Additionally, while the association between toxic landscapes and LBW was strongest for NHW women in the lower income quartiles, this pattern was not observed for NHB and Hispanic women, who experienced a stronger association between toxic landscapes and LBW in the upper and middle income groups.⁶⁰ While this study assessed adverse life events only one year prior to childbirth (or one year prior to conception) and did not incorporate adverse childhood experiences (ACEs) specifically, these findings suggest that not all adverse life events may impact women equally across all race and socioeconomic groups.

2.4.2 Not all ACEs are created “equal”

While traditionally the majority of ACE studies have operationalized ACE exposures using frequency and count measures,^{16–18,58} there is research need to assess distinctions between different types of ACEs, as not all ACEs may be “equal” in their adverse impact on health outcomes.^{19,61–64} Indeed, some studies have found evidence of a “synergistic” interaction pattern between ACEs, whereby patterns of co-occurring ACEs create an aggregate effect on health outcomes that is much greater than the sum or product of the individual effects of each ACE separately.^{61–63}

For example, Briggs et al. (2021) examined different pairs of 10 ACEs (including sexual abuse, physical abuse, emotional abuse, neglect, exposure to domestic violence, mental health illness among caregivers, medical illness or substance abuse, and parental loss, separation or

bereavement) and found that sexual abuse formed the greatest number of synergistic pairings of ACEs, meaning its co-occurrence with another ACE contributed to a greater proportion of the attributable risk for clinical behavioral problems among children and adolescents than the sum or product of the effects of the ACEs individually.⁶¹ While this study did not examine perinatal outcomes specifically, the findings suggest that certain ACEs may exert greater detrimental impacts on health outcomes than other.

Furthermore, the concept that “not all ACEs are equal” was observed in a study of $n = 2259$ pregnant women from the Pregnancy Outcomes and Community Health Study (POUCH), where Margerison-Zilko et al. (2017) found that sexual abuse during childhood and adulthood was associated with increased odds of PTD (aOR 1.9, 95% CI 1.0, 3.5), but physical abuse was not.¹⁹ Moreover, life stressors (i.e., abuse/witnessing violence, loss, economic stress, and substance use) experienced in the 6 months prior to childbirth was not associated with PTD.¹⁹ These findings suggest that 1) specific ACEs may be more strongly associated with adverse birth outcomes than others and 2) There may be sensitive periods during which specific types of life stressors may exert their impacts on adverse birth outcomes.

2.4.3 Summary of Literature Gaps for Section 2.4

Few studies on ACEs have assessed the influence of adverse childhood experiences on adverse birth outcomes such as PTD with specific attention to disparities by race and SES subgroups using diverse, nationally representative datasets. Moreover, few studies in the perinatal literature have assessed whether specific ACEs, or co-occurring patterns of ACEs, exert greater detrimental effects on PTD risk than others.

2.5 Theoretical Frameworks: Biological Embedding of Life Adversity and Birth Outcomes

Theoretical frameworks across social epidemiology, perinatal epidemiology, and developmental psychology are critical to understanding how ACEs may manifest to impact women's reproductive health outcomes. These frameworks heavily informed my dissertation work and my conceptualization of the hypothesized physiological, behavioral, and psychosocial mechanisms by which ACEs may influence the occurrence of PTD (See Figure 1 in Chapter 3).

2.5.1 Social Determinants of Health and Health Equity

The social determinants of health theory posits that social conditions and societal structures influence the development of disease, health outcomes, as well as disparities in health outcomes.^{65,66} Widely recognized social determinants of health include access to health care, quality of healthcare, education, neighborhood, housing and physical environment, diet and nutrition, social support networks, sex, race/ethnicity, and socioeconomic resources.^{65–68} Importantly, the risk of, and disparities in, preterm delivery, is heavily influenced by social factors, namely, race/ethnicity and socioeconomic status.⁵ Additionally, disparities in the prevalence of adverse childhood experiences exist by race and socioeconomic status.²⁸

Therefore, research which aims to understand racial and socioeconomic influences in the relationship between ACEs and PTD is critical to identifying at-risk subgroups and informing the development of appropriate clinical interventions to reduce disparities and, ultimately, promote health equity. Health equity is “the principle underlying a commitment to reduce—and, ultimately, eliminate—disparities in health and in its determinants, including social determinants” (Braveman, 2014, p. 6).⁶⁹ One way in which my dissertation addresses health equity is by assessing whether disparities in my exposure-outcome relationship exist within race and socioeconomic status subgroups rather than assuming that ACEs and protective factors affect

all women equally. Another way I incorporated health equity is by including seven categories of race. Many studies often exclude groups with smaller sample sizes or lump multiple groups together, but I felt that it was important to take advantage of the diverse sample that Add Health offers and to increase representation of commonly underrepresented groups of women.

2.5.2 Psychosocial Theory

Closely related to social determinants of health, psychosocial theory underscores the importance of the “host-agency-environment” triad in determining health and disease; the goal of this theory is to understand the phenomena of why not all individuals who have a certain exposure develop disease or a certain health outcome, and why some individuals display resistance to disease development (Krieger, 2001, p. 669).⁷⁰

Decades of research from developmental psychology since the 1960s has demonstrated that not all children who experience adversity go on to develop adverse health or developmental outcomes.^{43,49,71–73} In application to the perinatal literature, not all women who experience life stressors either during the preconception period or during pregnancy may necessarily experience adverse birth outcomes,^{14,19,27,74,75} even though adverse life events have been associated with adverse birth outcomes.^{14,18,60,76} Therefore, it is important to determine what may be contributing to the positive pregnancy outcomes observed in these women, in spite of experiences of adversity, so that we may inform interventions to promote healthy pregnancies in other women who may be at-risk due to a history of adverse life events.

Moreover, the susceptibility of an individual to developing adverse health outcomes depends on both environmental factors (i.e., foster care placement) and psychosocial factors (i.e., social support, religiosity). Indeed, certain factors such as social support and religiosity have been found to buffer against adverse birth outcomes for women who have a history of adverse

life experiences.^{74,76–78} My research will examine the effects of two potential protective factors, religiosity and spirituality, on the association between adverse childhood experiences and PTSD to determine whether these factors buffer against the impact of ACEs on PTSD and PTSD disparities.

2.5.3 Lifecourse Theory, Biological Embedding, and the Concept of Sensitive Periods

Lifecourse theory is essential to understanding how exposures at different stages of human development (from birth, childhood, adolescence, to adulthood) accumulate and interplay to continuously impact health status and health outcomes across the lifespan.^{37,65,79} For example, Adler and Stewart (2010) explain the “dynamic relationship between SES and health” throughout the lifecourse, whereby health status during childhood subsequently influences that child’s future health as well as their ability to attain education, income, and occupation later in life (p. 10).⁷⁹ This framework informed my dissertation conceptual diagram as I hypothesized the variables of maternal income, education, and occupation as mediators in the relationship between ACEs and PTSD and therefore did not control for them (see Figure 1 in Chapter 3).

Furthermore, my research aims were developed through the framework of a lifecourse study design, specifically, the “latent effects” model discussed by Pollitt et al. (2005), which “hypothesizes that adverse early life experiences increase the risk of [outcome] in later life...” (p. 2).⁸⁰ The acknowledgement of adverse childhood experiences as important life stressors with implications for distal health and developmental outcomes has been long studied by developmental psychologists.^{32,33,36,49} I apply this model as a guiding framework in my dissertation to understand how early life adversity occurring during childhood and adolescence has the potential to impact the risk of PTSD later in life.

Another foundational concept in lifecourse theory is the accumulation of risk exposures and protective factors across the lifespan, which is particularly important to hypothesized

explanations for racial and socioeconomic disparities in PTD that guided my dissertation work. Notably, Lu and Halfon (2003) depicted how differences in exposures to risk and protective factors across the developmental lifespan affect trajectories of reproductive potential and disparities in birth outcomes between White women and Black women.¹¹ Lu and Halfon (2003) hypothesized that disparities in adverse birth outcomes between White and Black women may stem from differences in levels of protective factors and accumulation of risk exposures across the lifecourse.¹¹ In application of this model, I examined differences in various multi-level protective factors by race and SES among a sample of $n = 2474$ pregnant women in my preliminary dissertation research,⁸¹ while in the first manuscript of this dissertation, I examine differences in a specific risk factor, ACEs, across race and SES subgroups of women.

2.5.3.1 Sensitive Periods in Development and the Biological Embedding of Stressors

In this section, I will provide a detailed discussion of the physiological mechanisms by which early life adversity can impact future disparities in adverse birth outcomes in adulthood, based on theory and literature from developmental psychology, which informed the conceptual framework for my dissertation (Figure 1, Chapter 3).

Specific stages of human development constitute what are known as sensitive periods, or “...times during development when experience exerts a very strong influence on the brain and on behavior” (Cicchetti, 2015, p. 319).⁸² While related to the concept of critical periods, which is defined as a “window of heightened brain plasticity for encoding specific environmental inputs through experience expectant mechanisms that results in irreversible changes in brain function with permanent effects on behavior” (Nelson & Gabard-Durnam, 2020, p. 134)⁸³, sensitive periods differ in that they allow for plasticity to occur even after the period ends.⁸³ Plasticity refers to “the ability of the brain to reorganize neuronal pathways that are based on new

experienced encountered throughout the course of life span development.” (Cicchetti, 2015, p. 319).⁸² Developmental periods with increased opportunity for plasticity, such as the pubertal period in adolescence, has important implications for the development of clinical interventions,^{84,85} as will be discussed later in Chapter 7.

Childhood and adolescence are hypothesized to be sensitive periods for the “biological embedding” of both positive (i.e., a nurturing infant-caregiver relationship) and negative experiences (i.e., caregiver-perpetuated abuse and neglect).^{51,83} Biological embodiment refers to how life experiences “get under the skin,”³³ like a physiological footprint of our day-to-day experiences. Krieger (2001) defines biological embodiment as “...how we literally incorporate, biologically, the material and social world in which we live, from conception to death” (p. 672).⁷⁰

It is hypothesized that exposure to early life stressors during these sensitive periods of development initiate changes in the structure and functioning of multisystemic neuroendocrine and immunological systems that influence an individual’s future disease risk and developmental outcomes.^{32–35,51,83} I define *stressor* as an “actual or perceived threat to an organism” and *stress* as the organism’s “response to the stressor” (Schneiderman et al., 2005, p. 1).⁸⁶ To be more specific, Cicchetti & Walker (2001) defined stress “as a perceived threat to an organism’s homeostasis and as a situation that causes increases in autonomic nervous system activity or hormone secretion” (p. 414).³⁶ Stressors can be physical (i.e., interpersonal violence), psychological (i.e., fear), and immunological (i.e., virus).³⁶ In this dissertation, I will specifically examine a physical and psychological life stressor, ACEs, that occur during the sensitive periods of childhood and adolescence, as a hypothesized mechanism that may impact disparities in PTSD.

While stress responses were developed evolutionarily to protect organisms against threats in the environment and to promote adaptation for survival, prolonged and chronic activation of the stress response system can be physiologically harmful, leading to disruptions in neuroendocrine, metabolic, and immunological systems.^{33,35,36}

In particular, the hypothalamic pituitary adrenal axis (HPA axis) is a neuroendocrine system that plays a central role in the human stress response, as well as other physiological systems (e.g., metabolic and immune), and is important to maintaining the body's adaptative and regulatory functions critical to homeostasis (i.e., blood pressure, heart rate).^{33,35,36} When this stress system is triggered, the hypothalamus in the brain releases specific hormones, such as the corticotrophin-releasing hormone (CRH) and arginine vasopressin (AVP), which activates the pituitary gland to release another hormone, called adrenocorticotrophic hormone (ACTH). ACTH travels through the blood and reaches an organ, called the adrenal glands, which sit on top of the kidneys.³⁵ In response, ACTH releases hormones called glucocorticoids, also known as cortisol in humans.³⁵

Glucocorticoids have a diverse range of roles in the stress response system and influence gene expression in response to activation of the HPA system.^{33,35} Most importantly, the HPA system entails its own negative and positive feedback system that maintains appropriate activation, but also suppression once a stressor is removed. Consequently, this critical feedback system can become dysregulated as a result of chronic activation, and it is this specific dysregulation process that been prominently implicated in the development of disease and pathology.^{32,33,35,36} As an example given by Gunnar (2007), "...an acute and short-lived stress response of the HPA axis may help prevent the immune system from "overshooting," but

prolonged stress activation of the HPA axis may chronically suppress immune functioning, increasing susceptibility to infections” (p. 131).³⁵

One of the potential indicators of HPA axis dysregulation is abnormal patterns in the diurnal cycle of cortisol production (i.e., production of either too high or too low levels of cortisol).^{33,35} Children with experiences of severe maltreatment in early life have been found to have abnormalities in HPA axis functioning, as reflected by higher than average cortisol levels.^{35,84,85,87,88} Gunnar & colleagues (2007) have observed similar elevations in baseline cortisol in studies of orphaned children.³⁵ These patterns of HPA axis activity have also been linked to adverse behavioral and psychological developmental outcomes in these children.^{35,84,85}

Why do early life adverse experiences have the potential to disrupt physiological systems in this way? Adverse childhood experiences represent a “violation of the expectable environment” (Nelson & Gabard-Durnam, 2020, p. 133),⁸³ which refers to the situation whereby “experiences that are expected to occur (in order to confer survival and adaptation to the environment) either do not occur (e.g., lack of caregiving; lack of nutrition) or are atypical in some way (e.g., physical abuse)” (Nelson & Gabard-Durnam, 2020, p. 134).⁸³ As elaborated by Nelson & Gabard-Durnam (2020), “The reason an absence of an expected experience or the presence of an atypical experience matter can be attributed to the experience-driven nature of brain development. When cortical specialization is driven by experience, atypical experiences or the lack of experiences during those windows should lead to atypical patterns of brain development” (p. 134).⁸³ For example, the quality of the infant-caregiver relationship (i.e., nurturing, secure attachment vs. insecure attachment and insensitive care) is critical to the regulation of the HPA axis and the child’s socio-emotional development in early life.^{32,35,83,89–91} When this nurturing and supportive caretaking environment is absent in early life, and an

unsupportive, neglectful, or abusive environment is present, mental and physical health consequences may result.⁸³

These adverse early life exposures can become biologically embedded in the form of physiological changes which can include shifts in epigenetic processes, changes to the hypothalamic pituitary adrenal (HPA) axis functioning and stress reactivity, changes in neural networks and connectivity, and metabolic dysfunction, including immunosuppression and chronic inflammation.⁵¹ This “early programming mechanism” (Lu and Halfon, 2003, p. 16)¹¹ triggered during sensitive periods has the potential to influence lifelong health outcomes.^{32,51,83}

Applying these concepts to the perinatal literature, it has been hypothesized that exposure to maternal stressors leads to dysregulation of the HPA axis and neuroimmune functioning, resulting in increased vulnerability to infections and inflammation.^{92–95} For example, higher levels of pro-inflammatory cytokines have been observed among women who deliver preterm.^{92,93} Studies have also found that higher maternal cortisol levels are associated with an increased risk of preterm birth,^{96–99} although the findings have been mixed.^{100–105}

Furthermore, one of the physiological changes that has been linked to early life adversity is earlier onset of menarche among girls,^{106–109} which may also be a risk factor for PTD.¹¹⁰ Belsky et al. (1991) developed an evolutionary theory of socialization that outlined two developmental trajectories, strongly rooted in John Bowlby’s attachment theory,⁸⁹ that result from childhood experiences with the potential to impact future reproductive behavior and success.¹⁰⁶

One trajectory depicts children who grow up in early caretaking environments characterized by maltreatment and subsequently develop adverse outcomes throughout the lifecourse.¹⁰⁶ The trajectory of these children starts with insecure attachment to caregivers in

childhood, followed by early onset of puberty and early sexual activity in adolescence, and results in disruptions in the formation of stable relationships and commitment to raising children during adulthood.¹⁰⁶ On the other hand, children who grow up in supportive and nurturing caretaking environments develop secure attachments to caregivers in childhood, exhibit later onset of puberty and sexual activity.¹⁰⁶ These children are therefore able to form more stable relationships and display higher commitment to raising children during adulthood.¹⁰⁶ Support for this theory was demonstrated by Moffitt and colleagues (1992), who observed that dysfunctional family environments and absence of a father was associated with earlier age of menarche among adolescent girls.¹⁰⁷

Furthermore, ACEs have been linked to risky sexual behaviors, unintended pregnancy, sexually transmitted infections, as well as adverse behaviors during pregnancy (alcohol use, smoking, substance use), which are also risk factors for PTD.^{16,38,39,58,111} While my dissertation does not assess causal mechanisms between ACEs and PTD, I do investigate whether occurrence of ACEs during a sensitive period of childhood and adolescence are associated with PTD later in life, an important endeavor given the important role that ACEs may play in women's reproductive health.

Collectively, the findings from the developmental psychology literature provide an important lifecourse framework that lends plausibility to the process by which early life stressors may become biologically embedded to subsequently impact women's reproductive health outcomes. In my dissertation, I will apply these frameworks by assessing the association between adverse childhood experiences occurring before 18 years of age and odds of PTD.

2.5.4 Summary of Section 2.5

In this dissertation, I apply interdisciplinary theoretical frameworks across lifecourse epidemiology and developmental psychology to conceptualize how early adverse experiences may become biologically embedded during the sensitive periods of childhood and adolescence, and subsequently impact disparities in birth outcomes.

2.6 Protective Factors and Resilience Theory

With the understanding that exposure to life stressors, including adverse childhood experiences, may not necessarily be controllable or avoidable, there is growing attention of the need for a paradigm shift that incorporates salutogenesis. Salutogenesis is the promotion of well-being and positive adaptation through identification of and bolstering of protective factors,^{112,113} as opposed to an exclusive focus on the pathogenesis approach of risk factor identification.¹¹² This concept of positive adaptation in the face of major life adversity refers to the process of resilience, defined as “the process of, or capacity for, an outcome of successful adaptation despite challenging or threatening circumstances” (Masten, Best, & Garmezy, 1990, p. 426).⁷³

Resilience theory arose from research in the 1960s and 1970s at the intersection of developmental psychology, psychopathology, and child development that identified a group of children exhibiting positive developmental outcomes despite experiencing adverse life events.^{49,73,114} These studies identified several characteristics in these children (i.e., easy-going temperament, maturity, positive relationship with an adult) that contributed to their unexpected positive adjustment and seemed to buffer them from their circumstances of severe adversity.^{72,73} These factors are known as protective factors, or factors which “moderate the effects of individual vulnerabilities or environmental hazards so that the adaptational trajectory is more

positive than would be the case if the protective factor were not operational” (Masten, Best, & Garmezy, 1990, p. 426).⁷³

The process of resilience entails an “interaction between both risk and protective processes, internal and external to the individual, that act to modify the effects of an adverse life event” (Olsson et al., 2003, p. 2).¹¹⁵ Protective factors that may serve to buffer against life stressors to promote resilient health outcomes can function across many socioecological levels, including the individual (self-esteem, sense of humor, hopefulness, spirituality, optimism), family (social support), and community (social cohesion, reciprocal exchange) levels.^{43,49,72,74,76,115} As individuals may draw upon these protective factors during periods of severe stress to promote well-being, protective factors may thus potentially buffer women against adverse pregnancy and birth outcomes in the face of adversity.

2.7 Protective Factors & Preliminary Research from Zamani-Hank et al. (2022)

In my preliminary dissertation research from Zamani-Hank et al. (2022),⁸¹ I set out to investigate whether seven types of potential protective factors across the individual, interpersonal, and community levels decreased the risk of PTD among a prospective cohort of pregnant women from Michigan (POUCH; 1998-2004), and whether this relationship differed by race and SES subgroups of women. The following sections (2.7.1, 2.7.2, 2.7.3, and 2.7.4) contain excerpts from this research which provide context and background as the building block for this dissertation study.

2.7.1 Protective Factors and Health-Related Outcomes

As aforementioned in Section 2.6, because life stressors may not always be avoidable or preventable, research which aims to assess the role(s) of potential protective factors in promoting positive health outcomes is critical, especially for women who may experience a

disproportionate number of life stressors. Elaborating upon this point in my preliminary dissertation research, Zamani-Hank et al. (2022) stated,

“Black women and low SES women often have greater exposure to an array of adverse life experiences due to “multiple marginalizations” at the intersection of race, sex/gender, and socioeconomic position.¹¹⁶ While dismantling the systemic contributions to adversity are critical to creating equity in pregnancy health and birth outcomes, understanding the protective factors that promote healthy pregnancy outcomes for women in “multiply marginalized”¹¹⁶ positions in society may provide useful targets for intervention” (Zamani-Hank et al., p. 244).⁸¹

A diverse array of protective factors has been identified in the literature as promoting positive health outcomes. Zamani-Hank et al. (2022) elaborate upon some of these protective factors in the following excerpt,

“Among adults, specific protective factors such as social support have been associated with multiple health behaviors and outcomes, including decreased risk of depression,¹¹⁷ self-efficacy in relation to substance use recovery,¹¹⁸ improved weight loss outcomes for obesity,¹¹⁹ improved glycemic control in diabetes,¹²⁰ smoking cessation,¹²¹ and greater adherence to antiretroviral therapy for HIV.¹²² Having low levels of social networks has been associated with increased risk of stroke and cardiovascular mortality,¹²³ while low functional social support has been associated with higher risk of mortality among patients with coronary heart disease.¹²⁴

Similarly, religiosity and spirituality have been associated with improved quality of life among patients with cardiovascular disease,¹²⁵ lower blood pressure,¹²⁶ fewer symptoms of depression,^{127,128} and better management of diabetes.^{129,130}

Availability and use of specific protective factors appear to differ by gender, race/ethnicity, and socioeconomic status. For example, women are more likely to seek social support and report higher religiosity compared to men.^{131–133} Black individuals, particularly Black women, report greater religious involvement.^{133–135} Among low SES individuals, protective factors such as religiosity, high levels of perceived control, having a purpose in life, and high optimism have been identified as promoting positive health outcomes in the face of adversity.^{136,137} These findings provide a basis for investigating whether and how protective factors influence disparities in adverse pregnancy outcomes by race/ethnicity and SES” (Zamani-Hank et al., 2022, p. 244).⁸¹

Specifically, my dissertation will assess how two potential protective factors, religiosity and spirituality, influence the relationship between adverse childhood experiences and PTD, and whether this relationship differs across race and SES subgroups.

2.7.2 Protective Factors and Birth Outcomes

While my dissertation specifically assesses religiosity and spirituality as potential protective factors against PTD, it is important to discuss what is currently known about the relationship between protective factors and birth outcomes. Zamani-Hank et al. (2022) provided the following summary of the literature on factors which have been identified as being protective in the context of birth outcomes,

“There is increased attention to studying how protective factors, particularly social support, may either 1) directly reduce risk of adverse pregnancy outcomes or 2) buffer against negative impacts on pregnancy outcomes of factors such as adverse childhood experiences, perceived stress, and stressful life events.^{14,77,78,138}

For example, among female teenagers enrolled in a WIC program who had uncomplicated healthy pregnancies, factors at both the individual-level (positive outlook, self-efficacy, and prenatal care attendance) and family level (parental and partner social support) were identified as protective influences, emphasizing the role that such factors may play in contributing to resilient pregnancy outcomes among low-SES women.⁷⁴ However, in a meta-analysis, investigators noted no evidence of a direct association between social support and preterm birth based on the pooled findings of eight studies.⁷⁸

On the other hand, pooled findings from two studies which examined the buffering effects of social support found that women with low social support and high stress levels experienced higher odds of preterm birth compared to women with high social support and high stress levels.⁷⁸ Specifically, women with high levels of cumulative psychosocial stress (operationalized by ‘state anxiety’ and history of mental health problems, abuse, and negative feelings about pregnancy timing) and low levels of perceived social support experienced higher odds of preterm birth (OR 2.09, 95% CI 1.07, 4.07).¹³⁸

The second study examined multi-level protective factors such as self-esteem, mastery, partner support, social support, neighborhood support and found that women with higher levels of stress compared to protective factors (as operationalized by a higher stress-to-capital ratio [SCR]), had higher odds of experiencing premature labor compared to women who had lower stress-to-capital ratios (OR=1.36; p=0.03).⁷⁶ Furthermore, Black and Hispanic women had higher mean SCR scores compared to White women (p<0.001, respectively) while low-income women had higher SCR scores compared to higher-income women.⁷⁶

Together, these findings suggest that protective factors may reduce the risk of adverse pregnancy outcomes in the context of stress, and that protective factors may differ by race/ethnicity and socioeconomic status.

While providing significant contributions to an understanding of protective factors in relation to pregnancy outcomes, previous studies in the perinatal literature have (1) focused primarily on social support and rarely incorporated data on additional protective factors at multiple socioecological levels and (2) typically not assessed how protective factors vary by race/ethnicity and SES” (Zamani-Hank et al., p. 245).⁸¹

2.7.3 Preliminary Research on Protective Factors from the POUCH Study (Zamani-Hank et al., 2022)⁸¹

To address the aforementioned gaps in the literature, I conducted preliminary dissertation research utilizing data from the Pregnancy Outcomes and Community Health (POUCH) Study,¹³⁹ a prospective cohort study of pregnant women (n=3,019) from five diverse communities across Michigan between 1998-2004 to examine seven potential protective factors at multiple socio-ecological levels (individual, interpersonal, neighborhood) during pregnancy and determine if they: 1) differ in prevalence by race/ethnicity and SES; and 2) are associated with risk of PTD overall or within specific race/ethnicity and SES groups.

Addressing these research objectives, Zamani-Hank et al. (2022) found that,

“White women reported significantly higher levels of self-esteem, mastery, perceived social support, emotional social support, instrumental social support, and reciprocity compared to Black women ($p < 0.01$, respectively). Black women reported significantly higher levels of religiosity (71.7% vs. 56.9%; $p < 0.01$) compared to White women. However, the race/ethnicity differences in the continuous variables were all

small in magnitude (Hedges's g range 0.1-0.4) except for instrumental social support, which was moderate (Hedges's $g = 0.5$, data not shown). Differences in emotional social support and religiosity were small in magnitude between White and Black women (Cramer's $V = 0.1$, respectively). A significantly higher proportion of Black women delivered preterm compared to White women ($p < 0.01$)” (Zamani-Hank et al., 2022, p. 248, 250).⁸¹

Furthermore, Zamani-Hank et al. (2022) observed that,

“All seven protective factors differed significantly by SES ($p < 0.01$, respectively). High SES women (top quartile) reported significantly higher levels of self-esteem, mastery, perceived social support, instrumental social support, and reciprocity compared to both middle SES (2nd and 3rd quartiles) and low SES (bottom quartile) women ($p < 0.01$, respectively), and middle SES women reported significantly higher levels of all these variables ($p < 0.01$, respectively) compared to low SES women (bottom quartile), with the exception of reciprocity ($p = 0.13$).

While differences in all continuous protective factors between low and middle SES women, and between middle and high SES women, were small in magnitude (Hedges's g range 0.1-0.4), the differences between low SES and high SES women were large in magnitude for instrumental social support (Hedges' $g = 0.8$) and moderate for perceived social support (Hedges's $g = 0.7$), mastery (Hedges's $g = 0.7$), and self-esteem (Hedges's $g = 0.6$). Differences in emotional social support and religiosity by SES were small in magnitude (Cramer's $V = 0.1$). There was no statistically significant difference in the proportion of women who experienced PTD by SES ($p = 0.08$).

Among Black and White women combined, none of the seven protective factors were significantly associated with odds of PTD in unadjusted or adjusted logistic regression models. However, the association between religiosity and PTD differed significantly by race/ethnicity ($p=0.08$) and SES ($p=0.07$). Black women who reported religiosity had decreased odds of PTD compared to Black women who did not report religiosity, and this approached significance (OR 0.6, 95% CI 0.4, 1.0). Low SES women who reported religiosity had decreased odds of PTD compared to low SES women who did not report religiosity (OR 0.6, 95% CI 0.4, 0.9). The associations between perceived social support, emotional social support, and reciprocity and PTD also differed significantly by SES ($p=0.05$, $p<0.01$, $p=0.06$, respectively).

Low SES women who reported higher perceived social support and reciprocity had decreased odds of PTD compared to low SES women who did not, although this was marginally significant (OR 0.9, 95% CI 0.8-1.0; OR 0.9, 95% CI 0.8-1.0, respectively). The association between emotional social support and PTD was not statistically significant for middle SES (OR 0.8, 95% CI 0.3-2.2) or low SES women (OR 0.6, 95% CI 0.3-1.3)” (Zamani-Hank et al., 2022, p. 250).⁸¹

2.7.4 Discussion on Findings from the POUCH Study (Zamani-Hank et al., 2022)⁸¹

In discussion of our findings regarding protective factors, race/ethnicity, and SES, Zamani-Hank et al. (2022) provided the following synthesis:

“While our study found that White women reported higher levels of all protective factors—with the exception of religiosity—compared to Black women during pregnancy, our findings are not consistent with those of Jesse et al. (2005) who found that African American women in rural prenatal clinics had higher self-esteem, religiosity, spirituality,

and social support compared to Caucasian women.¹⁴⁰ On the other hand, in a sample from an urban prenatal clinic, Jesse et al. (2009) found that African American women had lower self-esteem, higher social support from others, and comparable levels of partner social support compared to Caucasian women.¹⁴¹

Discrepancies between our findings and those of previous work may be due in part to differences in study population (e.g., urban vs. rural; low-income vs. general), sample size, different types of social support assessed, context in which social support is assessed (e.g., coping with a health condition vs. caregiving), and methods of assessing protective factors.¹⁴²

In addition, our findings suggested a gradient effect, whereby high SES women reported the highest levels of protective factors, followed by middle SES women who reported intermediate levels of protective factors, and low SES women who reported the lowest levels of protective factors. Women from lower socioeconomic backgrounds may have fewer social support networks from which to obtain resources, compared to high SES women.¹⁴³

Our finding of higher religiosity among Black women compared to White women concurs with literature on the importance of religion as a coping mechanism and source of comfort within the Black community, especially for Black women.^{134,144–147} Chatters et al. (2008) state, “...Religious orientations and strategies are an especially prominent and robust component of the coping repertoires of African Americans who are more likely than their Whites to report their use in response to a variety of problems and contexts including health issues, caregiving burdens, chronic poverty, poor neighborhood conditions, structural exclusion, and interpersonal and structural racism” (p. 373).¹³⁴

Moreover, religiosity may be an important source of support for Black women especially in the face of life stressors.^{134,135,146,148}

Pregnancy represents a significant period of change for women during which spirituality may play an important role in conferring support and comfort.^{149,150} In our study, although religiosity was asked during pregnancy, it was assessed as a general question, not specific to the prenatal period. Future research using a longitudinal approach is needed to better contextualize the role of religiosity and/or spirituality in the lives of reproductive-aged women, including whether findings differ according to race/ethnicity” (Zamani-Hank et al., 2022, p. 251).

In addition, Zamani-Hank et al. (2022) provided the following analysis of their findings regarding protective factors and PTD:

“We found that the specific set of protective factors linked to a reduction in PTD differed by race/ethnicity and SES, although most odds ratios only approached statistical significance. Religiosity was associated with an approximately 40% reduction in the odds of PTD for low SES women and Black women, consistent with previous studies supporting the protective influence of religiosity on health-related outcomes- including PTD, depression from HIV-related stigma, diabetes, and hypertension- among Black women.^{135,148,151,152}

Religiosity is also hypothesized to serve as a buffer against the adverse circumstances associated with poverty among low-income populations.¹³⁷ Specifically, “Higher levels of religiosity may provide a pathway out of multi-problem behavioral patterns that can accompany limited resources by promoting better coping mechanisms for economic instability and stress....” (Joshi et al., 2009, p. 2).¹³⁷ For example,

religiosity has been linked previously with reduced risk of smoking, improved positive well-being and health behaviors, self-efficacy, and decreased risk of depression among low-income women.¹³⁷

Religiosity also has been associated with positive health behaviors during pregnancy including good nutrition, and abstaining from alcohol, smoking, or substance use.^{153–155} Previous POUCH studies also found that women with highest levels of stress hormones during mid-pregnancy had significantly elevated risk of spontaneous PTB compared to women with lowest levels of stress hormones.¹⁵⁶ Furthermore, women who reported higher levels of hostility and anomie during mid-pregnancy had increased risk of PTD in the POUCH study.¹⁵⁷ Thus, religiosity and/or spirituality may protect against the risk of PTD through potential buffering of environmental, physiological, and psychosocial stressors.

Moreover, religiosity has been associated with upward mobility,¹⁵⁸ other potential pathway between religiosity and lower PTD risk. Women in the POUCH study who experienced upward socioeconomic mobility from childhood to adulthood exhibited a lower risk of PTD,¹⁵⁹ as well as delivering a small-for-gestational age (SGA) infant.¹⁶⁰ Upward social mobility may serve to decrease a woman's risk of PTD by reducing the body's "wear and tear" in the context of allostatic load,¹⁶⁰ and/or by conferring greater access to certain resources (health care, recreational facilities, education).

The findings of previous research on the impact of social support on PTD among low SES women are mixed.^{141,161} Among an urban sample of low-income Black non-Hispanic women, emotional social support was associated with significantly reduced odds of preterm birth, but socializing social support, instrumental social support, and

interactive social support were not.¹⁶¹ However, among a sample of low-income pregnant women from a rural health clinic, social support was not significantly associated with preterm birth.¹⁴¹

Nevertheless, having neighbors who can lend help in times of need, as assessed by neighborhood reciprocity, can contribute to one's perceived levels of social support,^{162,163} and thus influence pregnancy outcomes.⁷⁶ Furthermore, the quality of a neighborhood environment, including safety, social and physical disorder, and walkability, impacts the prevalence of depression and perceived stress levels during pregnancy,¹⁶⁴ both of which represent risk factors for PTD.⁹² Positive aspects of a neighborhood, including reciprocity and social cohesion, buffer against depression specifically among women,¹⁶⁵ highlighting the importance of community-level protective factors on women's health outcomes.

Thus, our finding that reciprocity buffers against PTD among low SES women may suggest possible mediating mechanisms through its impact on pregnant women's mental health and perceived stress levels" (Zamani-Hank et al., 2022, pp. 251-252).⁸¹

Finally, Zamani-Hank et al. (2022) concluded that,

"Overall, our findings suggest that the *specific set* of protective factors that buffer against adverse birth outcomes like PTD may differ among women by race/ethnicity and SES, a finding that corroborates previous research.¹⁴¹ On the other hand, among all women, we did not find that protective factors at the individual, interpersonal, or neighborhood levels were independently associated with PTD. The "protective" aspect of protective factors on health outcomes may not become manifest unless adversity is presented.^{49,115} Indeed, Margerison-Zilko et al. (2017) found that certain types of

adversity, specially childhood sexual abuse, was associated with increased risk of PTD in the POUCH cohort¹⁹” (Zamani-Hank et al., 2022, p. 252).⁸¹

Informed by my preliminary findings in Zamani-Hank et al. (2022),⁸¹ my dissertation incorporates a specific measure of adversity, adverse childhood experiences, to determine whether two types of protective factors, religiosity and spirituality, moderate the association between adverse childhood experiences and PTD risk.

2.7.5 The role of religiosity and spirituality in pregnancy and birth outcomes

The majority of studies that have examined religiosity and spirituality (hereafter, “R/S”) in the context of pregnancy and reproductive health have focused on the impacts of R/S on mental health during pregnancy,¹⁶⁶ mental health during the postpartum period,¹⁶⁷ health behaviors during pregnancy like tobacco use and substance use,¹⁶⁶ contraceptive use,¹⁶⁸ and risky sexual behaviors.¹⁶⁹ Most of these studies have focused on either religiosity *or* spirituality and often do not make distinctions between religiosity *and* spirituality. More importantly, there is a dearth of empirical literature on the influence of R/S on birth outcomes.

Indeed, I found only one, outdated, published empirical study that examined the effects of religiosity on birthweight. In a study of n=6566 pregnant women in the Mater-University of Queensland Study of Pregnancy (MUSP), Najman et al. (1988) found that women who were members of 13 Christian religious sects (i.e., Mormon, Assembly of God, Brethren) had significantly higher mean birthweights (adjusted for maternal age, parity, marital status, and income) than Christians who did not attend church frequently ($p<.001$), but this significance disappeared once the means were additionally adjusted for cigarette and alcohol use.¹⁷⁰

Furthermore, a PubMed search of religiosity and preterm birth yielded only six findings as of June 2022, only one of which examined the impacts of R/S on preterm birth. In a study of n

= 91 low-income Mexican-immigrant women from a prenatal clinic in Texas, Page et al. (2021) found that frequency of prayer and level of religiosity significantly *increased* the odds of PTB, contrary to their hypothesis.¹⁷¹ In addition, a PubMed search of spirituality and preterm birth yielded 26 findings as of June 2022, of which only one study examined R/S and preterm birth—the same study of Page et al. (2021).¹⁷¹ These search results convey the critical gaps in the literature regarding research that assesses the impacts of religiosity and spirituality on PTB, which my dissertation addresses.

While most of the studies in the literature do not distinguish between religiosity and spirituality and often use the terms interchangeably,^{172–174} it is important to understand their differences. While religiosity and spirituality are related concepts, and, hence, often denoted as “R/S” in the literature, there are distinctions in their respective definitions.^{174–176} Religiosity (or sometimes known as religiousness in the literature) has been defined as, “the degree which an individual believes, follows, and practices a religion” (Damiano et al., 2019, p. 5).¹⁷⁴ Religion is “defined as a personal set or institutionalized system of religious attitudes, beliefs, and practices, and is the service or worship of God or the supernatural” (Victor and Treschuk, 2020, p. 107).¹⁷² Spirituality is generally a broader concept and encompasses the process of finding meaning and a sense of peace in life.^{172,174,176} It has been formally defined as, “...a dynamic and intrinsic aspect of humanity through which persons seek ultimate meaning, purpose, and transcendence, and experience relationship to self, family, others, community, society, nature, and the significant or sacred. Spirituality is expressed through beliefs, values, traditions, and practices (Puchalski et al., 2014, p. 646).”¹⁷⁷ Hence, if one identifies as being spiritual, they may not necessarily identify as being religious, and vice versa.¹⁷⁵

Because of these distinctions in definition, it is important to assess whether religiosity and spirituality may exert their effects differently on health outcomes. Therefore, in this body of work, I address these literature gaps by examining religiosity and spirituality separately, while also examining an aggregate R/S variable comprising questions pertinent to both constructs.

While studies demonstrate that religiosity and spirituality can be relied on by parents as sources of strength in the face of traumatic pregnancy and birthing experiences (i.e., pregnancy loss, stillbirth, NICU),^{178–180} my PubMed search of “religiosity and preterm birth and adverse childhood experiences” as well as “spirituality and preterm birth and adverse childhood experiences” yielded 0 results as of June 2022, underscoring that research on the relationship between ACEs, preterm birth, and R/S is a major gap in the literature that my dissertation addresses.

Our findings from Zamani-Hank et al. (2022) highlight the importance of assessing religiosity as a potentially important protective factor against PTD for pregnant women, particularly for Black women and women with low SES.⁸¹ However, as discussed in Zamani-Hank et al. (2022), the full “protective” effects of a protective factor on PTD may not become manifest unless adversity is encountered.⁸¹ Therefore, to comprehensively assess the effects of R/S on PTD in the context of a specific stressor, my dissertation incorporates a measure of adversity (i.e., ACEs) to determine whether religiosity and spirituality moderate the association between adverse life experiences and PTD risk.

Research Gaps: There are few published studies in the literature which have examined the effects of R/S on birth outcomes and no studies, to my knowledge, which have examined the relationship between ACEs, R/S, and PTD, let alone differences by race and SES. Furthermore,

the studies which do examine religiosity and spirituality in the context of pregnancy do not distinguish between religiosity and spirituality, even though they have important distinctions.

2.8 Summary of Outstanding Gaps in the Literature

While foundational research has demonstrated that ACEs can negatively impact pregnancy health and birth outcomes and that R/S can serve as important protective factors for health-related outcomes, few studies have **1)** used interdisciplinary theoretical frameworks across social epidemiology and developmental psychology to analyze how ACEs may impact PTD **2)** assessed the influence of ACEs on PTD by race and socioeconomic status subgroups using diverse, national samples **3)** assessed whether specific ACEs, or co-occurring patterns of certain ACEs, may exert greater detrimental effects on PTD risk than others and **4)** examined the relationship between religiosity, spirituality, ACEs, and PTD. These gaps prevent a complete understanding of how the interplay between adverse life experiences and protective factors may influence disparities in PTD, undermining our ability to develop effective interventions.

2.9 Specific Aims of this Dissertation

Acknowledging these gaps in the literature while building upon the important contributions of previous research, **the overall goal** of this dissertation is to investigate the relationship between adverse childhood experiences, preterm delivery, and two types of potential protective factors (religiosity and spirituality) in a large, longitudinal sample of women using a health equity framework and lifecourse approach. I will address this goal through three specific aims:

- 1. Determine the association between specific adverse childhood experiences and odds of PTD and evaluate whether this relationship differs across race and SES.**
- 2. Identify subgroups of women characterized by early life patterns of ACEs and determine the association between subgroup membership and odds of PTD.**

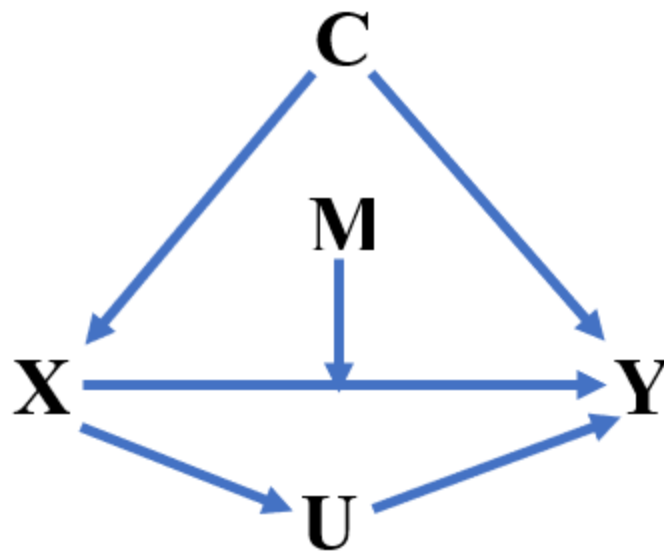
- 3. Examine the role that potential protective factors (i.e., religiosity and spirituality) play in the association between adverse childhood experiences and odds of PTD, and whether these factors operate differently by race and SES.**

CHAPTER 3: METHODOLOGY

3.1 Conceptual Framework

The conceptual framework of the hypothesized relationships between adverse childhood experiences, PTD, and covariates guiding this dissertation work is depicted in Figure 1 below. Specifically, I hypothesize that adverse childhood experiences will be associated with increased odds of PTD among women overall, controlling for race and childhood socioeconomic status, which I conceptualize as potential confounders in the relationship between ACEs and PTD (Figure 1). Next, I hypothesize that two types of potential protective factors, religiosity and spirituality, will modify the association between ACEs and PTD (Figure 1). Finally, I hypothesize that the relationship between ACEs and PTD will differ in stratified analyses by race and SES subgroups, and that the degree to which the potential protective factors modify the association between ACEs and PTD will also differ within race and SES subgroups. Thus, because my approach is also to examine whether this exposure outcome relationship differs by stratified analyses of race and SES, I also consider race and SES as potential modifiers.

Furthermore, theory and literature suggest that the experience of adverse events during childhood can subsequently impact age of menarche, age at which women give birth (i.e., maternal age), parity, social relationships including marital status, adverse health behaviors (i.e., risky sexual activity, smoking, alcohol use), mental health, as well as socioeconomic status in adulthood,^{16,32,79,107,181} all of which can influence the risk of PTD. Therefore, I conceptualize these variables as mediators in the pathway between ACEs and PTD (Figure 1), and I do not control for them in my analyses.



KEY

X: Exposure
Adverse childhood experiences

Y: Outcome
PTD

C: Confounders
Race
Childhood socioeconomic status

M: Effect Modifiers
Religiosity
Spirituality
R/S
Race
Childhood SES

U: Mediators

Sociodemographic
Maternal education
Maternal occupation
Maternal income
Maternal age
Marital status

Health Behaviors
Alcohol use, smoking
Risky sexual activity

Mental health conditions
Depression, anxiety

Physiological & reproductive characteristics
Early age at menarche
Parity

Figure 1. Conceptual diagram of the hypothesized relationships between adverse childhood experiences, PTD, and covariates.

3.2 Study Design

I used data from the National Longitudinal Study of Adolescent to Adult Health (Add Health; 1994-2018), a nationally representative longitudinal cohort study of adolescents initiated in 1994 by the University of North Carolina at Chapel Hill and funded by the NIH National Institute of Child Health and Human Development.¹⁸²

The main objective of this prospective study was to utilize a life course framework to study the diverse biomedical, behavioral, environmental, and psychosocial influences on the health outcomes and behaviors of adolescents and their development in adulthood.¹⁸²⁻¹⁸⁴ Add Health consists of five waves of longitudinal follow-up of adolescents in grades 7-12 (ages 12-19) who were recruited at study initiation in 1994: Wave I (1994-1995), Wave II (1996), Wave III (2001-2002), Wave IV (2008), and Wave V (2016-2018) and includes in-school, at-home, and parental components of the survey administration.^{182,183}

Add Health used a school-based, multistage sampling method, whereby the primary sampling unit consisted of schools and the secondary sampling unit consisted of students from those schools.¹⁸³ The sampling frame consisted of $n = 80$ U.S. high schools identified in Quality Education Database.¹⁸³ Each high school was paired with a respective “feeder” middle school, from which students went on to attend the identified high school.¹⁸³ This resulted in a sampling frame of over 100,000 students and parents contacted to participate in Wave I (1994-1995).¹⁸³ Due to the stratified, clustered sampling design of Add Health which oversampled for specific populations (i.e., not all participants had an equal probability of selection),¹⁸⁵ I applied survey weights to all analyses.

A total of $n=90,118$ students from grades 7-12 (aged 12-19 years), for whom parental consent was granted, participated in the in-school questionnaire component of Wave I.^{182,183} A

sample of students (n=20,745) were then selected to participate in at-home interviews.¹⁸³ A summary of the target population, source population, and study population of Add Health is illustrated in Figure 2.

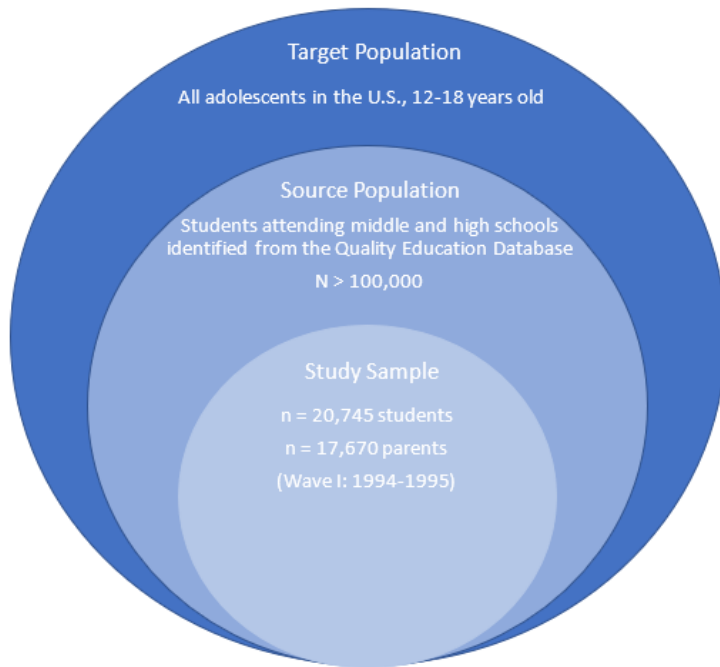


Figure 2. Summary of the target population, source population, and study population of Add Health (1994-2018)¹⁸³

Add Health questionnaires were administered via computer-assisted personal interview (CAPI) and Audio Computer-Assisted Self Interview (ACASI) techniques in Waves I-IV, which have been found to increase data quality of self-reported data, particularly in regard to sensitive information.^{183,186} A mixed-mode method was adopted in Wave V due to reduced funding, which comprised primarily of self-administered web and mail questionnaires, as well as in-person interviews.¹⁸² Non-responders were followed up in-person and via phone in Wave V.¹⁸² Add Health has demonstrated high response rates (>80% of participants in Wave IV from the original sample) which compare favorably with other national longitudinal surveys which have multi-

year gaps between waves of data collection.¹⁸² For example, the Midlife in the United States Survey had a response rate of 75% in the 2004-2006 wave of data collection.¹⁸²

3.3 Study Population and Inclusion Criteria

Of the 20,743 adolescents enrolled in Wave I, I restricted my analytic sample to female adolescents enrolled in 1994-1995 during Wave I who had a pregnancy that ended in a live birth by 2018 and reported data on preterm delivery for the first birth in Wave V (See Figure 3). Thus, after excluding male adolescents from my sample (n=10,263) and female adolescents who did not have a pregnancy that ended in a live birth by 2018 (n=6,446), I arrived at my reference population of n=4,034 (Figure 3). After removing female adolescents who did not report information on PTD for the first birth (n=71) in Wave V and female adolescents who were missing data on any weighting variables (n=66) or race (n=13), I arrived at the final analytic sample of n=3,884 used in Chapters 5 and 6. After removal of female adolescents with missing data on ACEs (n=117), I arrived at the final analytic sample of n=3,767 used in Chapter 4. A summary of the target population, reference population, and study sample of this dissertation study are summarized in Figure 4. While I acknowledge that not only those who identify as women may be able to give birth, the dataset solely captures the biological sex categories of male and female, preventing us from disaggregating gender from sex.

The analytic sample size in Chapter 4 differs from that of Chapters 5-6 due to application of the full-information maximum likelihood approach to address observations with missing data. Full-information maximum likelihood (FIML) is a method used in structural equation modeling (i.e., latent class analyses) to account for observations with missing data on the variables (i.e., indicators) used to inform the latent class.¹⁸⁷ FIML was applied to the latent class analyses conducted in Chapters 5 and 6 to account for the observations with missing data on the ACEs

(n=117). Therefore, these observations did not need to be removed from the sample for these chapters. However, as FIML cannot be implemented in SAS for logistic regression models,¹⁸⁸ FIML was not applied to the dataset for Manuscript 4.

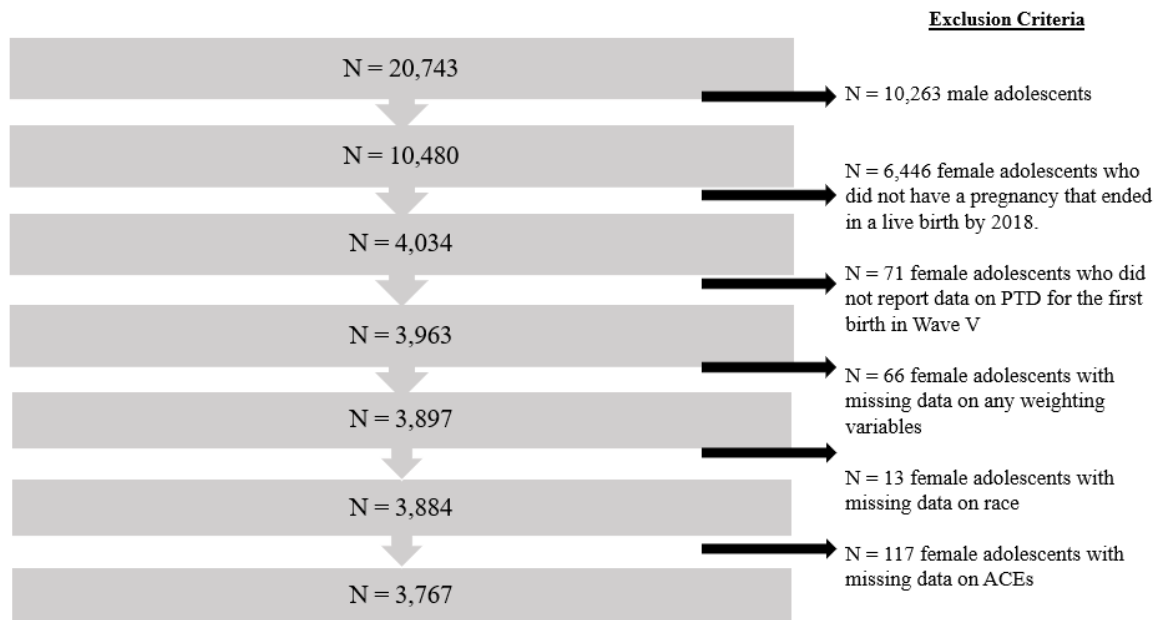


Figure 3. Analytic sample derivation used to assess the association between adverse childhood experiences (ACEs) and PTD among women in the National Longitudinal Study of Adolescent to Adult Health (1994-2018).

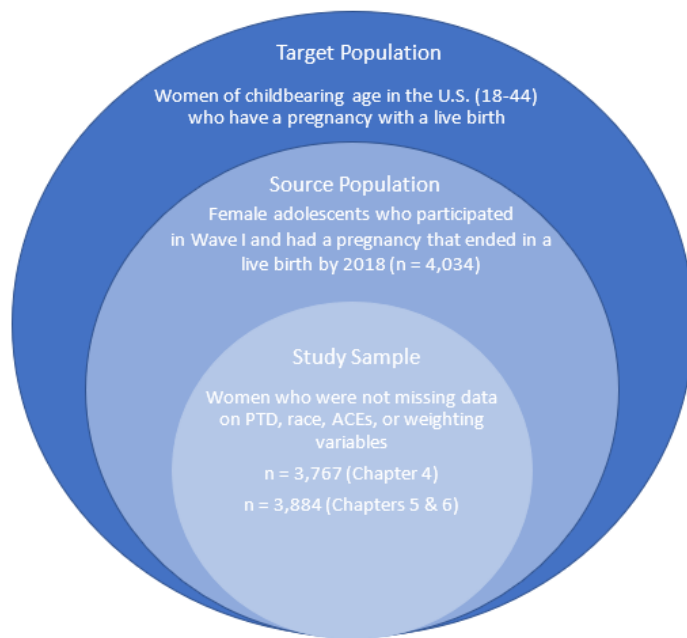


Figure 4. Summary of the target population, source population, and study population of this dissertation study using the National Longitudinal Study of Adolescent to Adult Health (1994-2018)

To evaluate whether removal of observations with missing data on key variables in the two analytic samples significantly changed the distribution of variables between the analytic samples and the reference population (and therefore introducing the potential for bias), I conducted a descriptive assessment of bias (Table 1). Because the distribution of the variables was not significantly different between each of the analytic samples, respectively, and the reference population, the likelihood of systematic bias was deemed low, which strengthens the internal validity of my findings.

Table 1. Assessment of bias between the reference population and two analytic samples on sociodemographic and health-related characteristics among women in the National Longitudinal Study of Adolescent to Adult Health (1994-2018).

	Reference Population (Women with live birth pregnancies) Max N = 4,034		Manuscripts 2 & 3 Analytic Sample Max N = 3,884		Manuscript 1 Analytic Sample Max N = 3,767	
	N	(Wt%)	N	(Wt%)	N	(Wt%)
Race						
White	2346	(68.1)	2297	(68.7)	2226	(68.5)
Black	773	(14.6)	723	(14.4)	702	(14.4)
Hispanic	538	(10.5)	517	(10.3)	502	(10.4)
Asian	205	(2.9)	197	(2.9)	194	(3.0)
Pacific Islander	35	(0.5)	32	(0.5)	29	(0.5)
American Indian or Alaska Native	93	(2.4)	89	(2.4)	86	(2.4)
Other	31	(0.8)	29	(0.8)	28	(0.8)
Socioeconomic status (SES)						
Low	513	(12.6)	487	(12.5)	473	(12.7)
Middle	2476	(61.0)	2382	(61.1)	2309	(61.1)
High	1045	(26.4)	1015	(26.4)	985	(26.2)
Marital Status						
Missing	356	(8.7)	324	(8.2)	314	(8.0)
Married/Cohabiting	2986	(75.3)	2893	(75.7)	2807	(76.0)
Not Married/Cohabiting	692	(16.0)	667	(16.1)	646	(16.0)
Maternal age at time of delivery (years)						
Missing	229	(6.2)	198	(5.4)	190	(5.0)
≤19	597	(14.5)	562	(14.5)	537	(14.3)
20-24	1090	(28.3)	1048	(28.2)	1021	(28.3)
25-29	1043	(24.6)	1021	(24.9)	996	(25.1)
30-34	818	(20.2)	807	(20.7)	783	(20.8)
>35	257	(6.1)	248	(6.2)	240	(6.1)
Preterm Delivery (PTD)						

Table 1 (cont'd).

Yes	520	(11.6)	514	(11.8)	501	(11.7)
Adverse Childhood Experiences (ACEs)						
Sexual abuse	225	(6.2)	217	(6.3)	212	(6.2)
Physical abuse	765	(20.1)	738	(20.2)	721	(20.4)
Emotional abuse	1865	(47.6)	1793	(47.7)	1750	(48.2)
Neglect	1674	(42.0)	1614	(42.3)	1585	(42.8)
Family Member suicide attempt or death	229	(6.1)	216	(6.1)	209	(6.0)
Foster care placement	94	(2.7)	90	(2.7)	87	(2.7)
Protective Factors	Mean (SE)		Mean (SE)			
Religiosity	1.4	(.02)	1.4	(.02)	NA ¹	NA ¹
Spirituality	1.5	(.02)	1.5	(.02)	NA ¹	NA ¹
R/S	10.4	(.06)	10.4	(.06)	NA ¹	NA ¹

¹Variables not assessed in Manuscript 1

3.4 Measures

3.4.1 Exposure

I assessed six adverse childhood experiences (ACEs) as my primary exposure variables (Table 2). ACEs were self-reported retrospectively by female study subjects across Waves I, III and IV (Table 2). I examined three main categories of ACEs, including abuse (including sexual, physical, and emotional), neglect, and household dysfunction (attempted suicide or death by suicide of family members, foster care placement). The selection of these ACEs was guided by the ‘conventional ACEs’ included in the original ACE study by the CDC and Kaiser Permanente,^{16,17} as well as the list of ‘expanded ACEs.’⁵⁵

Physical abuse, sexual abuse, and emotional abuse (one question each) were modeled as binary variables (yes/no) based on frequency of occurrence; if the reported frequency was ≥ 1 , it was operationalized as “yes,” and “no” if the reported frequency was 0. Foster care placement was also modeled as binary (yes/no) based on incidence of occurrence.

Neglect was assessed as a composite of three questions and modeled as a binary variable (yes/no) based on frequency of occurrence: If the reported frequency was ≥ 1 , it was operationalized as “yes,” and “no” if the reported frequency was 0, for each question respectively. If a participant answered “yes” to any of the 3 questions, they were categorized as “yes” to having experienced neglect, and “no” if otherwise.

Suicide by family members was assessed via two questions pertaining to suicide attempt or death by suicide of family members and modeled as binary (yes/no): if the respondent answered “yes” to either question, they were categorized as “yes”, and “no” if otherwise. While the original Add Health survey question used the phrase “successful suicide,” I use the language “death by suicide” in accordance with changing protocols to avoid stigmatizing language such as

“successful suicide.”^{189,190} Table 2 provides detail on all six ACEs, period of data collection, survey questions, and operationalization.

Table 2. Adverse childhood experiences assessed in the National Longitudinal Study of Adolescent to Adult Health (1994-2018)¹

ACEs	Period of data collection	Survey questions	Operationalization
Abuse			
Physical	Wave IV	Before your 18 th birthday, how often did a parent or adult caregiver hit you with a fist, kick you, or throw you down on the floor, into a wall or down stairs?	Yes/No
Sexual	Wave IV	Before your 18 th birthday, how often did a parent or other adult caregiver touch you in a sexual way, force you to touch him or her in a sexual way, or force you to have sexual relations?	Yes/No
Emotional	Wave IV	Before your 18 th birthday, how often did a parent or other adult caregiver say things that really hurt your feelings or made you feel like you were not wanted or loved?	Yes/No
Neglect	Wave III	By the time you started 6 th grade, how often had your parents or other adult caregivers left you home alone when an adult should have been with you? How often had your parents or other adult caregivers not taken care of your basic needs, such as keeping you clean or providing food or clothing? How often had social services investigated how you were taken care of or tried to take you out of your living situation?	Yes/No
Household Dysfunction			

Table 2 (cont'd).

Suicide	Wave I	Have any of your family members tried to kill themselves during the past 12 months?	Yes/No
		Have any of your family members succeeded in killing themselves during the past 12 months?	
Foster care placement	Wave III	Did you ever live in a foster home?	Yes/No

¹These questions are from Add Health, funded by grant P01 HD31921 (Harris) from the Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD), with cooperative funding from 23 other federal agencies and foundations. Add Health is currently directed by Robert A. Hummer and funded by the National Institute on Aging cooperative agreements U01 AG071448 (Hummer) and U01AG071450 (Aiello and Hummer) at the University of North Carolina at Chapel Hill. Add Health was designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris at the University of North Carolina at Chapel Hill. Information on how to obtain the Add Health data files is available on the Add Health website (<https://addhealth.cpc.unc.edu>). No direct support was received from grant P01-HD31921 for this project.

3.4.2 Outcome

The primary outcome of interest was preterm delivery (PTD), defined as a birth occurring before 37 weeks of gestation. PTD was modeled as a binary variable (yes/no) based on women's self-reported information in Wave V based on the question "Was this baby born preterm?". Research demonstrates that the accuracy of gestational age data collected by maternal recall compares favorably to data obtained by medical records or birth certificates.^{191,192}

3.4.3 Covariates

Race/ethnicity. (White, Black, Hispanic, Asian, Pacific Islander, American Indian/Alaska Native, Other) was collected via self-report on the Wave V survey questionnaire. If a respondent reported multiple races, they were asked to indicate the racial group with which they best identified in a follow-up question. In this study, I define race as, "...a social classification based on phenotype that governs the distribution of risks and opportunities in our race-conscious society" (Jones, 2001, p. 300).¹⁹³ While race is a social construct and has no basis in biology or genetics, it can carry substantial physiological and psychosocial implications for the health and well-being of individuals and communities.^{193–196}

Furthermore, to promote grammatical justice and equity in reference to race,¹⁹⁷ I use intentional capitalization for all racial subgroups throughout the writing of this dissertation: "Black," "White," "Hispanic," "Asian," "Pacific Islander," and "American Indian/Alaska Native."¹⁹⁷ In addition, while I use six broad categories of race in this study, it is important to recognize that each group has considerable within-group heterogeneity and comprises diverse nationalities and ethnicities.

Socioeconomic status (SES). I used childhood SES, as opposed to adulthood SES, to measure socioeconomic status for study respondents, based on my conceptual framework as

previously discussed (Figure 1). Following the method developed by Slaughter-Acey et al. (2016), I captured SES as a composite construct of six indicators including mother's occupation, father's occupation, mother's education, father's education, annual household income, and receipt of public assistance (Table 3).¹⁶⁰ Each of these measures were converted into binary variables where 0 represented "low SES" and 1 represented "high SES".¹⁶⁰ Missing data on any of these variables were categorized as "low SES."¹⁶⁰ The scores for each of the six variables were then summed to create a composite SES variable with values ranging from 0 to 6. Based on the distribution of scores of the composite SES variable, three categories of SES were created: low (where score < lowest quartile (Q1) where Q1 = 2), middle (score \geq 2nd quartile (Q2) and \leq 3rd quartile (Q3) where Q2=2; Q3=3), and high (score > upper quartile (Q4) where Q4 = 3).¹⁶⁰

Table 3. Socioeconomic variables from Wave I (1994) of the National Longitudinal Study of Adolescent to Adult Health used to operationalize childhood socioeconomic status.¹

Socioeconomic variables	Low SES (0)	High SES (1)
Maternal occupation	Sales worker (i.e., insurance agent, store clerk)	Professional (i.e., doctor, lawyer, scientist)
Paternal occupation	Restaurant worker or personal service (i.e., waitress, housekeeper)	Professional (i.e., teacher, librarian, nurse)
	Craftsperson (i.e., toolmaker, woodworker)	Manager (i.e., executive, director)
	Construction worker (i.e., carpenter, crane operator)	Technical (i.e., computer specialist, radiologist)
	Mechanic (i.e., electrician, plumber, machinist)	
	Factory worker or laborer (i.e., assembler, janitor)	
	Transportation (i.e., bus driver, taxi driver)	
	Military or security (i.e., police officer, soldier, fire fighter)	
	Farm or fishery worker	
	Other	
	None	
	Missing	

Table 3 (cont'd).

Maternal education Paternal education	Eighth grade or less	Went to a business, trade, or vocational school after high school
	More than eighth grade, but did not graduate from high school	Went to college, but did not graduate
	Went to a business, trade, or vocational school	Graduated from a college or university
	High school graduate	Professional training beyond a four-year college or university
	Completed a GED	
	Never went to school	
	Missing	
Gross household income in 1994²	≤\$32,000	>\$32,000
	Missing	
Receipt of public assistance	Yes	No
	Missing	

¹Operationalization informed by the Slaughter-Acey method (Slaughter-Acey et al., 2016)¹⁶⁰

²Income threshold informed by the national gross median household income in the United States in 1994 (U.S. Census Bureau, 1996).¹⁹⁸

Religiosity & Spirituality. I assessed two types of potential protective factors, religiosity and spirituality, as potential effect modifiers of the association between adverse childhood experiences and PTSD. While religiosity and spirituality are related concepts and often used interchangeably or combined in the literature, they are distinct constructs with different meanings.^{174,175,199} In acknowledgment of these distinctions, I examined religiosity and spirituality individually, as well as a combined spirituality and religiosity composite variable (R/S) comprised of three items that examined aspects of both spirituality and religiosity (Table 4).

I modeled religiosity and spirituality (one question each) as continuous variables based on a Likert scale of responses, with values ranging from 0 (not religious at all; not spiritual at all)

to 3 (very religious; very spiritual), respectively (Table 4). I also developed a combined religiosity and spirituality variable (R/S), modeled as continuous, based on the sum of the scores across three questions which assessed both spiritual and religious beliefs. The values for this composite variable ranged from 3-15, where a higher score indicated higher R/S. These measures were collected during Add Health Wave III (2001-2002), when the women were young adults (ages 18-26). Thus, these variables were assessed after the period during which the occurrence of ACEs had already ended (before 18 years of age). See Table 4 for details on the assessment of religiosity and spirituality variables.

A comprehensive visual summary of the Add Health waves of data collection, respective ages of participants, and timing of assessment of all key variables in this dissertation is depicted in Figure 5.^{182,183}

Table 4. Religiosity and spirituality variables captured in Wave III (2001-2002) of the National Longitudinal Study of Adolescent to Adult Health, 1994-2018.¹

Variable	Item(s) in measure	Possible responses	Operationalization of variable
Religiosity	To what extent are you a religious person?	0- Not religious at all 1- Slightly religious 2- Moderately religious 3- Very religious	Continuous (0-3); higher score means higher religiosity
Spirituality	To what extent are you a spiritual person?	0- Not religious at all 1- Slightly religious 2- Moderately religious 3- Very religious	Continuous (0-3); higher score means higher spirituality
Combined religiosity and spirituality variable (R/S)	To what extent do you agree with the following statement? Angels are present to help or watch over me	1- Strongly disagree 2- Disagree 3- Neither agree nor disagree 4- Agree 5- Strongly agree	Continuous (3-15); higher score means higher spirituality/religiosity
	To what extent do you agree with the following statement? I employ my religious or spiritual beliefs as a basis for how to act and live on a daily basis.	1- Strongly disagree 2- Disagree 3- Neither agree or disagree 4- Agree 5- Strongly agree	
	To what extent do you agree with the following statement? What seem to be coincidences in my life are not really coincidences...I am being led "spiritually"	1- Strongly disagree 2- Disagree 3- Neither agree or disagree 4- Agree 5- Strongly agree	

¹These questions are from Add Health, funded by grant P01 HD31921 (Harris) from the Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD), with cooperative funding from 23 other federal agencies and foundations. Add Health is currently directed by Robert A. Hummer and funded by the National Institute on Aging cooperative agreements U01 AG071448 (Hummer) and U01AG071450 (Aiello and Hummer) at the University of North Carolina at Chapel Hill. Add Health was designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris at the University of North Carolina at Chapel Hill. Information on how to obtain the Add Health data files is available on the Add

Table 4 (cont'd).

Health website (<https://addhealth.cpc.unc.edu>). No direct support was received from grant P01-HD31921 for this project.

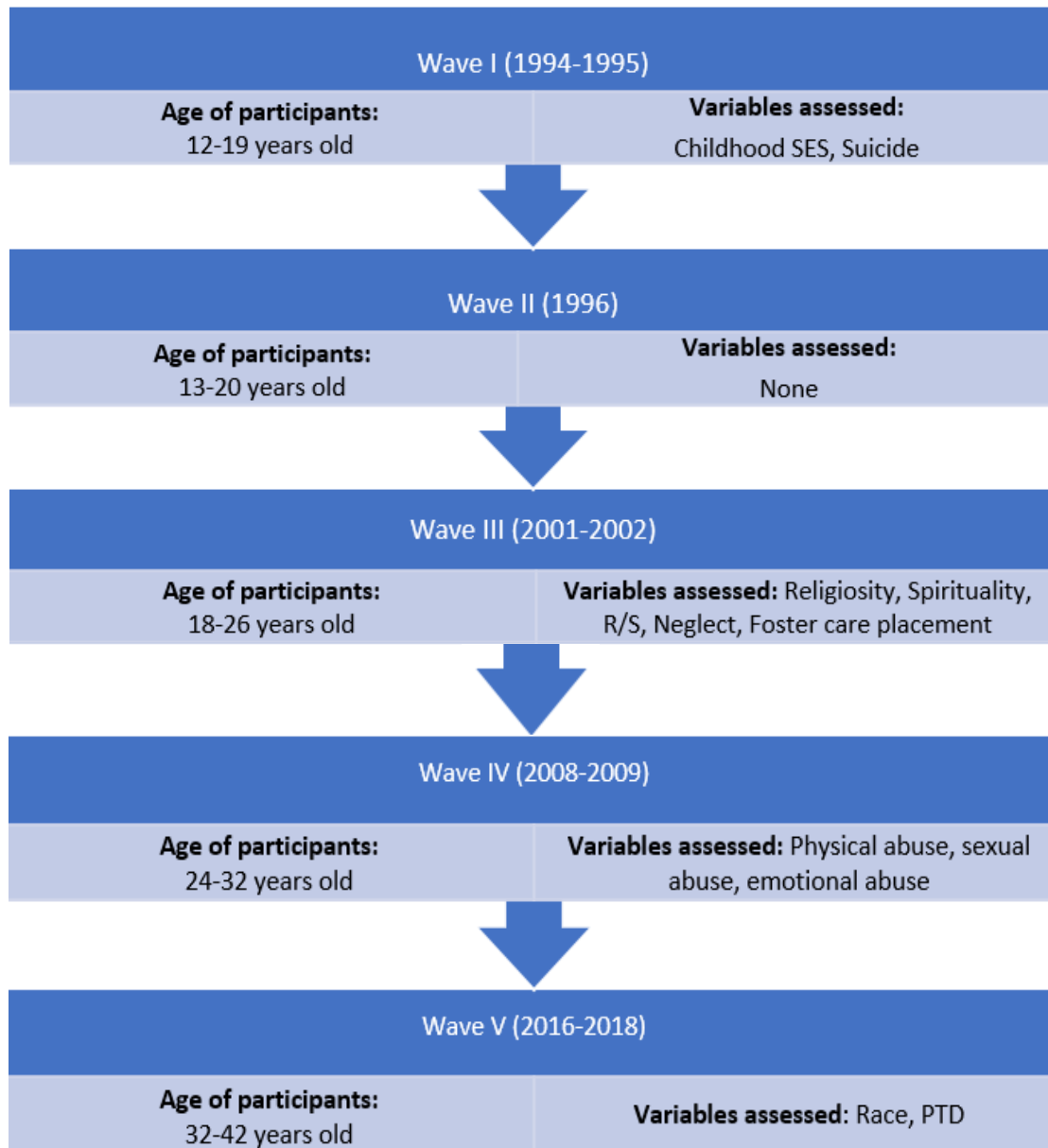


Figure 5. Timeline of Add Health data collection (1994-2018), respective ages of participants during each wave, and key Add Health variables assessed in this dissertation study.

3.5 Analytic Strategy

3.5.1 Aim 1: Methodologic Approach for Manuscript 1

3.5.1.1 Assessment of Missing Data.

While over 5% of the observations were missing data on ACEs, race, PTD, and weighting variables (n=267; 6.6%), the assessment of bias (Table 1) indicated that removal of these observations did not significantly change the makeup of the analytic sample compared to the reference population, lowering the likelihood of systematic bias, and increasing the internal validity of my findings. Therefore, these observations were removed from the reference population (n=3,897) to arrive at the final analytic sample of n=3,767 for Manuscript 1.

3.5.1.2 Descriptive Analyses

I conducted frequency procedures to derive the respective sample sizes and weighted percentages for the analytic sample by race, SES, maternal age, ACEs, and PTD variables. To assess statistically significant differences in the prevalence of PTD by race and SES, I used survey weighted Wald chi-square tests ($\alpha = 0.05$). To calculate the mean number of ACEs (mean ACE score) in the overall sample and within race and SES subgroups, and whether the means differed significantly across the groups, I used survey-weighted ANOVA procedures with Tukey post-hoc pairwise comparison tests (significance level of $\alpha=0.05$). To account for the stratified, clustered sampling structure of Add Health, all analyses were weighted. All analyses for Aim 1 (Manuscript 1) were conducted using SAS software statistical package 9.4 (Cary, NC).

3.5.1.3 Regression Analyses

I conducted unadjusted and adjusted survey-weighted logistic regression analyses to estimate the odds ratios (OR) for the association between six ACEs and PTD among women overall, as well as stratified within race and SES subgroups. The adjusted logistic regression

analyses for the overall sample of women controlled for both race and SES, while the adjusted logistic regression analyses stratified by race were adjusted for SES, and the SES-stratified analyses were adjusted for race. All six ACEs were included in each logistic regression model so that the ACEs controlled for the effects of one another. The logistic regression models estimated the odds ratios (OR) and their respective 95% confidence intervals (CI). I considered the odds ratios to be statistically significant if the null value of 1.0 was not included in the 95% confidence interval. If the upper or lower bound of the 95% CI of the odds ratio included the value of 1.0, I considered this as approaching statistical significance.

Equation 1.1 depicts the logistic regression model specification for the association between six ACEs and the log-odds of PTD, adjusting for race and SES as confounders. While the logistic regression model estimates the log-odds (or logit) of PTD (Equation 1.1), I report odds ratios throughout the manuscript for purposes of interpretability. White women and women with high SES were the reference groups for race and SES, respectively, as these groups represent the most privileged groups of women in the United States based on access to resources such as education and financial assets.

(1.1) $\text{logit}(Y_i) = \beta_0 + \beta_1(\text{sexual abuse}) + \beta_2(\text{physical abuse}) + \beta_3(\text{emotional abuse}) + \beta_4(\text{neglect}) + \beta_5(\text{suicide}) + \beta_6(\text{foster care placement}) + \beta_7(x_7) + \beta_8(x_8) + \beta_9(x_9) + \beta_{10}x_{10} + \beta_{11}x_{11} + \beta_{12}x_{12} + \beta_{13}x_{13} + \beta_{14}x_{14}$ where $Y_i = 1$ for PTD, and where 'White' is the reference group for race and 'High SES' is the reference group for SES such that:

$$x_7 = \begin{cases} 1 & \text{if Black} \\ 0 & \text{if not Black} \end{cases}; x_8 = \begin{cases} 1 & \text{if Hispanic} \\ 0 & \text{if not Hispanic} \end{cases}; x_9 = \begin{cases} 1 & \text{if Asian} \\ 0 & \text{if not Asian} \end{cases};$$

$$x_{10} = \begin{cases} 1 & \text{if Pacific Islander} \\ 0 & \text{if not Pacific Islander} \end{cases}; x_{11} = \begin{cases} 1 & \text{if AIAN} \\ 0 & \text{if not AIAN} \end{cases}; x_{12} = \begin{cases} 1 & \text{if Other} \\ 0 & \text{if not Other} \end{cases};$$

$$x_{13} = \begin{cases} 1 & \text{if low SES} \\ 0 & \text{if not low SES} \end{cases}; x_{14} = \begin{cases} 1 & \text{if middle SES} \\ 0 & \text{if not middle SES} \end{cases}$$

3.5.2 Aim 2: Methodologic Approach for Manuscript 2

3.5.2.1 Assessment of Missing Data

Less than 5% of the observations were missing data on PTD, race, and weighting variables (n=150; 3.7%). The assessment of bias (Table 1) showed that removal of these observations did not significantly change the composition of the analytic sample compared to the reference population, indicating a low probability of systematic bias, and promoting the internal validity of my findings. Therefore, this justified the removal of these observations with missing data from the reference population (n=4,034), which resulted in the final analytic sample of n=3,884 for Manuscript 2. As previously described, Full Information Maximum Likelihood was applied to address observations with missing data on ACEs (n=117), and therefore these observations were not removed from the analytic sample.

3.5.2.2 Application of Latent Class Analysis to Perinatal Epidemiology

To achieve Aim 2 of this dissertation work, I employed latent class analysis (LCA), a type of structural equation modeling (SEM) that allows for the identification of underlying, unobservable subgroups of individuals who exhibit similarities on certain characteristics within a heterogeneous population.^{187,200–202} LCA, also known as finite mixture modeling, reflects a “person-centered” methodologic technique because it pays special attention to the similarities and differences in individuals’ response patterns across various characteristics, as opposed to variable-centered approaches, which aim to determine overall relationships between variables in a population without necessarily paying attention to individual variability in those relationships.^{200,201,203} Person-centered methods are becoming increasingly popular analytic techniques in the social and behavioral sciences, especially in the context of developmental research.^{200,201,203}

There are several limitations to the variable-centered approach that the person-centered approach addresses. For example, a lack of attention to individual variability in the relationships between variables is one of the primary methodological limitations of the variable-centered approach.²⁰³ To this point, Bogat et al. (2016) explain, "...rarely is it the case that models generated using all subjects in a sample or population describe specific individuals or subgroups. Researchers seldom decompose these models to examine possible subgroup differences" (p. 802).²⁰³ Furthermore, a lack of a significant main effect discovered between variables does not necessarily mean that underlying relationships do not exist across subgroups of the sample or population.^{203,204}

As a case study example, in Zamani-Hank et al. (2022) we found that six types of protective factors (self-esteem, mastery, religiosity, perceived, emotional, and instrumental social support) were not significantly associated with preterm delivery in the overall sample of pregnant women (i.e., there were no main effects).⁸¹ Based on these results only, one could erroneously conclude that the examined protective factors are not protective against PTD among all groups of women included in the study (White, Black, low SES, middle SES, and high SES). However, upon looking at the interaction effects between protective factors and race and socioeconomic status, respectively, a different relationship was observed. Namely, we found that religiosity was associated with 40% lower odds of PTD among low SES women (OR 0.6, 95% CI 0.4-0.9) and among Black women (OR 0.6, 95% CI 0.4–1.0).⁸¹ The key takeaway from this study was that protective factors may not operate the same across all subgroups of women, a conclusion that informed the development of this dissertation work to incorporate religiosity and spirituality as potential protective factors to examine in the relationship between ACEs and PTD.

However, while testing for interaction effects can be helpful in identifying differences in relationships between variables across subgroups in the population, these methods are not equipped to identify broader, underlying patterns in individuals' responses to all variables used in a study, particularly if using a large number of variables and a large sample size. To identify patterns in responses in all variables in a study, one would need to analyze the entire matrix (i.e., a contingency table or cross-tabulation) of responses.¹⁸⁷

As an example, in this dissertation, I assess the latent construct of childhood adversity by using six different binary indicators of ACEs. Because there are six ACE indicators and two possible responses for each indicator (yes/no), the total number of possible response patterns in the contingency table would be $2^6 = 64$. In application of the example discussed by Collins & Lanza (2010, pp. 25-26)¹⁸⁷ to my research, a table of example response patterns for the six ACE indicators in my study is depicted in Table 5. It would take a great deal of time to assess the response patterns for all of the $n=3,884$ women in my sample. Thus, the purpose of latent class analysis is to assess the types of response patterns across all individuals in the sample, as well as the frequency of individuals who report specific response patterns, to determine whether there are underlying subgroups of individuals based on the pattern of these responses.¹⁸⁷ This is one of the primary advantages that latent class techniques offer.¹⁸⁷

Table 5. Example response patterns across six ACE indicators of the latent class model for n=64 total response patterns.

Adverse Childhood Experiences						
	Sexual abuse	Physical abuse	Emotional abuse	Neglect	Suicide	Foster care placement
Response pattern 1	Yes	No	Yes	Yes	No	No
Response pattern 2	Yes	Yes	Yes	Yes	Yes	Yes
Response pattern 3	No	No	No	No	No	No
.....						
.....						
Response Pattern 64	Yes	No	Yes	Yes	No	Yes

While there is a growing body of literature applying person-centered approaches in perinatal epidemiology,^{60,159,205} more perinatal studies would benefit from the application of these techniques to acquire an enhanced understanding of the variation in, and interactions between, diverse risk and protective factors that may impact persistent racial disparities in adverse birth outcomes among women. Referencing this need for person-centered approaches in perinatal epidemiology, Deichen Hansen (2021, p. 2)²⁰⁵ states,

“....although enthusiasm for holistic analyses have been embraced conceptually within the perinatal health field, existing studies on racial disparities have largely been characterized by variable-center approaches that fail to capture the complexity of women’s lived experiences and that have led to a “fundamental mismatch” between the field’s predominantly linear analytic methods (such as regression analyses) and the desire to engage in person-centered work. In order to enhance public health’s capacity to further promote equitable perinatal health outcomes, research must reflect the vast heterogeneity among pregnant US women that contributes to variation in perinatal health outcomes” (Deichen Hansen, 2021, p. 2).²⁰⁵

Elaborating upon these points by Deichen Hansen (2021), the varying prevalence of adverse birth outcomes by race and socioeconomic status, as well as the wide array of hypothesized physiological and psychosocial correlates of PTD and disparities in PTD, reflects a

high degree of variability or intra-group differences among women who give birth in the United States.²⁰⁵ Therefore, methodological approaches which better account for this variability across subgroups of women and aim to close the gap of this “fundamental mismatch” of methods in the field of perinatal epidemiology are necessary (Deichen Hansen, 2021, p. 2).^{205,206} Therefore, in this dissertation work I apply this person-centered approach using latent class analysis to examine subgroup differences in a potentially important risk factor for disparities in PTD, adverse childhood experiences, as well as subgroup differences in the relationship between ACEs, potential protective factors, and PTD by race and socioeconomic status subgroups.

3.5.2.3 Conceptual Diagram and Selection of Indicators

Latent class analysis assumes that there are latent, or unobservable, homogeneous groups within a heterogeneous population that cannot be directly observable but captured through observable variables or characteristics, known as indicators.¹⁸⁷ Latent class models assume that indicators are determined by two factors: 1) the latent variable and 2) measurement error, which is associated with each indicator.¹⁸⁷ Unlike standard regression methods, structural equation models do not assume that variables are assessed perfectly; hence, the models account for errors in measurement of the variables.^{187,207}

Latent class models incorporate a key assumption called the *local independence assumption*, which states that within a specific latent class, the observed indicators must be independent of one another (Collins & Lanza, 2010, pp. 45-47).¹⁸⁷ However, observed indicators are allowed to correlate in the dataset overall.¹⁸⁷ This is depicted in my conceptual diagram in Figure 6, as there are no arrows which connect the indicators to each other.

Informed by this latent class framework, six binary measures of ACEs were selected for the latent class analysis: physical abuse, sexual abuse, emotional abuse, neglect, suicide attempt

or death by a family member, and foster care placement. The selection of these ACEs was informed by the literature on both conventional and expanded categories of ACEs.^{17,55} Figure 6 depicts the path diagram of this hypothesized latent class model. All latent class analyses were conducted using Mplus Version 8 (Muthén & Muthén, 1998-2017).²⁰⁸

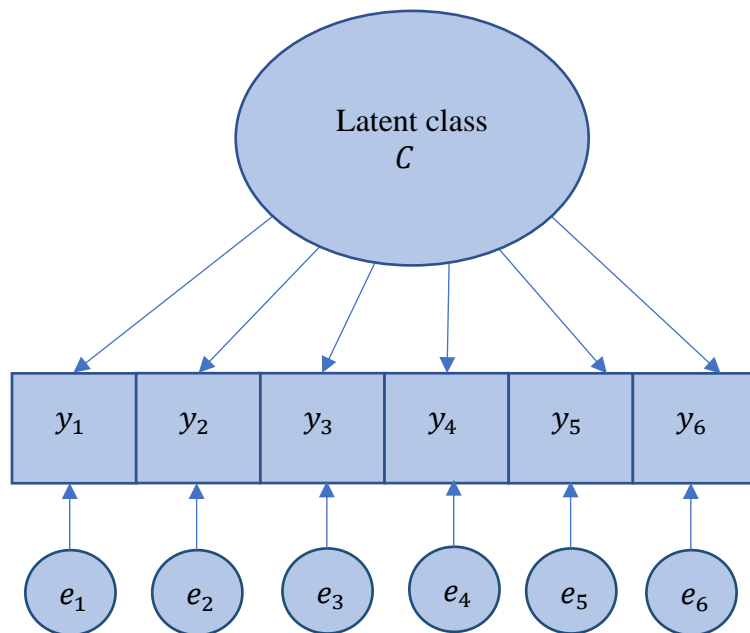


Figure 6. Path diagram of the six ACE indicators used to inform the latent class C in the National Longitudinal Study of Adolescent to Adult Health (n=3,884), 1994-2018 where y1 (physical abuse); y2 (sexual abuse); y3 (emotional abuse); y4 (neglect); y5 (attempted suicide or death by family member(s)); y6 (foster care placement). The measurement error associated with each indicator is denoted by e_1 - e_6 .

3.5.2.4 Class Enumeration and Evaluation of Model Fit

Identifying the appropriate number of latent classes (i.e., a process called class enumeration) is based upon evaluation of optimal model fit.^{187,209} As discussed by Collins and Lanza (2010), optimal model fit is assessed by a combination of statistical criteria (i.e., fit statistics) and parsimony (i.e., the simplicity of a model based on the number of parameters).¹⁸⁷ To identify the number of latent classes characterized by patterns of six adverse childhood

experiences occurring before 18 years of age, I performed latent class analysis for discrete outcomes using Mplus Version 8.²⁰⁸ To determine the best-fitting model across 2 through 4 classes, I compared values for the most commonly used fit statistics: entropy, Akaike information criterion (AIC), Bayesian information criterion (BIC), consistent Akaike information criterion (CAIC), sample size-adjusted Bayesian information criterion (ssBIC). The model that exhibited the lowest overall values for these fit statistics was selected as the best-fitting model.

After selection of the two-class model with the best fit, I assessed two key parameter estimates for the latent class model: 1) the latent class prevalences (denoted by gamma, γ) and the 2) item-response probabilities (denoted by rho, ρ).¹⁸⁷ These estimates were derived via robust maximum likelihood estimation in Mplus.

The latent class prevalences refer to the estimated proportion of individuals assigned to each latent class of the model, also known as class proportions or mixing probabilities.^{187,210} The sum of the prevalences (i.e., probabilities) across all latent classes is equal to one.¹⁸⁷ The item-response probabilities, also known as conditional within-class probabilities, is the probability of reporting a specific response (i.e., “yes” or “no” for the binary indicators), given that an individual is in a specific latent class, known as latent class membership.^{187,210} The class with the highest estimated prevalence (i.e., with the largest proportion of individuals in the class) was designated as the reference class. Thus, the latent class model is primarily defined by the latent class prevalences and the item-response probabilities, as depicted in Equation 1.2 (Collins & Lanza, 2020, p. 41).¹⁸⁷

$$(1.2) \quad P(Y = y) = \sum_{c=1}^C \gamma_c \prod_{j=1}^J \prod_{r_j=1}^{R_j} \rho_{j,r_j|c}^{I(y_j=r_j)}$$

where $P(Y=y)$ is the overall probability (P) of observing a pattern of responses (Y) equal to a specific response pattern for a particular ACE indicator; Σ is the sum of all the latent class prevalences (γ) across the total number of latent classes (C); c = the specific latent class (i.e., latent class 1 vs. 2); $\prod_{j=1}^J \prod_{r_j=1}^{R_j} \rho_{j,r_j|c}^{I(y_j=r_j)}$ is the conditional within-class probability where J = total number of ACE indicators (i.e., 6); j = a specific indicator (e.g., neglect); r_j = response to a specific ACE indicator j (e.g., reporting “yes” to neglect); $\rho_{j,r_j|c}$ is the probability of observing a specific response (e.g., “yes” to neglect), given membership in a specific latent class c ; I represents an indicator variable for which the response pattern of a specific ACE (y_j) is equivalent to a specific value for that indicator (r_j ; i.e., 1 to indicate “yes” for neglect; and 0 to indicate “no” for neglect). Equation obtained from Collins & Lanza, 2020, p. 41.¹⁸⁷

3.5.2.5 Prediction of Latent Class Membership by Covariates

To evaluate whether latent class membership differs by two covariates of interest, race and socioeconomic status, I implemented Vermunt’s three-step approach²¹¹ using Mplus 8²⁰⁸. In summary of the steps outlined by Bauer & Steinley (2020, p. 8.8-8.13), Step 1 of Vermunt’s approach entails fitting the latent class model without the covariates of interest, while Step 2 performs modal class assignment, a classification process by which study participants are assigned to the class for which they have the highest probability of belonging to (i.e., highest posterior probability).^{210,211} In addition to deriving class assignment, Step 2 also calculates log-odds for the classification probabilities, which represent the classification error, or the degree to which individuals are correctly placed in the class to which they truly belong.^{210,211}

In Step 3, the modal class assignment is then treated as a proxy variable to represent the latent class that each individual belongs to, while the logits derived from Step 2 are entered into the model to indicate the error level associated with modal class assignment.^{210,211} Ultimately, Step 3 provides the regression coefficients for regressing latent class on race and SES to assess whether the likelihood of latent class membership differs by race and SES.²¹⁰ This step also calculates the odds ratio, which represents, for an individual in a specific race or SES group, the odds of being in one latent class compared to reference group (White women for race; high SES women for SES).

Because the race and SES variables included more than two categories, dummy variables were created to facilitate the interpretation of comparisons between groups. All latent class models conducted in Mplus were survey-weighted. Equation 1.3 states the logistic regression model specification for the prediction of latent class membership by race and SES, where race and SES control for one another. Figure 7 illustrates the path diagram for the prediction of latent class membership by race and SES.

(1.3) $\text{logit}(Y_i) = \beta_0 + \beta_1(x_1) + \beta_2(x_2) + \beta_3(x_3) + \beta_4(x_4) + \beta_5(x_5) + \beta_6(x_6) + \beta_7(x_7) + \beta_8(x_8)$ where $Y_i = 1$ for membership in class i and where 'White' is the reference group for race and 'High SES' is the reference group for SES such that:

$$x_1 = \begin{cases} 1 & \text{if Black} \\ 0 & \text{if not Black} \end{cases}; x_2 = \begin{cases} 1 & \text{if Hispanic} \\ 0 & \text{if not Hispanic} \end{cases}; x_3 = \begin{cases} 1 & \text{if Asian} \\ 0 & \text{if not Asian} \end{cases};$$

$$x_4 = \begin{cases} 1 & \text{if Pacific Islander} \\ 0 & \text{if not Pacific Islander} \end{cases}; x_5 = \begin{cases} 1 & \text{if AIAN} \\ 0 & \text{if not AIAN} \end{cases}; x_6 = \begin{cases} 1 & \text{if Other} \\ 0 & \text{if not Other} \end{cases};$$

$$x_7 = \begin{cases} 1 & \text{if low SES} \\ 0 & \text{if not low SES} \end{cases}; x_8 = \begin{cases} 1 & \text{if middle SES} \\ 0 & \text{if not middle SES} \end{cases}$$

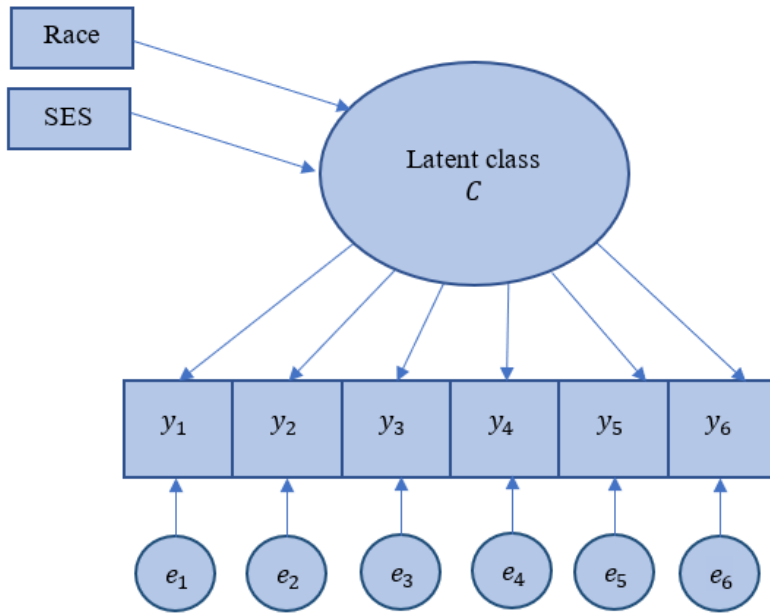


Figure 7. Path diagram for the prediction of latent class membership by two covariates, race and socioeconomic status, in the National Longitudinal Study of Adolescent to Adult Health (n=3,884), 1994-2018 where y1 (physical abuse); y2 (sexual abuse); y3 (emotional abuse); y4 (neglect); y5 (attempted suicide or death by family members); y6 (foster care placement); e₁-e₆ (measurement error for each indicator).

3.5.2.6 LCA with Distal Outcome Analysis

To evaluate whether class membership predicts the distal outcome of PTSD, Vermunt's manual three-step approach was used,²¹¹ as is required for models with discrete distal outcomes with non-normal distributions.²¹⁰ The three-step approach for distal outcomes is identical to the approach used in section 3.4.2.4 for assessing class predictors, except that in Step 3, a model parameter constraint was added to assess whether the threshold of PTSD differs across the classes.²¹⁰ Because PTSD is a binary variable and not a continuous variable, it does not exhibit a mean, but a threshold, which is a log-odds parameter that is used to calculate the within-class probability of the outcome.²¹⁰ The relationship between the probability of an outcome and the

threshold parameter is depicted in Equation 1.4 (Bauer & Steinley, 2020, p. 7.4).²¹⁰ A Wald test was conducted to assess whether the thresholds of PTD differed significantly across classes ($\alpha = 0.05$). Figure 8 depicts the conceptual diagram of the distal outcome analysis. The model specification for the logistic regression between PTD and latent class membership is illustrated in Equation 1.5.

(1.4) $\delta_{jk} = \frac{1}{1 + e^{v_{jk}}}$ where δ_{jk} is the probability of reporting “yes” to PTD for an individual in class k , and v_{jk} is the threshold parameter estimate (Bauer & Steinley, 2020, p. 7.4).²¹⁰

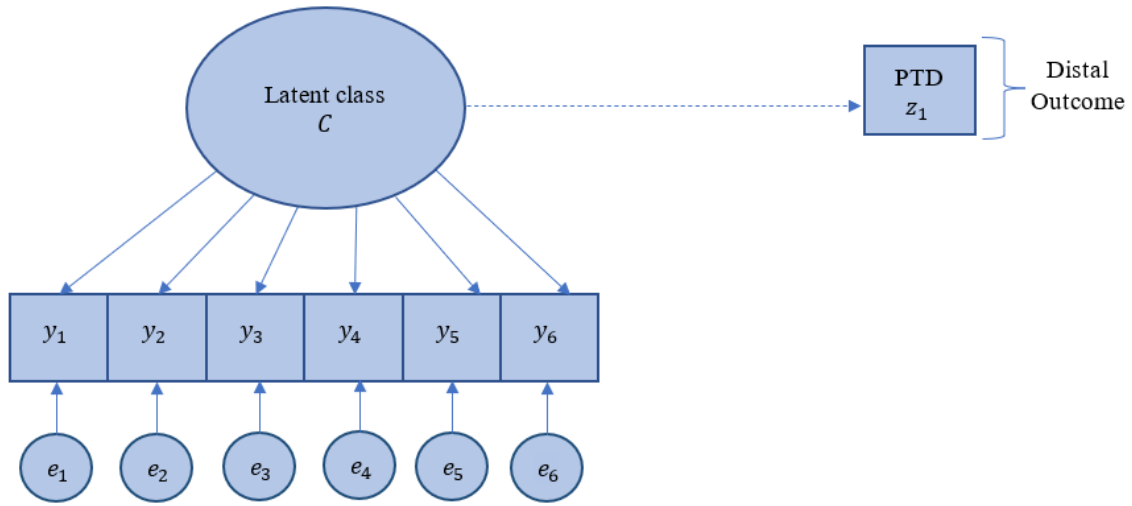


Figure 8. Path diagram of the latent class distal outcome analysis of the association between latent class membership and PTD in the National Longitudinal Study of Adolescent to Adult Health (n=3,884), 1994-2018 where y1 (physical abuse); y2 (sexual abuse); y3 (emotional abuse); y4 (neglect); y5 (attempted suicide or death by family members); y6 (foster care placement); z1 (PTD); e1-e6 (measurement error for each indicator)

(1.5) $\text{logit}(Y_i) = \beta_0 + \beta_1(x_{1,i}) + \beta_2(x_2) + \beta_3(x_3) + \beta_4(x_4) + \beta_5(x_5) + \beta_6(x_6) + \beta_7(x_7) + \beta_8(x_8) + \beta_9(x_9)$ where $Y_i = 1$ for PTB, $x_1 = \text{membership in class } i$, and where ‘White’ is the reference group for race and ‘High SES’ is the reference group for SES such that:

$$x_2 = \begin{cases} 1 & \text{if Black} \\ 0 & \text{if not Black} \end{cases}; x_3 = \begin{cases} 1 & \text{if Hispanic} \\ 0 & \text{if not Hispanic} \end{cases}; x_4 = \begin{cases} 1 & \text{if Asian} \\ 0 & \text{if not Asian} \end{cases};$$

$$x_5 = \begin{cases} 1 & \text{if Pacific Islander} \\ 0 & \text{if not Pacific Islander} \end{cases}; x_6 = \begin{cases} 1 & \text{if AIAN} \\ 0 & \text{if not AIAN} \end{cases}; x_7 = \begin{cases} 1 & \text{if Other} \\ 0 & \text{if not Other} \end{cases};$$

$$x_8 = \begin{cases} 1 & \text{if low SES} \\ 0 & \text{if not low SES} \end{cases}; x_9 = \begin{cases} 1 & \text{if middle SES} \\ 0 & \text{if not middle SES} \end{cases}$$

3.5.3 Specific Aim 3: Methodologic Approach for Manuscript 3

3.5.3.1 Assessment of Missing Data

Less than 5% of the observations had missing data on PTD, race, and weighting variables (n=150; 3.7%). The assessment of bias (Table 1) indicated that removal of these observations did not significantly change the representativeness of the analytic sample with regard to the reference population. This indicates that there is a low probability of selection bias which enhances the internal validity of the findings. Therefore, these observations were removed from the reference population (n=4,034) to arrive at the final analytic sample of n=3,884 for Manuscript 3. As previously described, Full Information Maximum Likelihood was applied to address observations with missing data on ACEs (n=117), and therefore these observations were not removed from the analytic sample.

3.5.3.2 Latent Class Moderation Analysis

To determine whether the association between latent class membership and PTD varied across levels of three potential protective factors (religiosity, spirituality, and R/S, respectively), I implemented a latent class moderation analysis using Vermunt's manual three-step approach.^{210,211} Steps 1 and 2 are identical to the approaches previously described in Section 3.4.2. In Step 3, modal class assignment is designated as the proxy variable for latent class membership and PTD is regressed on each respective protective factor in the overall model statement as well as for each latent class statement. Unadjusted and adjusted models (controlling

for race and SES) were conducted for the overall sample (n=3,884). In addition, I conducted race-stratified analyses (adjusted for SES) and SES-stratified analyses (adjusted for race) to determine whether the effect modification by potential protective factors of the association between latent class membership and PTD differed within race and SES subgroups.

Because latent class membership cannot be treated as an independent variable on which the outcome variable can be regressed in Mplus, effect modification by the respective protective factors was inferred by examining whether the effect of PTD on religiosity, spirituality, and R/S, respectively, differed significantly across the classes as informed by a Wald test ($\alpha = 0.05$). Because interaction effects are considered symmetric, if a variable M modifies the relationship between exposure (X) and outcome (Y), then X also modifies the relationship between M and Y.²¹² Therefore, if the Wald test provides evidence that the relationship between religiosity and PTD differs significantly by latent class membership, this is statistically equivalent to stating that the relationship between latent class membership and PTD differs by religiosity. While statistically equivalent, my conceptual framework informs my conceptualization of religiosity, spirituality, and R/S as effect modifiers, not latent class membership.

Based on the findings of the Wald Test, I conducted supplemental analyses to evaluate the direction of the effect modification i.e., to evaluate how the probability of PTD differed across the four levels of religiosity and spirituality in the overall sample as well as across the 12 levels of R/S (Manuscript 3). Supplemental analyses were not conducted for the race and SES stratified models. Equation 1.6 depicts the model specification for the effect modification analysis and Figure 9 depicts the conceptual diagram of the latent class moderation analysis.

$$(1.6) \logit(Y_i) = \beta_0 + \beta_1(x_{1,i}) + \beta_2(x_2) + \beta_3(x_3 * x_2) + \beta_4(x_4) + \beta_5(x_5) + \beta_6(x_6) + \beta_7(x_7) + \beta_8(x_8) + \beta_9(x_9) + \beta_{10}(x_{10}) + \beta_{11}(x_{11})$$

where $Y_i = 1$ for PTB, $x_1 =$ membership in class i , $x_2 =$ protective factor (i.e., religiosity, spirituality, R/S, respectively), $x_3 * x_2 =$ interaction term between class membership and protective factor; and where

‘White’ is the reference group for race and ‘High SES’ is the reference group for SES such that:

$$x_4 = \begin{cases} 1 & \text{if Black} \\ 0 & \text{if not Black} \end{cases}; x_5 = \begin{cases} 1 & \text{if Hispanic} \\ 0 & \text{if not Hispanic} \end{cases}; x_6 = \begin{cases} 1 & \text{if Asian} \\ 0 & \text{if not Asian} \end{cases};$$

$$x_7 = \begin{cases} 1 & \text{if Pacific Islander} \\ 0 & \text{if not Pacific Islander} \end{cases}; x_8 = \begin{cases} 1 & \text{if AIAN} \\ 0 & \text{if not AIAN} \end{cases}; x_9 = \begin{cases} 1 & \text{if Other} \\ 0 & \text{if not Other} \end{cases};$$

$$x_{10} = \begin{cases} 1 & \text{if low SES} \\ 0 & \text{if not low SES} \end{cases}; x_{11} = \begin{cases} 1 & \text{if middle SES} \\ 0 & \text{if not middle SES} \end{cases}$$

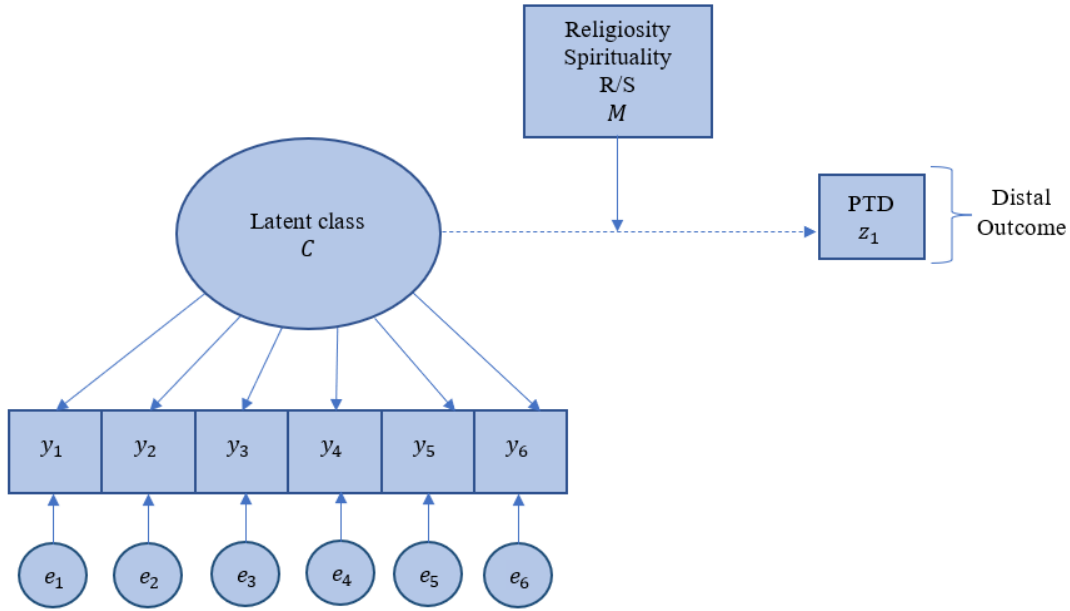


Figure 9. Conceptual diagram of the moderation analysis of the association between latent class membership and PTD by religiosity, spirituality, and R/S in the National Longitudinal Study of Adolescent to Adult Health (n=3,884), 1994-2018 where y1 (physical abuse); y2 (sexual abuse); y3 (emotional abuse); y4 (neglect); y5 (attempted suicide or death by family members); y6 (foster care placement); z1 (PTD); e1-e6 (measurement error for each indicator); and M represents each of the three potential protective factors, religiosity, spirituality, and R/S.

3.6 Human Subjects Protection and Institutional Review Board Determination

This research project was determined exempt from human subjects research under 45 CFR 46.104(d) 4(ii) by the Institutional Review Board (IRB) at Michigan State University on February 10, 2020 (Appendix Figure A1). Furthermore, our data-use agreement (DUA) to access the restricted-use Add Health dataset to conduct this dissertation work was approved by the Carolina Population Center at the University of North Carolina at Chapel Hill on April 16, 2020 (PI: Dr. Claire Margerison, Contract #02042001). The restricted-use dataset received from Add Health is de-identified and therefore contains no names, IDs, addresses, or full dates to ensure maximum protection for the study subjects. Per the DUA, annual progress reports were submitted to Add Health and approved on May 12, 2022, and April 13, 2021.

3.7 Add Health Restricted Data Use Agreement and Contractual Acknowledgement

This research uses data from Add Health, funded by grant P01 HD31921 (Harris) from the Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD), with cooperative funding from 23 other federal agencies and foundations. Add Health is currently directed by Robert A. Hummer and funded by the National Institute on Aging cooperative agreements U01 AG071448 (Hummer) and U01AG071450 (Aiello and Hummer) at the University of North Carolina at Chapel Hill. Add Health was designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris at the University of North Carolina at Chapel Hill.

CHAPTER 4: RACIAL AND SOCIOECONOMIC DISPARITIES IN THE ASSOCIATION BETWEEN ADVERSE CHILDHOOD EXPERIENCES AND PRETERM DELIVERY

4.1 Introduction

Preterm delivery (PTD), which occurs when a baby is born earlier than 37 weeks of gestation, constitutes a major child and maternal public health problem in the United States, with stark racial and socioeconomic disparities in PTD which have existed for decades. Specifically, the prevalence of PTD is highest among Black women, at 14.4% as of 2020, compared to 9.1% for White women.² A disproportionate exposures to life stressors among women is hypothesized as an important pathway through which the risk for PTD may be influenced.^{11,15,92,94}

Adverse childhood experiences (ACEs), or adverse life events occurring before age 18, are a unique type of life stressor that have been associated with a multitude of adverse physical, mental, and behavioral health outcomes.^{16,17} Importantly, disparities in ACE prevalence also exist by race and socioeconomic status.^{28–30} Because adverse childhood experiences occur during a sensitive period of childhood and adolescence, their occurrence is hypothesized to trigger a series of physiological changes that may influence neuroendocrine, immune system, and reproductive system functioning, and therefore future disease risk in adulthood.^{32,34–36,51,83}

While there is a growing literature studying the impacts of ACEs on adverse reproductive and pregnancy-related health outcomes among women,^{19–21,39} there is an overall gap in the literature of studies which have examined the impacts of ACEs on preterm delivery. While a recent systematic review by Sulaiman et al. (2020) identified nine studies which have examined the association between total number of ACEs and PTD, most of these studies did not **1)** use large, nationally representative datasets; **2)** pay particular attention to examining whether the impacts of ACEs on PTD risk differed across race and socioeconomic status subgroups using

person-centered approaches nor did they **3)** assess whether specific ACEs were associated with higher risk of PTD than others, as most studies used a cumulative count of ACEs.¹⁸

Therefore, the overall goal of my study was to address these gaps in the literature by determining the association between specific adverse childhood experiences and odds of PTD and evaluating whether this relationship differs across race and SES using a nationally representative sample of n=3,767 women from the National Longitudinal Study of Adolescent to Adult Health (Add Health; 1994-2018). Specifically, my study aimed to **1)** Examine whether differences exist in the mean ACE score by race and socioeconomic status among women who give birth and **2)** Assess the association between six ACEs and PTD among women overall, as well as within race and SES subgroups.

4.2 Methods

4.2.1 Study design, dataset, and study population

I used the National Longitudinal Study of Adolescent to Adult Health (Add Health), a nationally representative longitudinal study of adolescents initiated in 1994 with the objective of assessing the diverse health outcomes and health behaviors of adolescents in the United States and to subsequently evaluate their health and development into adulthood.¹⁸² Add Health comprises five waves of data follow-up: Wave I (1994-1995), Wave II (1996), Wave III (2001-2002), Wave IV (2008-2009), and Wave V (2016-2018) and includes in-school, at-home, and parental components of survey administration.^{182,183}

Add Health used a school-based, multi-stage sampling design, whereby the primary sampling unit consisted of schools and the secondary sampling unit consisted of students from those schools.^{183,184} The final study sample consisted of 90,118 students for the in-school survey component, of which, n = 20,745 students were selected to participate in the at-home survey

component and to be followed up in all subsequent waves of follow-up (also known as the ‘core sample’).¹⁸³ The at-home survey administration, which is the primary focus of the current study, employed computer-assisted personal interview (CAPI) and Audi Computer-Assisted Self-Interview (ACASI) techniques for all questionnaires to enhance the validity of self-reported data.^{183,186}

After excluding male adolescents from our sample (n=10,263) and female adolescents who did not have a pregnancy that ended in a live birth (n=4,034) by 2018, I arrived at my reference population of n = 4,034 female adolescents (Figure 10). Next, I excluded female adolescents who did not report data on PTD for the first birth in Wave V (n=71) and removed observations with missing information on any weighting variables (n=66), race (n=13), and ACEs (n=139), to arrive at my final analytic sample of n = 3,767 female adolescents (Figure 10).

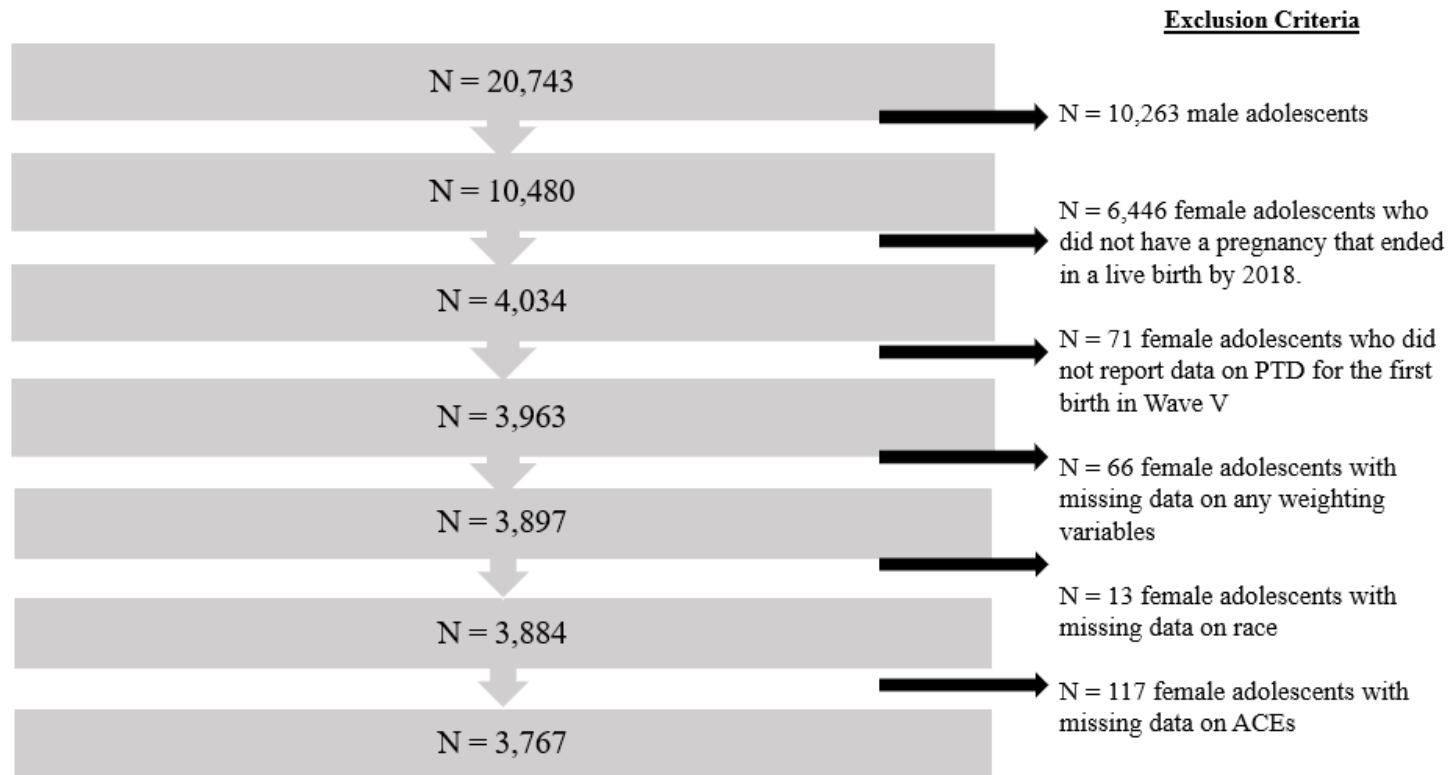


Figure 10. Analytic sample used to assess the association between ACEs and PTD in the National Longitudinal Study of Adolescent to Adult Health (n=3,767), 1994-2018

4.2.2 Measures

Exposure variables. I assessed adverse childhood experiences (ACEs) as my exposure variable. Specifically, I examined seven types of ACEs across three broad categories of ACEs: 1) abuse (sexual abuse, physical abuse, emotional abuse), 2) neglect, and 3) household dysfunction (attempted suicide or death by family members, foster care placement). The selection of these ACEs was guided by the ‘conventional ACEs’ included in the original ACE study by the CDC and Kaiser Permanente,¹⁷ as well as the list of ‘expanded ACEs.’⁵⁵

I operationalized physical, sexual, and emotional abuse (one question each), and neglect (based on a composite of three questions), by creating a binary variable based on the frequency of occurrence: “yes” (1) for reported frequency of ≥ 1 , “no” (0) for reported frequency of 0. Suicide was assessed as a composite based on two questions inquiring whether family members have attempted to kill themselves or whether they “succeeded in committing suicide” in the past 12 months and modeled as a binary variable (yes/no). While the original survey question included the phrase “successful suicide,” I use the language “death by suicide” in accordance with growing calls in the field of psychology to avoid stigmatizing language such as “successful suicide.”^{189,190} Appendix Table B1 summarizes the six ACEs, the period of data collection, survey questions, and operationalization for the current study.

Outcome variable. My primary outcome of interest is preterm delivery (PTD), defined as a delivery that occurs before 37 weeks of gestation. PTD was collected by respondents by self-report and modeled as a binary variable (Yes/No). Studies demonstrate that the accuracy of maternal recall of gestational age compares favorably with data obtained from medical records or birth certificates.^{191,192}

Covariates.

Race/ethnicity. (White, Black, Hispanic, Asian, Pacific Islander, American Indian/Alaska Native, Other) was collected via self-report. If a respondent self-reported as multi-racial, a follow-up question was asked to inquire about the racial category with which they best identified with. In this study, I refer to race as, "...a social classification based on phenotype that governs the distribution of risks and opportunities in our race-conscious society" (Jones, 2001, p. 300).¹⁹³ Race is not a biological construct but a social construct that can significantly impact people's health and well-being.^{193–196} Furthermore, while I use six categories of race in this study, I acknowledge that there is a diverse array of ethnicities and nationalities within each group. Finally, throughout this paper, I intentionally capitalize the names for all racial subgroups to promote language that enhances grammatical racial equity.¹⁹⁷

Childhood socioeconomic status. Childhood SES was captured as a composite construct of six indicators including mother's occupation, father's occupation, mother's education, father's education, annual household income, and receipt of public assistance (Appendix Table B2).¹⁶⁰ Guided by the method developed by Slaughter-Acey et al. (2016), each of these variables were converted into binary variables where 0 represented "low SES" and 1 represented "high SES".¹⁶⁰ Missing data on any of these variables were categorized as "low SES."¹⁶⁰ The scores for each of the six variables were then summed to create a composite SES variable with values ranging from 0 to 6.¹⁶⁰ Based on the distribution of scores of the composite SES variable, three categories of SES were created: low (where score < lowest quartile (Q1) where Q1 = 2), middle (score ≥ 2nd quartile (Q2) and ≤ 3rd quartile (Q3) where Q2=2; Q3=3), and high (score > upper quartile (Q4) where Q4 = 3).¹⁶⁰ Figure 11 depicts a summary of the Add Health waves of data collection, respective ages of participants, and timing of assessment of all key variables in this study.^{182,183}

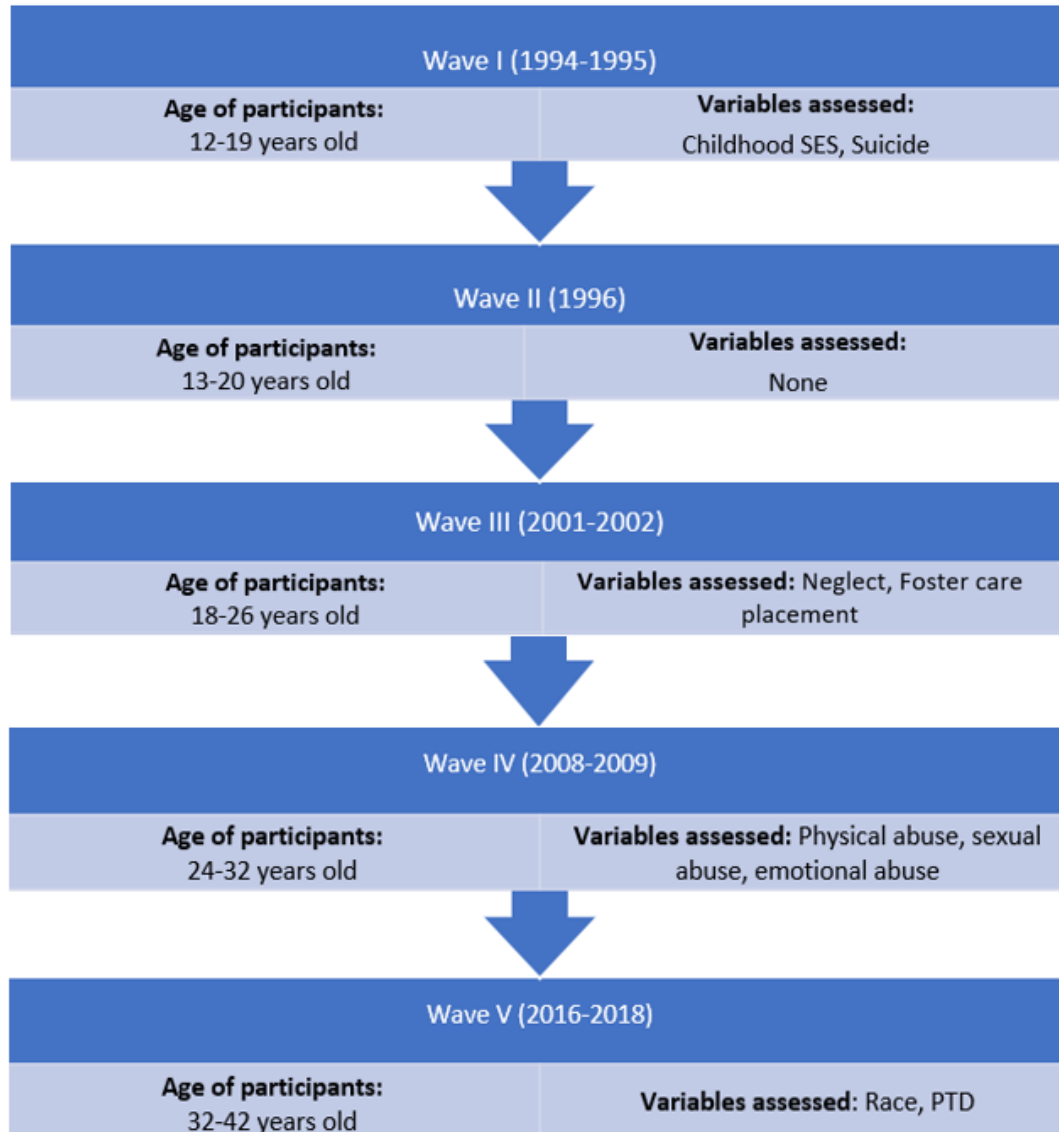


Figure 11. Timeline of Add Health data collection (1994-2018), respective ages of participants during each wave, and key Add Health variables assessed in this study.

4.2.3 Statistical Analyses

I weighted all descriptive and regression analyses to account for the stratified, multistage sampling design of the Add Health study. All analyses were conducted using SAS software statistical package 9.4 (Cary, NC).

Assessment of Missing Data. There were $n = 267$ total observations (comprising 6.6% of the reference population sample size of $n=4,034$) which were missing data on PTD, weighting variables, race, and ACEs. The assessment of bias (Appendix Table B3) indicated that exclusion of these observations did not significantly change the makeup of the analytic sample compared to the reference population with regard to exposure variables, outcome, and covariates. This provides evidence that the likelihood for selection bias is low, which promotes the internal validity of my findings. Therefore, these observations were removed from the reference population ($n=4,034$) to arrive at the final analytic sample of $n=3,767$ for this study.

Descriptive analyses. Survey-weighted frequency and regression procedures were conducted to calculate the means and frequencies for all variables of interest, respectively (Tables 6 and 7). I conducted a Wald chi-square test to determine statistically significant differences in the prevalence of PTD by race and SES using a significance level of $\alpha = 0.05$ (Table 7). I implemented survey-weighted ANOVA procedures with Tukey post-hoc pairwise comparison tests to evaluate statistically significant differences in mean ACE score across race and SES subgroups using a significance level of $\alpha = 0.05$ (Table 7).

Regression Analyses. I conducted unadjusted and adjusted survey-weighted logistic regression analyses to determine the association between six ACEs and the odds of PTD among women overall (Table 8), as well as in stratified analyses by race (Appendix Table B4) and SES (Appendix Table B5). Adjusted logistic regression analyses among women overall controlled for both race and SES, while adjusted logistic regression analyses stratified by race were adjusted for SES, and vice-versa for SES-stratified analyses. All six ACEs were included in each logistic regression model so that the ACEs controlled for the effects of one another. Odds ratios were deemed statistically significant if the null value of 1.0 was not included in the 95% confidence

interval (CI). If the upper or lower bound of the 95% CI included the value of 1.0, this was deemed as approaching statistical significance. Significant odds ratios from Appendix Table B4 were selected for display in Table 9. I identified white women and women with high SES to be the reference groups for race and SES, respectively, as they represent the most privileged demographic groups of women in the United States, based on access to resources such as education and financial assets. The logistic regression model specification for the association between six ACEs and PTD is depicted by Equation 1.6.

(1.6) $\text{logit}(Y_i) = \beta_0 + \beta_1(\text{sexual abuse}) + \beta_2(\text{physical abuse}) + \beta_3(\text{emotional abuse}) + \beta_4(\text{neglect}) + \beta_5(\text{suicide}) + \beta_6(\text{foster care placement}) + \beta_7(x_7) + \beta_8(x_8) + \beta_9(x_9) + \beta_{10}x_{10} + \beta_{11}x_{11} + \beta_{12}x_{12} + \beta_{13}x_{13} + \beta_{14}x_{14}$ where $Y_i = 1$ for PTD, and where 'White' is the reference group for race and 'High SES' is the reference group for SES such that:

$$x_7 = \begin{cases} 1 & \text{if Black} \\ 0 & \text{if not Black} \end{cases}; x_8 = \begin{cases} 1 & \text{if Hispanic} \\ 0 & \text{if not Hispanic} \end{cases}; x_9 = \begin{cases} 1 & \text{if Asian} \\ 0 & \text{if not Asian} \end{cases};$$

$$x_{10} = \begin{cases} 1 & \text{if Pacific Islander} \\ 0 & \text{if not Pacific Islander} \end{cases}; x_{11} = \begin{cases} 1 & \text{if AIAN} \\ 0 & \text{if not AIAN} \end{cases}; x_{12} = \begin{cases} 1 & \text{if Other} \\ 0 & \text{if not Other} \end{cases};$$

$$x_{13} = \begin{cases} 1 & \text{if low SES} \\ 0 & \text{if not low SES} \end{cases}; x_{14} = \begin{cases} 1 & \text{if middle SES} \\ 0 & \text{if not middle SES} \end{cases}$$

4.2.4 IRB Approval

The use of the Add Health Restricted Use Dataset to conduct this study was approved by the Carolina Population Center at the University of North Carolina at Chapel Hill. Furthermore, this research work was determined exempt from human subjects research under 45 CFR 46.104(d) 4(ii) by the Institutional Review Board (IRB) at Michigan State University (Appendix Figure A1).

4.3 Results

Descriptive analyses. Table 6 shows the descriptive characteristics of the $n = 3,767$ women who comprised my analytic sample. Nearly 70% self-identified as White while 14.4% identified as Black, 10.4% identified as Hispanic, 3.0% identified as Asian, 0.5% identified as Pacific Islander, 2.4% identified as American Indian or Alaska Native, and 0.8% self-reported as Other. Approximately 13% of women were from low SES backgrounds, 61.1% from middle SES backgrounds, and 26.2% from high SES backgrounds. Almost half of the women (48.2%) reported experiencing emotional abuse, 42.8% experienced neglect, 6.2% experienced sexual abuse, 6.0% reported a family member suicide attempt or death by suicide, and 2.7% reported ever being placed in foster care. The prevalence of PTD was 11.7%.

The mean ACE score among women overall in the sample was 1.3 (Table 7). There was a statistically significant difference in the mean ACE score by race ($p < .001$), although this was driven by only one pairwise difference. Specifically, a Tukey post-hoc comparison test (results not shown) found that women who identified as “Other” had a significantly lower mean ACE score compared to Asian women ($p < .01$, respectively). There were no significant differences in the mean ACE score between any other racial subgroups of women (all $p > .05$, respectively). Furthermore, there were no statistically significant differences in mean ACE score by SES ($p = 0.6$), nor were there any statistically significant differences in PTD prevalence by race ($p = 0.2$) or SES ($p = 0.6$) (Table 7). While there were not any statistically significant differences in PTD prevalence by race, the higher prevalences observed among Black women compared to White women are consistent with the PTD prevalences observed for these groups in national vital statistics reports from birth record data, although the observed prevalences for Asian,

American Indian or Alaska Native, Pacific Islander, and Hispanic women are higher compared to national prevalences.²²

Table 6. Descriptive characteristics of the analytic sample in the National Longitudinal Study of Adolescent to Adult Health, 1994-2018 (n=3,767)

	n (Weighted %)¹
Overall	3,767
Race	
White	2226 (68.5%)
Black	702 (14.4%)
Hispanic	502 (10.4%)
Asian	194 (3.0%)
Pacific Islander	29 (0.5%)
American Indian or Alaska Native	86 (2.4%)
Other	28 (0.8%)
Socioeconomic status (SES)	
Low	473 (12.7%)
Middle	2309 (61.1%)
High	985 (26.2%)
Marital status²	
Married/Cohabiting	2807 (76.0%)
Not Married/Cohabiting	646 (16.0%)
Maternal age at time of delivery (years)	
≤ 19	537 (14.3%)
20-24	1021 (28.3%)
25-29	996 (25.1%)
30-34	783 (20.8%)
≥ 35	240 (6.1%)
Preterm Delivery (PTD)	501 (11.7%)
Adverse childhood experiences (ACEs)	
Sexual abuse	212 (6.2%)
Physical abuse	721 (20.4%)
Emotional abuse	1750 (48.2%)
Neglect	1585 (42.8%)
Family member suicide attempt or death	209 (6.0%)
Foster care placement	87 (2.7%)

¹All analyses were weighted to account for the stratified sampling design of the Add Health study

²Data on marital status missing for n = 314 observations

Table 7. Descriptive characteristics of mean ACE score and PTD by race and SES in the National Longitudinal Study of Adolescent to Adult Health, 1994-2018 (n=3,767)¹

Race	All women	White (n=2226)	Black (n=702)	Hispanic (n=502)	Asian (n=194)	Pacific Islander (n=29)	AI/AN (n=86)	Other (n=28)	
	Mean (95% CI)								p-value ⁴
Mean ACE Score ²	1.3 (1.2-1.3)	1.3 (1.2-1.3)	1.2 (1.1-1.3)	1.3 (1.2-1.4)	1.4 (1.1-1.7)	1.3 (0.6-1.9)	1.2 (0.7-1.8)	0.8 (0.3-1.4)	<.001
	n (weighted %)								p-value ⁵
PTD ³ Yes	501 (11.7)	266 (10.5)	116 (14.0)	63 (13.5)	31 (17.1)	3 (18.3)	17 (15.3)	5 (22.5)	0.3
SES	Low (n=473)	Middle (n=2309)	High (n=985)						
	Mean (95% CI)			p-value ⁴					
Mean ACE Score ²	1.3 (1.1-1.4)	1.3 (1.2-1.3)	1.3 (1.2-1.4)	0.6					
	n (weighted %)			p-value ⁵					
PTD ³ Yes	71 (13.7)	299 (11.6)	131 (11.1)	0.6					

SE standard error; **AI/AN** American Indian or Alaska Native; **bolded** *p*-values indicate statistical significance at *p* < 0.01.

¹Data are weighted to account for the stratified sampling design of the Add Health study

²Mean number of ACEs based on sum of 6 binary ACE variables (sexual, physical, and emotional abuse; neglect; suicide; foster care placement)

³refers to proportion of women who responded “yes” to the question, “Was this baby born preterm?”

⁴*p*-value for survey weighted ANOVA

⁵*p*-value for survey weighted Wald chi-square test

Regression Analyses. The unadjusted and adjusted odds ratios for the association between six ACEs and PTD were not statistically significant among women overall (Table 8). The majority of the stratified analyses by race and SES were also not significant (Appendix Tables B4 and B5). However, a few associations between ACEs and PTD differed across race subgroups, particularly for White women and Black women, and these estimates were selected for display in Table 9. Specifically, the odds of PTD were 30% lower among White women who experienced physical abuse (aOR 0.7, 95% CI 0.5,1.0), although this approached significance. Also, the odds of PTD were 70% lower among Black women who experienced sexual abuse (aOR 0.3, 95% CI 0.1, 0.9) (Table 9). Due to small cell sizes, I was unable to obtain precise estimates for the odds ratios for the association between ACEs and PTD for select race and SES subgroups of women (Appendix Tables B4 and B5).

Table 8. Unadjusted and adjusted associations between six ACEs and odds of PTD among n=3,767 women in the National Longitudinal Study of Adolescent to Adult Health, 1994-2018¹

	OR (95% CI)	aOR (95% CI) ²
Sexual abuse	1.1 (0.6, 2.0)	1.2 (0.7, 2.0)
Physical abuse	0.8 (0.6, 1.1)	0.8 (0.6, 1.2)
Emotional abuse	1.2 (0.9, 1.6)	1.2 (0.9, 1.6)
Neglect	0.9 (0.7, 1.2)	0.9 (0.7, 1.2)
Suicide	1.0 (0.6, 1.7)	1.0 (0.6, 1.7)
Foster	1.3 (0.5, 3.2)	1.3 (0.5, 3.1)

¹Analyses are survey weighted to account for the sampling design of Add Health

²Odds ratios adjusted for race and SES

OR odds ratio; aOR adjusted odds ratio

Table 9. Unadjusted and adjusted associations between select ACEs and odds of PTD among White women and Black women in the National Longitudinal Study of Adolescent to Adult Health (n=3,767), 1994-2018¹

	White (n=2226)		Black (n=702)	
	OR (95% CI)	aOR (95% CI) ²	OR (95% CI)	aOR (95% CI) ²
Sexual abuse	NS	NS	0.3 (0.1, 1.0)	0.3 (0.1, 0.9)
Physical abuse	0.7 (0.5, 1.0)	0.7 (0.5, 1.0)	NS	NS

¹Analyses are survey-weighted to account for the sampling design of Add Health.

²Odds ratios are adjusted for SES

OR odds ratio; aOR adjusted odds ratio; NS Not significant

4.4 Discussion

To determine the impact of ACEs on PTD by race and socioeconomic status among a longitudinal sample of n=3,767 women from the National Longitudinal Study of Adolescent to Adult Health, this study **1)** Examined differences in the mean ACE score by race and socioeconomic status among women who give birth and **2)** Assessed the association between six ACEs and PTD among women overall, as well as within race and SES subgroups.

The results of this study can be summarized by five key findings. **First**, I found that the mean ACE score differed significantly by race ($p<.001$) but not by SES ($p=0.6$). **Second**, the prevalence of PTD did not differ significantly by race ($p=0.3$) or SES ($p=0.6$). **Third**, there were

no statistically significant associations between any of the six ACEs and odds of PTD among women overall (all p-values $>.05$, respectively). **Fourth**, due to small sizes for some cells, I was unable to obtain precise OR estimates for the association between ACEs and PTD for select subgroups of women. **Finally**, while the majority of ORs were not significant across all groups of women, there were a few associations between specific ACEs and PTD that differed across race subgroups, specifically for White women and Black women. These findings do not provide strong evidence that ACEs are associated with PTD among women in the overall sample or within race and SES subgroups, although PTD may operate differently within some subgroups and therefore it should not be assumed that all ACEs exert their effects equivocally on PTD in all groups of women. This carries important implications for the development of interventions to promote the preconception and pregnancy health of women.

While I found that the mean ACE score differed by race, this was primarily driven by the difference in scores between women who identified as “Other” and Asian women: specifically, Asian women had a significantly higher mean ACE score compared to women who identified as Other (1.4 vs. 0.8). This is interesting, as Asian women usually have lower ACE scores compared to other groups of women.²⁸ However, as we cannot disaggregate who comprises women who report as “Other,” this difference is difficult to interpret without additional context, although one assumption is that this group comprises multi-racial women who could not select a single racial group of their multi-race identity that they best identified with in the follow-up question.

The overall mean ACE score found among women in my sample (1.3, 95% CI 1.2-1.3) is significantly lower compared to the mean ACE score for women found by Giano et al. (2020) (1.64, 95% CI 1.62-1.67).²⁸ Furthermore, the mean ACE score found in my study for White

women (1.3, 95% CI 1.2-1.3), Black women (1.2, 95% CI 1.1-1.3), Hispanic women (1.3, 95% CI 1.2-1.4) were significantly lower than that found by Giano et al (2020) for White individuals (1.53, 95% CI 1.51-1.54), Black individuals (1.66, 95% CI 1.60-1.71), Hispanic individuals (1.63, 95% CI 1.58-1.69).²⁸ While Giano et al. (2020) found significant differences in mean ACE score between Blacks and Whites, my findings did not show this. There are several reasons that may explain discrepancies in study findings. First, it is important to note that while my calculated mean ACE scores are among women only, the mean ACE scores calculated by Giano et al. (2020) for racial subgroups comprise of both men and women.²⁸ Furthermore, while my sample consisted of women of reproductive age, the sample of women in Giano et al. (2020) consisted primarily of an older demographic (ages 45 and older).²⁸

Furthermore, unlike my study which did not find statistically significant differences in mean ACE score by SES, Giano et al. (2020) found that individuals with lower educational attainment, household income, and who were unemployed generally had higher ACE scores compared to those with higher educational attainment, household incomes, and employed status.²⁸ These differences could be attributed to the fact that my analytic sample comprised mostly of middle and higher SES women, who generally experience lower ACEs. In addition to the demographic differences between our samples that could explain differences in findings, the sample size of Giano et al. (2020) was significantly larger than mine ($n = 211,376$), providing greater power to detect differences.

While the overall prevalence of PTD in my study (11.7%) compares to the national prevalence of 10.5%,²² my finding of no significant differences in PTD prevalence by race and SES was unexpected, given known racial and SES disparities in PTD prevalence.²⁻⁴ One explanation is that my analytic sample comprised mostly of higher SES women, who are at

overall lower risk of adverse birth outcomes. Furthermore, while my dataset is nationally representative, my sample consisted predominantly of White women and had disproportionately smaller sample sizes of non-White women, which could bias my estimates.

While my study identified a few significant association between specific ACEs and PTD within select racial and SES subgroups of women, the majority of the associations were non-significant. These results are surprising, given the findings of previous studies assessing the impact of ACEs on preterm birth. In a systematic review of nine international studies examining the association between total ACEs and preterm birth, Sulaiman et al. (2021) found that seven of these studies found significant positive associations between ACEs and PTB.¹⁸ However, only four of the nine studies were conducted in the United States.^{58,213–215} A summary of the findings of these U.S.-based studies are as follows.

While my overall findings corroborate with those of McDonnell & Valentino (2016) who did not find that infant gestational age was correlated with either household dysfunction or maltreatment,²¹⁵ differences in the study design, sample, and methods of ACE operationalization between my study and these four U.S.-based studies from the systematic review prevent an unequivocal comparison of findings between the studies. Foremost, all of these studies operationalized ACEs as a count or sum of total ACEs experienced. While this is a common method in the literature, it prevents a more detailed assessment of whether specific ACEs exert their impacts differently on PTD. As evidence shows that “not all ACEs are equal” in their impact on health outcomes,^{61,62} the methods should be adapted to account for this. Indeed, while not included in the systematic review by Sulaiman et al. (2021),¹⁸ Margerison-Zilko et al. (2016) found that sexual abuse experienced during childhood, but not adulthood, increased the odds of late PTD (OR 1.5, 95% CI 1.0, 2.2), and sexual abuse experienced during both childhood *and*

adulthood increased the odds of any PTD (OR 1.9, 95% CI 1.0, 3.5) and late PTD (OR 2.2, 95% CI 1.1, 4.5).¹⁹ Furthermore, they did not find an association between physical abuse experienced during childhood or adulthood and the odds of PTD.¹⁹ While I found that physical abuse and emotional abuse were associated with increased odds of PTD, but not sexual abuse, among women overall, there were differences in the association between sexual abuse, physical abuse, and emotional abuse and PTD across race and SES subgroups.

Taken together, my findings and the findings of Margerison-Zilko et al. (2016) highlight the importance of examining the effects of specific ACEs on PTD as opposed to solely operationalizing these variables as a count or sum measure. Furthermore, these findings suggest that 1) specific ACEs may be more strongly associated with adverse birth outcomes than others and 2) There may be sensitive periods during which specific types of life stressors may exert their impacts on women's reproductive outcomes.

In addition to differences in the operationalization of ACEs in my study compared to the studies in the systematic review by Sulaiman et al. (2021), it is also important to note that all of these studies included pregnant women or postpartum women to assess the relationship between ACEs and PTB. This deviates from the design of my study, which assessed ACEs during the preconception period of women in my sample. As the women in these other studies were asked to report their ACEs during, or right after, pregnancy, this could result in recall bias, especially if they are experiencing a stressful pregnancy, pregnancy complications, or had given birth to an infant with an adverse birth outcome (i.e., PTD). In my study, however, information on ACEs were not collected in the same Wave as pregnancy information, which can lower the likelihood of this potential recall bias.

Finally, my study found the unexpected finding that certain ACEs, particularly sexual abuse for Black women and physical abuse for White women, were associated with a *lower* odds of PTD, which, to my knowledge, does not corroborate with any findings from the literature. It is possible that this counterintuitive finding is due to a potential selection bias, whereby Black and White women with lower overall levels of ACEs are more likely to have live birth pregnancies and therefore were disproportionately captured in my analytic sample, compared to women with high levels of ACEs who may not go on to have live birth pregnancies and therefore were not captured in my study. Because selection into my study is influenced by having both the exposure and the outcome (i.e., live birth pregnancy), this could have resulted in a collider bias that distorts the relationship between ACEs and PTD.

Moreover, women with lower levels of ACEs (compared to those with higher levels of ACEs) could also have additional protective factors (i.e., socioeconomic resources, access to high quality health care, social support, etc.) that lower their overall odds of adverse birth outcomes. An alternative explanation could be that experience of these specific ACEs lead Black and White women to engage in the process of resilience^{43,72} through reliance on and fostering of protective factors (such as social support, religiosity, or spirituality) that may ultimately buffer against the impact of ACEs on PTD, although the protective factor would have to be extremely strong to exert such an effect. Indeed, research shows that factors such as religiosity and spirituality may be protective against the impacts of life stressors, particularly for Black women.^{135,148}

Another alternative explanation for the lack of overall significant associations found between ACEs and PTD among women in my study is that a process of “recalibration” may occur during adolescence that resets any physiological disturbances potentially triggered by early

life stressors.^{84,85} DePasquale, Donzella, and Gunnar (2019) have found evidence of this recalibration effect in studies among institutionalized orphans who were subsequently adopted into nurturing environments and displayed a stabilization of abnormal HPA axis activity that was observed earlier in adolescence.⁸⁴ Similarly, women in my sample who experienced ACEs earlier in childhood could have encountered a more supportive and nurturing environment during adolescence that reset any physiological changes influenced by an early stressful environment, ultimately lowering their risk for future adverse health outcomes. In addition, because the majority of women in my sample gave birth at a younger maternal age (20-29 years old), the period of time between when they experienced ACEs and when they gave birth might not have been long enough for any adverse effects from early life to manifest and influence these pregnancy outcomes.³²

Strengths & Limitations. This study has several strengths. To my knowledge, this is the first study to examine the association between ACEs and PTD using a large, nationally representative dataset of women of reproductive age through a health equity lens by assessing whether this association differed across race and socioeconomic status subgroups.

Furthermore, my study design was heavily guided by theoretical frameworks across from lifecourse epidemiology and developmental psychology, which justifies the importance of looking at early life adversity in the preconception period as a mechanism by which disparities in future reproductive health outcomes may be impacted. Research that does not incorporate these frameworks are limited in their methodological design in that they examine at a narrow window of time very close to pregnancy (i.e., prenatal period), and may miss important risk factors that occur earlier in life. Furthermore, I used a person-centered approach in examining whether the associations between specific ACEs and PTD differed within race and SES subgroups, which is

an important approach for identifying subgroups of women who may be at greater risk of PTD given exposure to specific ACEs.

Despite these strengths, this study included several limitations. While the Add Health dataset was nationally representative, my analytic sample was not, and full data was not available for all groups of women. As such, the small sample sizes for select racial subgroups of women prevented me from obtaining more precise estimates of the association between ACEs and PTD for these women. However, given that Add Health is a uniquely large, diverse nationally representative dataset with five waves of data follow-up that uniquely incorporates data on health outcomes from childhood through adulthood, this illustrates the lack of nationally representative, longitudinal data on groups of people identifying as Black, Hispanic, Asian, Pacific Islander, and American Indian or Alaska Native.

Furthermore, I was only able to assess six types of ACEs; therefore, the findings may not be generalizable to other ACEs. Additional ACEs should be incorporated in future studies. Finally, while my study incorporated a lifecourse study design, it does not account for macro-level factors at the socioecological level^{216,217} that may contribute to, and perpetuate, disparities in both ACEs and PTD, such as institutional racism, and unequal access to education, health care, and insurance coverage.

4.5 Conclusion

In this study, I examined the impacts of specific ACEs on PTD by race and socioeconomic status among a longitudinal sample of $n=3,767$ women from the National Longitudinal Study of Adolescent to Adult Health (1994-2018). The majority of the associations between the six ACEs and PTD were not significant across race, SES, or within the overall sample. These results suggest the following possible explanations: 1) ACEs may not impact PTD

in hypothesized ways. 2) The length of time between when ACEs occur and when pregnancy occurs may not be long enough for any physiological disturbances associated with early life stress to manifest; or, alternatively, 3) Any physiological disturbances triggered by early life stress may be reset during adolescence, buffering against future adverse outcomes. The findings of this study contribute to an enhanced understanding of the role that specific ACEs play in PTD occurrence with consideration to race and SES difference, which is important for informing clinical interventions to improve women's preconception health and birth outcomes.

4.6 Acknowledgements

This research uses data from Add Health, funded by grant P01 HD31921 (Harris) from the Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD), with cooperative funding from 23 other federal agencies and foundations. Add Health is currently directed by Robert A. Hummer and funded by the National Institute on Aging cooperative agreements U01 AG071448 (Hummer) and U01AG071450 (Aiello and Hummer) at the University of North Carolina at Chapel Hill. Add Health was designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris at the University of North Carolina at Chapel Hill.

CHAPTER 5: THE ASSOCIATION BETWEEN ADVERSE CHILDHOOD EXPERIENCES AND PRETERM DELIVERY: A LATENT CLASS APPROACH

5.1 Introduction

The United States has one of the highest prevalences of PTD, a birth occurring earlier than 37 weeks of gestation, amongst all westernized countries.²¹⁸ In addition, significant racial and socioeconomic disparities in PTD have existed for decades in the United States.^{1–4,24} Specifically, Black women and women with low socioeconomic status experience higher prevalence of PTD compared to other women.² While the exact cause of these disparities has yet to be identified, it is hypothesized that disproportionate exposures to significant life stressors for these groups of women may increase their risk for adverse birth outcomes.^{11,27}

Adverse childhood experiences (ACEs), or negative life events occurring prior to 18 years of age, is a category of significant life stressors with significant implications for preconception and pregnancy health. Indeed, while the ACE literature has predominantly focused on examining the impacts of ACEs on health outcomes such as chronic diseases and mental health,^{16,17} a growing literature of studies have associated ACEs with adverse reproductive and pregnancy outcomes, including depression and anxiety during pregnancy, unintended pregnancy, alcohol use during pregnancy, and adverse birth outcomes.^{18,20,38–40}

However, most studies in the literature which have examined the associations between ACEs and PTD have **1)** not used diverse, nationally representative datasets; **2)** primarily focused on the impact of number of ACEs experienced (i.e., count and sum scores) as the predictor; and **3)** not used a health equity lens to assess whether associations might differ among women by race and socioeconomic status. While these foundational studies have provided an enhanced understanding of the influence of ACEs on PTD, these methodological gaps preclude **1)** the generalizability of findings to the diverse population of pregnant women in the United States²⁰⁵

2) an understanding of whether co-occurring patterns of ACEs exert greater detrimental effects on PTD than others; and 3) an understanding of whether ACEs impact all groups of women equally.

To address these methodological gaps, the overall goal of this study is to apply latent class techniques to provide a more nuanced understanding of the association between patterns of ACEs and PTD in a longitudinal sample of $n = 3,884$ women from the National Longitudinal of Adolescent to Adult Health (Add Health; 1994-2018) using a health equity lens. Specifically, I will 1) identify subgroups of women characterized by early life patterns of ACE occurrence; 2) determine the association between latent class membership and PTD and 3) examine whether race and SES play an important role in latent class membership or the association between latent class membership and PTD.

5.2 Methods

5.2.1 Study design, dataset, and study population

I used the National Longitudinal Study of Adolescent to Adult Health (Add Health) dataset, a nationally representative, prospective longitudinal study of adolescents initiated in 1994 by the Carolina Population Center and the University of North Carolina at Chapel Hill with the objective of studying the diverse social, behavioral, and biological influences on adolescent health and well-being, and their development into adulthood.^{182,183} Add Health used a multistage, stratified cluster sampling design to recruit adolescents from middle and high schools across the country.¹⁸³ Add Health entails five waves of follow-up: Wave I (1994-1995), Wave II (1996), Wave III (2001-2002), Wave IV (2008-2009), and Wave V (2016-2018) and includes in-school, at-home, and parental components of survey administration.^{182,183}

The current study focuses on the source population of $n = 20,745$ students who were selected to participate in the at-home survey component and to be followed up in all subsequent

waves of follow-up (i.e., the ‘core sample’).¹⁸³ The survey questionnaires for the at-home component employed computer-assisted personal interview (CAPI) and Audi Computer-Assisted Self-Interview (ACASI) techniques to improve the validity of self-reported data by respondents, especially pertaining to sensitive personal information.^{183,186}

After removing male adolescents (n = 10,263), female adolescents who did not have a pregnancy that resulted in a live birth by 2018 (n=6446), female adolescents who did not report information on preterm birth for the first birth in Wave V (n=71), female adolescents who were missing information on any weighting variables (n=66), and female adolescents who were missing information on race (n=13), I obtained a sample size of n = 3,884 for my final analytic sample of women (Figure 12). To maximize sample size and retain available data on any ACEs where provided by respondents, full-information maximum likelihood was applied to account for women who were missing data on these variables (n=117).^{187,219}

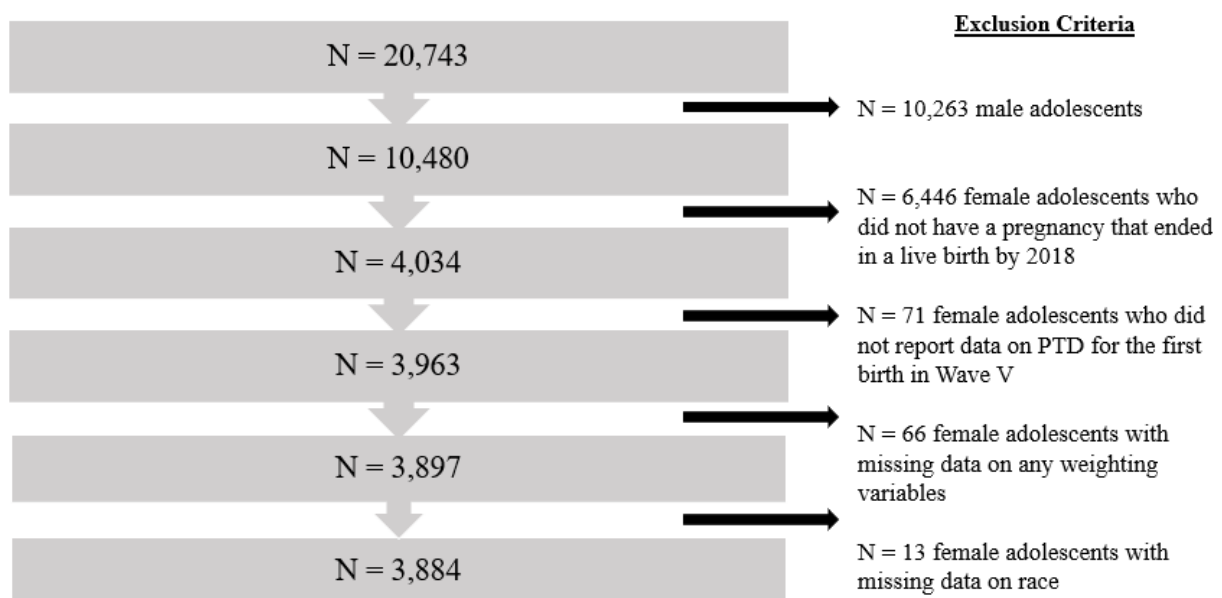


Figure 12. Analytic sample used to assess the association between patterns of ACEs and PTD in the National Longitudinal Study of Adolescent to Adult Health, 1994-2018.

5.2.2 Measures

Exposure variables. The primary exposure variables of interest were six adverse childhood experiences (ACEs) across three broad domains of ACEs including 1) abuse (sexual abuse, physical abuse, emotional abuse), 2) neglect, and 3) household dysfunction (family member attempted suicide or death by suicide, and foster care placement) (see Appendix Table C1). The selection of these ACEs was informed by the ‘conventional ACEs’ included in the original ACE study by the CDC and Kaiser Permanente,^{16,17} as well as the list of ‘expanded ACEs.’⁵⁵

I operationalized physical, sexual, and emotional abuse (each assessed by one question, respectively) and neglect (a composite of three questions) by creating a binary variable based on frequency of occurrence: “yes” for reported frequencies of ≥ 1 and “no” for reported frequencies of “0”. Suicide was captured by two questions pertaining to attempted suicide or death by suicide of family members and modeled as a binary variable (yes/no). While the original Add Health survey question used the phrase “successful suicide,” I used the language “death by suicide” in accordance with changing protocols in psychology to avoid stigmatizing language such as “successful suicide.”^{189,190}

Outcome variable.

I assessed preterm delivery (PTD) as my primary outcome of interest, defined as a delivery that occurs prior to 37 weeks of gestation. Information on PTD (for the first birth only) was collected via self-report and modeled as a binary variable (yes/no). Previous studies show that data on gestational age collected by maternal recall compares favorably to data obtained by medical records or birth certificates.^{191,192} Data on PTD (Wave V: 2016-2018) was collected at a

different time point than the ACEs (Waves I: 1994-1995, Wave III: 2001-2001, Wave IV: 2008-2009).

Covariates.

Race (White, Black, Hispanic, Asian, Pacific Islander, American Indian/Alaska Native, Other) was captured via self-report. If a respondent indicated they were multi-racial, a follow-up question was asked of respondents to indicate the racial group they best identified with. In this study, I refer to race as, "...a social classification based on phenotype that governs the distribution of risks and opportunities in our race-conscious society" (Jones, 2001, p. 300).¹⁹³ While race is a social construct and is not biologically or genetically based, it holds important implications for the health and well-being of individuals and communities.¹⁹³⁻¹⁹⁶ Furthermore, while I use six categories of race in this study, it is important to acknowledge that each of these subgroups are heterogeneous and comprise of diverse ethnicities and nationalities. Finally, to promote grammatical equity in reference to race I use intentional capitalization for all racial subgroups throughout this paper.¹⁹⁷

Socioeconomic status was assessed as childhood socioeconomic status and captured by a composite construct of six indicators (mother's occupation, father's occupation, mother's father's education, annual household income, and receipt of public assistance) based on the method developed by Slaughter-Acey et al., 2016 (Appendix Table C2).¹⁶⁰ Each of these variables were modeled as binary variables where 0 represented "low SES" and 1 represented "high SES."¹⁶⁰ Missing data on any of these variables were categorized as "low SES."¹⁶⁰ The scores for each of the six variables were then summed to create a composite SES variable with values ranging from 0 to 6.¹⁶⁰ Based on the distribution of scores of the composite SES variable, three categories of SES were created: low (where score < lowest quartile (Q1) where Q1 = 1),

middle (score \geq 2nd quartile (Q2) and \leq 3rd quartile (Q3) where Q2=2 and Q3=3), and high (score $>$ upper quartile (Q4) where Q4 = 3).¹⁶⁰

Figure 13 summarizes the Add Health waves of data collection, ages of participants at each wave of data collection, and timing of assessment of all key variables in this study.^{182,183}

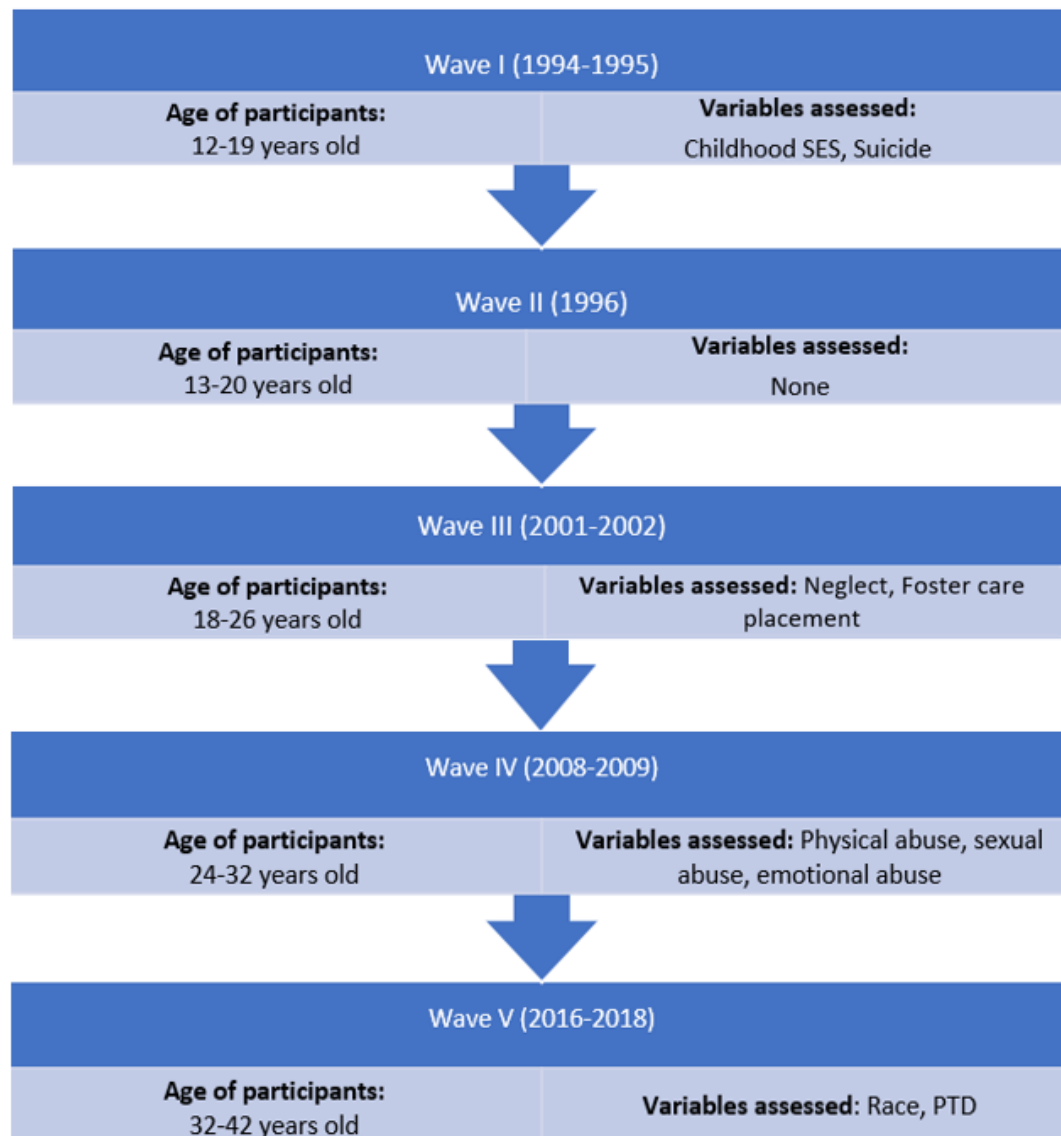


Figure 13. Timeline of Add Health data collection (1994-2018), respective ages of participants during each wave, and key Add Health variables assessed in this dissertation study.

5.2.3 Statistical Analyses

Assessment of Missing Data. Observations with missing data on PTSD, race, or weighting variables were excluded from the analytic sample ($n=150$; 3.7%). An assessment of bias indicated that the distribution of exposure variables, outcome variable, and covariates did not change significantly between the analytic sample and the reference population upon removal of these missing data, justifying the removal of these observations (Appendix Table C3). This

indicates that there is a low probability of selection bias and therefore strengthens the internal validity of my findings.

To maximize sample size and data provided on the six ACE indicators, I used full-information maximum likelihood (FIML) with the assumption of missing at random (MAR) to address missing data on these variables.^{187,219} Less than 5% (n=117) of the analytic sample was missing data on ACEs.

Descriptive analyses. The distribution of maternal characteristics, PTD, and adverse childhood experiences for the analytic sample was derived using survey-weighted frequency procedures. Percentages were weighted to account for the stratified, multistage sampling design of the Add Health Study. These analyses were conducted using SAS software statistical package 9.4 (Cary, NC).

Latent class analyses (LCA). I conducted all latent class analyses using Mplus Version 8.6 statistical software package (Muthén & Muthén, 1998-2017).²⁰⁸ Six ACEs, known as class indicators, were used to inform the latent classes (See Figure 14 for the conceptual path diagram). To initiate the process of class enumeration (i.e., identification of the number of latent classes), I fitted 2-class, 3-class, and 4-class models by conducting survey-weighted finite mixture analyses with robust maximum likelihood estimation (MLR) to account for the stratified, multistage sampling design of the Add Health study.

The optimal latent class model with the best fit was determined by assessment of the following fit statistics: log-likelihood, Akaike Information Criterion (AIC), Bayesian Information Criterion (BIC), Consistent Akaike Information Criterion (CAIC), Sample Size Adjusted BIC (ssBIC), Integrated Completed Likelihood Criterion with BIC approximation (ICL-BIC), and entropy. Smaller values indicated better model fit for all criteria with the

exception of entropy, where values closer to 1.0 indicate better classification separation between the classes (range of values from 0 to 1).^{187,209}

After identification of the optimal 2-class model, I assessed the two key parameters of the model: 1) the class prevalences, or the proportion of the sample captured by each class and 2) the estimated conditional within-class probabilities (i.e., item-response probabilities), which is the probability that a woman reported a specific response to an ACE (e.g., yes or no), given that they were in a specific latent class, for all six ACEs.¹⁸⁷ The class with the highest prevalence was designated as the reference class for all analytic comparisons.

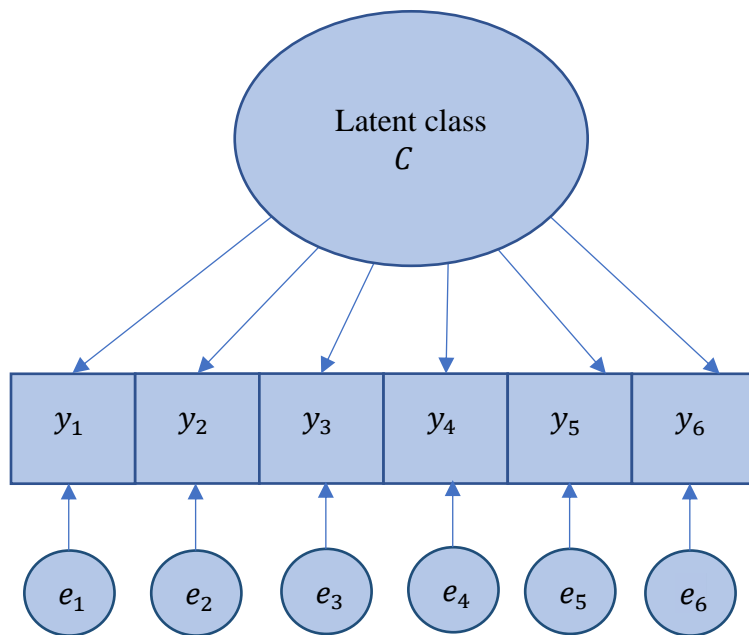


Figure 14. Path diagram of the six ACE indicators used to inform the latent class C in the National Longitudinal Study of Adolescent to Adult Health (n=3,884), 1994-2018 where y1 (physical abuse); y2 (sexual abuse); y3 (emotional abuse); y4 (neglect); y5 (household substance abuse risk); y6 (attempted suicide or death by family member(s)); y7 (foster care placement). The measurement error associated with each indicator is denoted by e_1 - e_6 .

Class Prediction Analyses. To assess whether race or SES predicted latent class membership, I implemented Vermunt's three-step approach.²¹¹ Following the Vermunt's process outlined by Bauer & Steinley (2020, p. 8.8-8.12),²¹⁰ in Step 1, the posterior probabilities were calculated for each case, which represents the probability that an individual is in a specific class, given their response for each ACE indicator.^{187,209,210} Step 2 entails the process of modal class assignment, whereby individuals are assigned to a latent class based on the highest value of the posterior probability.^{210,211} Step 2 also calculates the level of accuracy with which individuals are classified in the modal class assignment process, an indication of classification error.²¹⁰ In Step 3, the modal class assignment serves as a proxy variable for latent class membership and the logit values are incorporated to account for the level of classification error.²¹⁰ This final step provides us with the regression coefficients for the regression of latent class on the predictors of race and SES, as well as the odds ratios for latent class membership by race and SES. Figure 15 illustrates the path diagram for the class predictor analyses. Equation 1.8 depicts the logistic regression model specification for the prediction of latent class membership by race and SES, where race and SES control for one another.

Because both race and SES have three or more categories, dummy indicator variables were created to facilitate the interpretation with the reference group. As reflected in Equation 1.8, White women and high SES women were the reference groups for race and SES, respectively. I selected these groups to be the reference as they represent the most privileged groups of women in U.S. society based on access to resources such as education, income, and wealth.

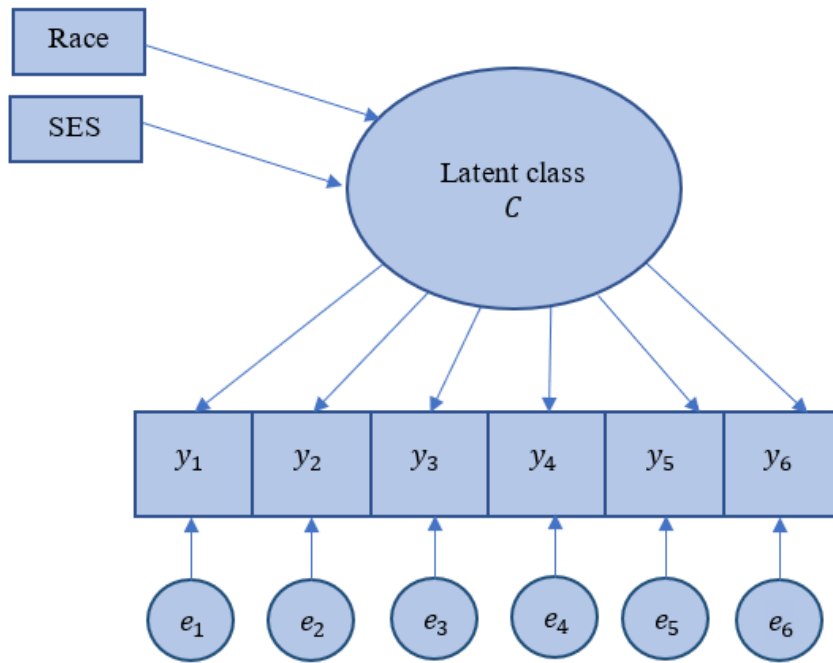


Figure 15. Path diagram for the prediction of latent class membership by two covariates, race and socioeconomic status (SES), in the National Longitudinal Study of Adolescent to Adult Health (n=3,884), 1994-2018 where y1 (physical abuse); y2 (sexual abuse); y3 (emotional abuse); y4 (neglect); y5 (attempted suicide or death by family members); y6 (foster care placement); z1 (PTD); e1-e6 (measurement error for each indicator)

(1.8) $\text{logit}(Y_i) = \beta_0 + \beta_1(x_1) + \beta_2(x_2) + \beta_3(x_3) + \beta_4(x_4) + \beta_5(x_5) + \beta_6(x_6) + \beta_7(x_7) + \beta_8(x_8)$ where $Y_i = 1$ for membership in class i and where 'White' is the reference group for race and 'High SES' is the reference group for SES such that:

$$x_1 = \begin{cases} 1 & \text{if Black} \\ 0 & \text{if not Black} \end{cases}; x_2 = \begin{cases} 1 & \text{if Hispanic} \\ 0 & \text{if not Hispanic} \end{cases}; x_3 = \begin{cases} 1 & \text{if Asian} \\ 0 & \text{if not Asian} \end{cases};$$

$$x_4 = \begin{cases} 1 & \text{if Pacific Islander} \\ 0 & \text{if not Pacific Islander} \end{cases}; x_5 = \begin{cases} 1 & \text{if AIAN} \\ 0 & \text{if not AIAN} \end{cases}; x_6 = \begin{cases} 1 & \text{if Other} \\ 0 & \text{if not Other} \end{cases};$$

$$x_7 = \begin{cases} 1 & \text{if low SES} \\ 0 & \text{if not low SES} \end{cases}; x_8 = \begin{cases} 1 & \text{if middle SES} \\ 0 & \text{if not middle SES} \end{cases}$$

Distal outcome analysis. To evaluate whether latent class membership predicted the distal outcome of PTD (See Figure 16 for the path diagram for the model), I conducted Vermunt's 3-Step approach using the same first two steps as described previously in class predictor analyses.^{210,211} However, in the third step, a model parameter constraint was added to assess whether the threshold of PTD differs significantly across the classes via a Wald Test of equality. Because PTD is a binary variable, it exhibits a threshold, as opposed to a mean. The threshold is an estimated parameter in the form of a logit derived from the third step of Vermunt's that is used to calculate the within-class probability of having the outcome (i.e., PTD).²¹⁰ Equation 1.9 shows how to calculate the probability of reporting a "yes" response to an ACE based on the threshold parameter estimate (Bauer and Steinley, 2020, p. 7.4).²¹⁰ Equation 1.10 depicts the model specification for the logistic regression analysis of PTD on latent class membership, controlling for race (reference group is White women) and SES (reference group is high SES women).

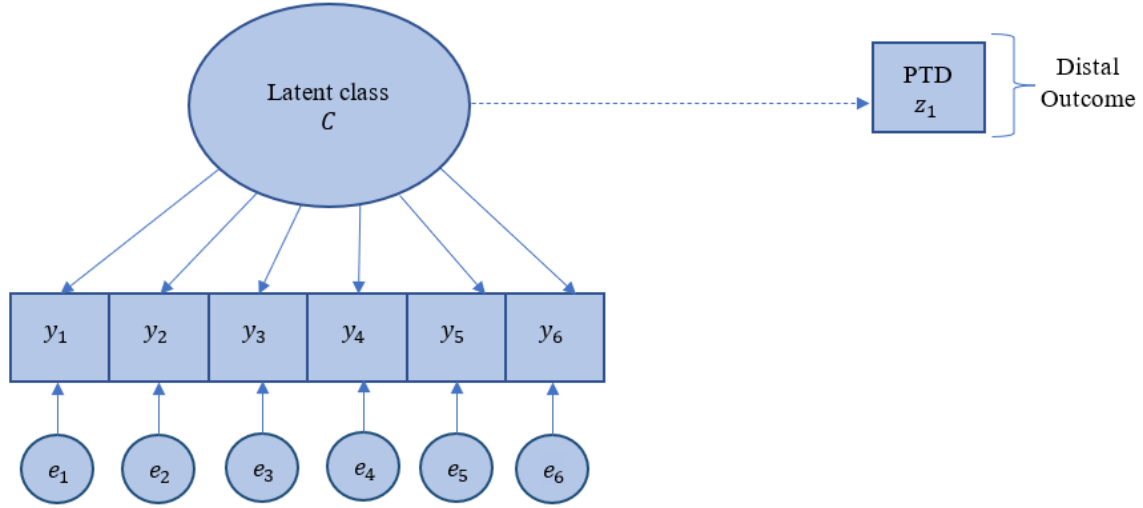


Figure 16. Path diagram of the latent class distal outcome analysis of the association between latent class membership and PTD in the National Longitudinal Study of Adolescent to Adult Health (n=3,884), 1994-2018 where y1 (physical abuse); y2 (sexual abuse); y3 (emotional abuse); y4 (neglect); y5 (attempted suicide or death by family members); y6 (foster care placement); z1 (PTD); e1-e6 (measurement error for each indicator)

$$(1.9) \delta_{jk} = \frac{1}{1 + e^{-v_{jk}}} \text{ where } \delta_{jk} \text{ is the probability of reporting "yes" for an individual in class } k,$$

and v_{jk} is the threshold parameter estimate (Bauer and Steinley, 2020, p. 7.4)²¹⁰

$$(1.10) \text{logit}(Y_i) = \beta_0 + \beta_1(x_{1,i}) + \beta_2(x_2) + \beta_3(x_3) + \beta_4(x_4) + \beta_5(x_5) + \beta_6(x_6) + \beta_7(x_7) +$$

$\beta_8(x_8) + \beta_9(x_9)$ where $Y_i = 1$ for PTB, $x_1 = \text{membership in class } i$, and where 'White' is the reference group for race and 'High SES' is the reference group for SES such that:

$$x_2 = \begin{cases} 1 & \text{if Black} \\ 0 & \text{if not Black} \end{cases}; x_3 = \begin{cases} 1 & \text{if Hispanic} \\ 0 & \text{if not Hispanic} \end{cases}; x_4 = \begin{cases} 1 & \text{if Asian} \\ 0 & \text{if not Asian} \end{cases};$$

$$x_5 = \begin{cases} 1 & \text{if Pacific Islander} \\ 0 & \text{if not Pacific Islander} \end{cases}; x_6 = \begin{cases} 1 & \text{if AIAN} \\ 0 & \text{if not AIAN} \end{cases}; x_7 = \begin{cases} 1 & \text{if Other} \\ 0 & \text{if not Other} \end{cases};$$

$$x_8 = \begin{cases} 1 & \text{if low SES} \\ 0 & \text{if not low SES} \end{cases}; x_9 = \begin{cases} 1 & \text{if middle SES} \\ 0 & \text{if not middle SES} \end{cases}$$

5.2.4 IRB Approval

The use of the Add Health Restricted Use Dataset to conduct this study was approved by the Carolina Population Center at the University of North Carolina at Chapel Hill. Furthermore, this research work was determined exempt from human subjects research under 45 CFR 46.104(d) 4(ii) by the Institutional Review Board (IRB) at Michigan State University (Appendix Figure A1).

5.3 Results

Descriptive analyses. Table 10 outlines the prevalence of maternal characteristics, PTD, and ACEs in the analytic sample (n=3,884). The majority of women were White (68.7%), from middle SES backgrounds (61.1%), and married or cohabiting (75.7%). The ACE with the highest prevalence was emotional abuse (47.7%), followed by neglect (42.3), physical abuse (20.2%), sexual abuse (6.3%), family member suicide attempt or death (6.1%), and foster care placement (2.7%). The prevalence of PTD was roughly 12%.

Latent class analyses. Table 11 provides a summary of the fit statistics for the 2-class, 3-class, and 4-class model. The 2-class model was determined to be the optimal class model with the best fit based on the smaller values for the majority of the fit indices (BIC, CAIC, ssBIC, ICL-BIC) compared to the 3-class and 4-class model.

Table 12 shows the estimated class prevalences and conditional within-class probabilities of six ACEs for the two latent class model. The class prevalences indicated that latent class 1 comprised 75.1% of the sample, while latent class 2 comprised 24.9% of the sample (Table 12). Class 2 was distinguished by significantly higher probabilities of reporting sexual abuse (.21), emotional abuse (1.0), physical abuse (.70), and foster care placement (.05) compared to Class 1 ($p < .05$, respectively). There were no significant differences in the conditional within-class

probabilities for neglect or suicide. For this reason, Class 2 was labeled the “High ACEs” class while Class 1 was labeled the “Low ACEs” class. Figure 17 illustrates the probability of reporting “yes” to the six ACEs, conditional on latent class membership.

Table 10. Descriptive characteristics of the analytic sample in the National Longitudinal Study of Adolescent to Adult Health, 1994-2018 (n=3,884)

	n (Weighted %)¹
Overall	3,884
Race²	
White	2297 (68.7%)
Black	723 (14.4%)
Hispanic	517 (10.3%)
Asian	197 (2.9%)
Pacific Islander	32 (0.5%)
American Indian or Alaska Native	89 (2.4%)
Other	29 (0.8%)
Socioeconomic status (SES)	
Low	487 (12.5%)
Middle	2382 (61.1%)
High	1015 (26.4%)
Marital status³	
Married/Cohabiting	2893 (75.7%)
Not Married/Cohabiting	667 (16.1%)
Maternal age at time of delivery (years)	
≤ 19	562 (14.5%)
20-24	1048 (28.2%)
25-29	1021 (24.9%)
30-34	807 (20.7%)
≥ 35	248 (6.2%)
Preterm Delivery (PTD)	514 (11.8%)
Adverse childhood experiences (ACEs)⁴	
Sexual abuse	217 (6.3%)
Physical abuse	738 (20.2%)
Emotional abuse	1793 (47.7%)
Neglect	1614 (42.3%)
Family member suicide attempt or death	216 (6.1%)
Foster care placement	90 (2.7%)

¹percentages are survey-weighted to account for stratified sampling design of Add Health

²n=324 women with missing data on marital status

³n=198 women with missing data on maternal age

⁴n=117 women with missing data on any ACEs; ACEs are not mutually exclusive categories

Table 11. Summary of fit statistics for the latent class analysis of six adverse childhood experiences in the National Longitudinal Study of Adolescent to Adult Health (n=3,884), 1994-2018

Class model	Log-likelihood	AIC	BIC	CAIC	ssBIC	ICL-BIC	Entropy
2	-9003.452	18032.90	18114.34	18127.34	18073.04	18115.78	0.72
3	-8989.821	18019.64	18144.93	18164.93	18081.38	18146.58	0.82
4	-8978.884	18011.77	18180.91	18207.91	18095.12	18182.17	0.63

Bolded values indicate the optimal value for the fit criteria; *AIC* Akaike Information Criterion; *BIC* Bayesian Information Criterion; *CAIC* Consistent Akaike Information Criterion; *ssBIC* Sample Size Adjusted BIC; *ICL-BIC* Integrated Completed Likelihood Criterion with BIC approximation

Table 12. The estimated latent class prevalences and conditional within-class probabilities¹ of six ACEs for the two latent class model identified among n=3,884 women in the National Longitudinal Study of Adolescent to Adult Health (1994-2018).

	Class 1 (Low ACEs Class)	Class 2 (High ACEs Class)	Statistically significant difference at $p < .05$
Latent class prevalences	75.1%	24.9%	
ACEs			
Sex abuse	.02	.21	*
Emotional abuse	.32	1.0	*
Physical abuse	.04	.70	*
Neglect	.43	.43	
Suicide	.06	.07	
Foster care placement	.02	.05	*

¹Probability of reporting “yes” to each of the six ACEs

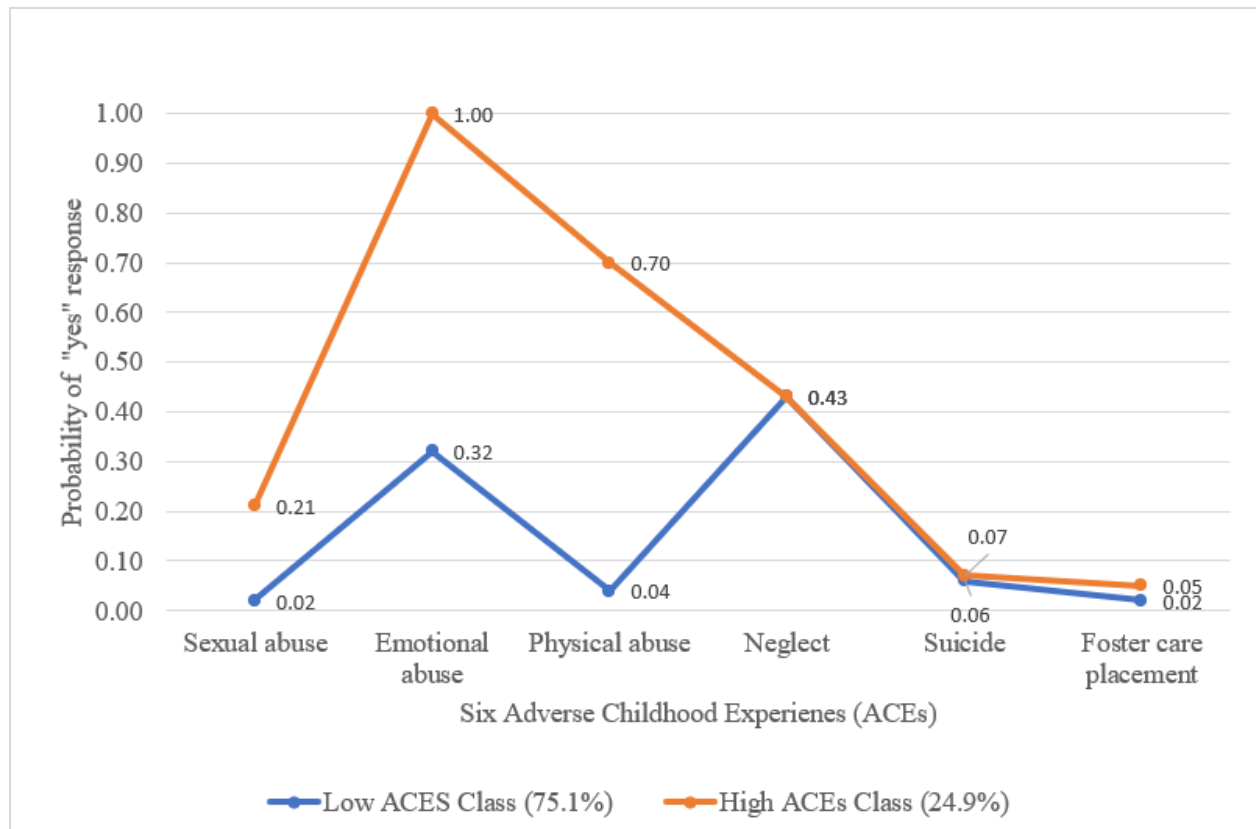


Figure 17. Probability of reporting “yes” to six adverse childhood experiences (ACEs) conditional on latent class membership among n=3,884 women in the National Longitudinal Study of Adolescent to Adult Health (1994-2018).

Class Prediction Analyses. The logits for the classification probabilities, which accounts for the level of misclassification error,²¹⁰ for the 2-class model derived from Vermunt’s 3-step approach for the class predictor analyses are depicted in Table 13. Using Equation 1.9 to calculate the probability based on the logit values, these findings convey that the probability of being assigned to the low ACEs class, given that the case is actually in low ACEs class, is high (.98), and the probability of being assigned to the low ACEs class, given that the case is actually in the high ACEs class, is low (.25), reflecting relatively good reliability for the modal class assignment process and good class separation.

Table 14 presents the findings for the class prediction analyses derived from the final step of Vermunt’s 3-Step approach. These results indicate that the odds of being in the high ACEs class compared to the low ACEs class does not significantly differ for Black women, Hispanic women, Asian women, Pacific Islander women, American Indian or Alaska Native women, and Other women, compared to White women (all 95% CIs encompass the null value of 1.0 for each group, respectively). Furthermore, the odds of being in the high ACEs class compared the low ACEs class does not differ significantly for low SES and middle SES women, compared to high SES women (both 95% CIs encompass the null value of 1.0, respectively).

Table 13. Logits for the classification probabilities for the two-class model derived from Vermunt’s 3-step approach

	Modal Class Assignment 1	Modal Class Assignment 2
Latent Class 1	-4.008	0.000
Latent Class 2	1.121	0.000

Table 14. Estimated odds ratios for the prediction of latent class membership by race and socioeconomic status subgroups among n=3,884 women in the National Longitudinal Study of Adolescent to Adult Health, 1994-2018

Class predictor	aOR ¹ (95% CI)
Race (ref=White)	
Black	0.9 (0.6, 1.3)
Hispanic	1.0 (0.6, 1.7)
Asian	1.7 (0.7, 3.7)
Pacific Islander	1.0 (0.2, 4.3)
AIAN	0.5 (0.1, 1.7)
Other	0.6 (0.1, 4.8)
SES (ref= High SES)	
Low SES	1.0 (0.7, 1.5)
Middle SES	0.9 (0.7, 1.3)

¹Odds of class membership for Class 2 (High ACEs Class) compared to the reference of Class 1 (Low ACEs Class); ORs for race subgroups are adjusted for SES and ORs for SES subgroups are adjusted for race

Distal Outcomes Analysis. Table 15 shows that the threshold estimates for PTD do not differ significantly across the low ACEs class and the high ACEs class for either the unadjusted or adjusted models, as demonstrated by the Wald Test of parameter constraints ($p=1.00$; $p=0.99$, respectively). These findings suggest that latent class membership does not predict PTD. Furthermore, Table 16 shows that the threshold estimates for PTD did not differ significantly across the high ACEs or low ACEs class in the race-stratified models (p -values all $>.05$, respectively) except for women who identified as Other ($p<.001$).

Among these women, the probability of PTD in the low ACEs class was significantly higher than the probability of PTD in the high ACEs class (0.25 vs. .03, respectively). In the SES-stratified models, the threshold estimates for PTD differed significantly between the low ACEs class and the high ACEs class for low SES women only ($p<.04$), whereby low SES women in the low ACEs class experienced a higher probability of PTD compared to low SES women in the high ACEs class (0.17 vs. .05, respectively; Table 16). The threshold estimates for PTD did not differ significantly across the latent classes for middle SES or high SES women ($p>.05$, Table 16).

Table 15. Threshold estimates for PTD by latent class in the distal outcomes analysis among $n=3,884$ women in the National Longitudinal Study of Adolescent to Adult Health, 1994-2018

	Class 1 Low ACEs Class		Class 2 High ACEs Class		Wald Test of Parameter Constraints ²
	p(PTB)	Threshold	p(PTB)	Threshold	
Model 1 (Unadjusted)	0.12	2.005	0.12	2.005	$X^2=0$, $df=1$ p -value = 1.00
Model 2 (Adjusted) ¹	0.097	2.227	0.098	2.225	$X^2=0$, $df=1$ p -value = 0.99

¹Adjusted for race and SES

Table 16. Race and SES-stratified distal outcomes analyses for PTD by latent class among
n=3,884 women in the National Longitudinal Study of Adolescent to Adult Health, 1994-2018¹

	Class 1 Low ACEs Class		Class 2 High ACEs Class		Wald Test of Parameter Constraints ²
	p(PTB)	Threshold	p(PTB)	Threshold	
Race subgroups					
White (n=2297)	0.10	2.085	0.12	1.906	X ² =0.59, df=1 p-value = 0.44
Black (n=723)	0.15	2.657	0.16	2.496	X ² =12, df=1 p-value = 0.73
Hispanic (n=517)	0.16	2.180	0.07	3.117	X ² =1.36, df=1 p-value = 0.24
Asian (n=197)	0.16	1.454	0.18	1.161	X ² =0.11, df=1 p-value = 0.74
Pacific Islander (n=32)	0.22	25.938	0.04	30.314	X ² =1.9, df=1 p-value = 0.16
AI/AN (n=89)	0.16	1.945	0.08	3.145	X ² =0.45, df=1 p-value = 0.50
Other ³ (n=29)	0.25	1.078	0.03	12.586	X ² =322.6, df=1 p-value < .001
SES subgroups					
Low SES (n=487)	0.17	1.848	0.05	3.092	X ² =4.1, df=1 p-value =.04
Middle SES (n=2382)	0.12	2.216	0.12	2.135	X ² =1.1, df=1 p-value = 0.75
High SES (n=1015)	0.10	2.159	0.14	1.735	X ² =1.3, df=1 p-value=0.26

¹Race-stratified analyses are adjusted for SES; SES-stratified analyses are adjusted for race

²Wald Test for statistically significant differences in Threshold estimates for PTD by latent class

³Refers to women who self-reported as “Other” in the Add Health Survey

Bolded values indicate statistical significance at p<.05

AI/AN American Indian or Alaska Native

5.4 Discussion

To determine the association between patterns of ACEs and PTD in a longitudinal sample of $n = 3,884$ women from the National Longitudinal of Adolescent to Adult Health (Add Health), this study **1)** identified latent classes of women characterized by early life patterns of ACEs **2)** evaluated the association between latent class membership and PTD **3)** examined whether race and SES play an important role in latent class membership or the association between latent class membership and PTD.

The results of this study demonstrate the following key findings. **First**, two underlying latent classes of women were identified, one characterized by low ACE prevalence and the other one by high ACE prevalence. Specifically, the high ACEs class was distinguished by a higher conditional probability of emotional abuse, physical abuse, sexual abuse, and foster care placement compared to the low ACEs class. **Second**, I found that race and SES did not predict latent class membership. **Third**, I found that latent class membership did not predict PTD. **Fourth**, I found that the relationship between class membership and PTD differed significantly only among women who identified as Other, but not among any other racial subgroups. **Finally**, the relationship between latent class membership and PTD differed significantly for low SES women, but not for middle or high SES women.

The finding that emotional abuse, physical abuse, sexual abuse, and foster care placement patterned together in the high ACEs class is not surprising, given that different forms of maltreatment and abuse commonly co-occur,^{16,32} often a reflection of an unstable caretaking environment.³² Furthermore, it makes sense that children who experience abuse are also likely to be placed in foster care.

In a sample of n=4336 women from the Pregnancy Risk Assessment Monitoring System (PRAMS), Deichen Hansen (2021) uncovered three types of latent classes (high risk, moderate risk, and low risk) based on maternal characteristics and risk of PTB and LBW.²⁰⁵ Furthermore, using data on n=111,330 women from PRAMS (2011-2015), Koning & Ehrental (2019) identified three clusters of women, one characterized by high levels of stressful maternal life events, another characterized mostly by illness-related maternal life events, and the third characterized by overall low levels of stressful maternal life events.⁶⁰ While these studies identified different numbers of subgroups of women compared to my study, the identification of groups of women with higher and lower levels of stressful life events are consistent themes.

My findings that race and SES did not predict latent class membership are unexpected, particularly in light of research that ACE prevalence varies significantly by both race and socioeconomic status, with Black, Hispanic, and American Indian or Alaska Native individuals and lower SES individuals experiencing a disproportionate burden of ACEs.²⁸⁻³⁰ Furthermore, my findings are discrepant with both Deichen Hansen (2021) and Koning & Ehrental (2019), as they found racial and socioeconomic differences in subgroups of women in their study.^{60,205}

The finding that latent class membership in the high ACEs class did not predict PTD was also unexpected, as previous research has linked these types of ACEs with PTD.¹⁸ Furthermore, my finding that membership in the high ACEs class did not predict PTD among race subgroups, except for women who identified as Other, was also surprising, as disparities in both ACEs and PTD differ significantly by race.^{2,28} Although this relationship differed among women who identified as Other, this was in the opposite direction of what I expected, as the probability of PTD was higher in the low ACEs class compared to the high ACEs class. While I found that latent class membership and PTD differed for low SES women, this was also in the opposite

direction than I expected, as low SES women in the low ACEs class experienced a higher probability of PTD compared to low SES women in the high ACEs class. This finding is also unexpected, as lower SES individuals are at greater risk of both PTD and ACEs.^{3,28} My findings are also discrepant with Koning & Ehrenthal (2019), who found that the overall relationship between stressor landscapes and probability of preterm birth varied by race and income quartile, although no statistical assessments of differences was provided.⁶⁰

There are several factors that may explain the discrepancies in findings between my study and those found in the literature. While my study specifically assessed ACEs, Koning & Ehrenthal (2019) and Deichen Hansen (2021) examined different kinds of exposures (i.e., stressful maternal life events in the year prior to childbirth and maternal demographic characteristics and health indicators during pregnancy) assessed among postpartum women in PRAMS.^{60,205} This could have resulted in recall bias, as women who experience pregnancy complications may be more likely to overreport stressful maternal life events or adverse health indicators during pregnancy. Furthermore, there were important differences in dataset (e.g., PRAMS datasets were not nationally representative), analytic sample size, and study population (i.e., both studies used fewer categories of race which may limit generalizability).^{60,205}

While I used a nationally representative dataset from Add Health, my analytic sample was not nationally representative, and the majority of my sample was comprised of White women and women from higher SES backgrounds. This may have biased the findings as these groups of women are at lower risk for both ACEs and PTD, potentially underestimating the relationship between latent class membership and PTD. Furthermore, women who experience high levels of ACEs may be more likely to 1) decide not to have children;¹⁰⁶ 2) have difficulties becoming pregnant due to physical trauma (e.g., physical and/or sexual abuse) or other health

conditions (e.g., depression, anxiety); 3) experience a pregnancy loss;⁵⁸ or 4) have an abortion as a result of an unintended pregnancy,^{39,111} and therefore may not be captured in my denominator of women who have pregnancies that end in a live birth, resulting in a potential selection bias that can underestimate the association between ACEs and PTD.

In addition, I modeled the ACEs as binary variables (yes/no) and therefore I did not account for other characteristics of ACEs such as frequency of occurrence (i.e., severity of ACEs), which may have a stronger impact on occurrence of adverse pregnancy outcomes. As the experience of a greater number of ACE types, particularly 4 or more types, can substantially increase the risk of detrimental health outcomes,^{17,32} the frequency of ACE occurrence may show a similar adverse effect on health outcomes. While frequency of occurrence was not available for all ACEs in my study, as a next step, I plan to incorporate frequency of occurrence where available for specific ACEs. In addition, each ACE was assessed by a different number of questions. Some ACEs, such as physical, emotional, and sexual abuse, were asked by only one question, which may not comprehensively capture the construct and result in residual confounding.

Furthermore, it is possible that the window of time between when ACEs occur and when pregnancy occurs is not long enough for the physiological disruptions potentially caused by early life stress to manifest on health outcomes.³² As most women in my study had a young maternal age (20-29 years), this is plausible. Alternatively, if women experience stressors early in childhood, there may be a potential for a resetting or “recalibration” of any stress-induced physiological disruptions during adolescence, especially if protective factors are encountered that make up for the early risk factors.^{33,84,85}

Furthermore, there may be a process of resilience that plays a role for women who experience life stressors- that is, women who experience stressors may not go on to have adverse health outcomes in pregnancy due to some protective factor, such as at the individual-level (i.e., personality characteristics, religiosity), interpersonal-level (i.e., social support), and community-level (i.e., neighborhood reciprocity), that buffers against the risks of stressors like ACEs on PTB. Indeed, studies have shown that factors such as social support, self-esteem, mastery, social cohesion, and reciprocal exchange are protective against the effects of life stressors on the risk of PTD.^{76-78,138} Furthermore, Zamani-Hank et al. (2022) found that religiosity was associated with a lower odds of PTD among Black women and low SES women.⁸¹ As a next step, I plan to assess the role of religiosity and spirituality as potential modifiers of the relationship between ACEs and PTD. Finally, it is possible that ACEs do not impact PTD in hypothesized ways. While the stressor hypotheses have been proposed as an explanatory mechanism for PTD disparities, the evidence is not clear.^{5,92,100,102,104}

Strengths & Limitations. This study exhibits several strengths that address the gaps of previous studies examining the relationship between ACEs and PTD. First, I used a large, longitudinal dataset with data spanning over 20 years, which provided a plethora of data on psychosocial variables such as adverse childhood experiences and potential protective factors. Secondly, my research questions were informed by interdisciplinary theoretical frameworks, particularly across developmental psychology and lifecourse epidemiology, which contribute a paradigm shift in thinking about the importance of women's health not just during the prenatal period, but during preconception as well.^{11,14,37} Identification of women during preconception counseling and prenatal screening assessments who may be at higher risk for adverse pregnancy outcomes (or other conditions, such as metabolic disorders, diabetes, depression, anxiety, etc.,

which may increase their risk of adverse pregnancy and birth outcomes) due to early life risk factors is important for purposes of clinical intervention (i.e., connection to counseling and therapy interventions).

In addition, I specifically assessed whether race and SES play an important role in class membership or the relationship between class membership and PTD, which is important for assessing whether specific groups of women are at greater risk of experiencing PTD as a result of early adverse life experiences. Furthermore, I used latent class modeling as a person-based methodological approach to determine whether co-occurring patterns of ACEs are important predictors for PTD, instead of the common methodological approach of using a sum or count of ACEs, which fails to account for potential interaction of risks among women.

Despite these strengths, this study entailed several limitations. While I took advantage of a large, nationally representative dataset, my final analytic sample was not nationally representative. Therefore, the diversity of my analytic sample was limited, as it was comprised mostly of White and higher SES women, which limits generalizability to specific racial and SES subgroups of women. However, given that Add Health is a uniquely large, diverse nationally representative dataset with five waves of data follow-up that incorporates data on health outcomes from childhood through adulthood, this illustrates the lack of data on groups of people identifying as Black, Hispanic, Asian, Pacific Islander, and American Indian and Alaska Native who have pregnancies in prospective studies that can be used to understand the impacts of early life experiences on pregnancy outcomes.

Furthermore, I was only able to assess six types of ACEs, which may limit generalizability of findings to other types of ACEs (i.e., parental death or incarceration). Finally, ACEs were operationalized as binary variables based on presence or absence of occurrence,

which does not account for severity or frequency of occurrence. Future studies should incorporate these characteristics into ACE measurement.

5.5 Conclusion

In this study, I applied latent class techniques to evaluate whether underlying subgroups of ACEs were associated with PTD in longitudinal sample of $n = 3,884$ women from the National Longitudinal of Adolescent to Adult Health (Add Health) using a health equity lens. Overall, I found that latent class membership did not predict PTD, and that race and SES, for the most part, did not play a significant role in latent class membership or the relationship between latent class membership and PTD. While taking into consideration the limitations of this study which limit generalizability of findings, these results suggest the following possibilities: 1) the window of time between early life stressors and pregnancy may not be long enough for adverse effects to manifest; 2) there may be a process of resilience due to specific protective factors, or possibly a recalibration process, at play that reduces the risk of PTD among women who experience ACEs during early life; or 3) ACEs may not exert their impacts in hypothesized ways on the risk of PTD. As a next step, future studies can assess whether protective factors play a role in the association between ACEs and PTD as one potential explanation.

5.6 Acknowledgements

This research uses data from Add Health, funded by grant P01 HD31921 (Harris) from the Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD), with cooperative funding from 23 other federal agencies and foundations. Add Health is currently directed by Robert A. Hummer and funded by the National Institute on Aging cooperative agreements U01 AG071448 (Hummer) and U01AG071450 (Aiello and Hummer) at the University of North Carolina at Chapel Hill. Add Health was designed by J. Richard Udry,

Peter S. Bearman, and Kathleen Mullan Harris at the University of North Carolina at Chapel Hill.

CHAPTER 6: DO RELIGIOSITY AND SPIRITUALITY MODIFY THE ASSOCIATION BETWEEN ADVERSE CHILDHOOD EXPERIENCES AND PRETERM DELIVERY? A LATENT CLASS MODERATION ANALYSIS

6.1 Introduction

Preterm delivery (PTD), occurring when an infant is delivered prior to 37 weeks of gestation, is one of the leading causes of infant mortality in the United States and of significant concern for child and maternal well-being.⁵ Moreover, significant racial and socioeconomic disparities in PTD have existed for over 30 years.^{2-4,24} Specifically, Black women exhibit a disproportionately higher burden of PTD compared to White women, while women from lower socioeconomic status (SES) backgrounds are at higher risk compared to women from higher SES backgrounds.^{2,3} While decades of foundational research have identified important risk factors for PTD,⁵ the exact cause of these disparities are still unclear.

A disproportionate exposure to life stressors has been proposed as one hypothesized mechanism by which disparities in PTD may be influenced among women.^{11,27} Specifically, adverse childhood experiences (ACEs), defined as adverse life events occurring in the first 18 years of life, are a form of life stressor with important potential repercussions for pregnancy and reproductive health. Indeed, ACEs have been associated with pregnancy loss, unintended pregnancy, depression during pregnancy, and adverse birth outcomes.^{14,18,20,39,58,111} This research suggests that while ACEs occur during an early period of development, their effects may resonate throughout the lifecourse. Furthermore, as ACEs are more highly prevalent among Black, Hispanic, and American Indian or Alaska Native children, and children from lower SES backgrounds, they can contribute to, and exacerbate, disparities in PTD.²⁹⁻³¹

While complete prevention of ACEs altogether is the optimal solution for both child and maternal health and well-being, prevention is not always feasible. Therefore, there is a critical

research need to identify factors that may be protective against the risks of early life adversity on adverse birth outcomes for purposes of clinical intervention.

Specifically, religiosity and spirituality (hereafter, R/S) are two types of potential protective factors that have been studied in the context of pregnancy and reproductive health, particularly on mental health during pregnancy, postpartum health, and health behaviors during pregnancy.^{166,167,220–223} Moreover, R/S have been shown to be sources of strength for parents in the face of traumatic pregnancy and birthing experiences such as pregnancy loss, still birth, and infants requiring NICU care.^{178–180,224}

However, very few studies have examined the impacts of religiosity and spirituality in the context of birth outcomes- specifically, I found only one published study which examined PTD of June 2022. In a study of n = 91 low-income Mexican-immigrant women from a prenatal clinic in Texas, Page et al. (2021) found that frequency of prayer and level of religiosity significantly *increased* the odds of PTD, contrary to their hypothesis.¹⁷¹ While these findings provide some insights into the association between religiosity and PTD, the sample size was small and not racially diverse, precluding an ability to assess whether the effects of R/S on PTD might differ across factors such as race and SES.

Addressing this gap in the literature, my previous research, using a sample of n=2474 pregnant women from the POUCH study in Michigan, found that while religiosity was not associated with PTD among women overall, it was associated with an approximately 40% reduction in the odds of PTD for both low SES women (OR 0.6, 95% CI 0.4-0.9) and Black women (OR 0.6, 95% CI 0.4-1.0).⁸¹ These findings suggest that protective factors such as religiosity may operate differently by race and SES to impact PTD.

Indeed, studies show that religiosity is a particularly important coping mechanism in the Black community, especially in the face of life stressors including “health issues, caregiving burdens, chronic poverty, poor neighborhood conditions, structural exclusion, and interpersonal and structural racism” (Chatters et al., 2008, p. 373).¹³⁴ Religiosity has also been discussed as an important coping mechanism among low-income populations, who may rely on religiosity and spirituality to cope with chronic life stressors including unpredictable and unstable financial situations.¹³⁷

Building upon this concept that religiosity and spirituality can be important resources in the face of life adversity, religiosity and spirituality have also been studied as factors that may buffer against the risk of adverse childhood experiences to promote positive health outcomes in adulthood. Indeed, in a sample of n=4041 adults aged 25-74 from the Midlife in the United States study, Homan & Hollenberger (2021) found that religious support during adulthood moderated the effects of adverse childhood experiences on physical health (i.e., self-rated health, functional limitations, and shortness of breath) in middle and late adulthood, although religious identification, private religious practice, or religious service attendance did not.²²⁵ Furthermore, in a sample of n=241 African-American adolescents from Texas among whom over 50% experienced four or more ACEs, Freeny et al. (2021) spirituality was associated with a lower odds of depression (OR=0.927, 95% CI 0.892, 0.998, p=.04).²²⁶

While these studies did not assess birth outcomes specifically, taken together, these studies provide a foundation for investigating whether religiosity and spirituality might similarly protect against the risk ACEs on adverse birth outcomes such as PTD. While studies demonstrate that religiosity and spirituality are relied on by parents as sources of strength in the face of traumatic pregnancy and birthing experiences (i.e., pregnancy loss, stillbirth, NICU),^{178–180} a

PubMed search of “religiosity and preterm birth and adverse childhood experiences” as well as “spirituality and preterm birth and adverse childhood experiences” yielded zero results as of June 2022, underscoring that research on the relationship between ACEs, preterm birth, and R/S is a major gap in the literature.

Furthermore, many studies in the literature examine the impacts of either religiosity *or* spirituality on health outcomes and often do not differentiate between the two concepts.^{172,173} While religiosity and spirituality are related concepts (and often examined as a single construct, “R/S”, in the literature), they have important distinctions.^{174,175} In this study, I will assess spirituality and religiosity as separate constructs, but also an aggregate construct (R/S) that combines aspects of both concepts.

Finally, as Zamani-Hank et al. (2022) previously demonstrated that religiosity may operate differently by race and SES,⁸¹ it is important to understand whether the degree to which religiosity and spirituality may influence PTD differs within racial and SES subgroups, as the assumption should not be made that all protective factors operate in the same way across all groups of women. In other words, conducting this type of research through a person-centered, healthy equity lens is important. Furthermore, as the CIs for the OR findings among Black women and low SES women in Zamani-Hank et al. (2022) were close to the null, this provides justification for conducting this research with a larger sample size.

Previously, in Chapter 5, I uncovered two latent classes among n=3,884 women in the National Longitudinal Study of Adolescent to Adult Health- one characterized by low levels of ACEs and the other characterized by high levels of ACEs. The current study aims to build upon this research and address the aforementioned gaps in the literature by examining the role that two potential protective factors, religiosity and spirituality, play in the association between ACEs and

PTD, and whether these factors operate differently by race and SES. Specifically, this study aims to **1)** Assess whether the mean levels of religiosity, spirituality, and a composite construct of religiosity and spirituality (R/S), differ across the high ACEs class and the low ACEs class; **2)** Evaluate the role that religiosity, spirituality, and R/S play in the association between latent class membership and PTD and **3)** Examine whether these potential protective factors operate differently within race and SES subgroups.

6.2 Methods

6.2.1 Study design, dataset, and study population

I conducted this study using restricted-use data from the National Longitudinal Study of Adolescent to Adult Health (Add Health), a nationally representative, prospective longitudinal study of adolescents initiated in 1994 by the Carolina Population Center and the University of North Carolina at Chapel Hill.^{182,183} The purpose of this study was to assess the diverse physiological, environmental, and psychosocial factors which influence adolescents' health outcomes, well-being, and their development in adulthood.¹⁸² Add Health entailed a stratified, multi-stage clustered sampling approach to select adolescents based on a national database of middle schools and high schools across the United States.¹⁸² The Add Health study incorporates in-school, at-home, and parental survey components and encompasses five waves of data follow-up spanning 24 years: Wave I (1994-1995), Wave II (1996), Wave III (2001-2002), Wave IV (2008-2009), and Wave V (2016-2018).^{182,183}

This study focuses on the sample of $n = 20,745$ students who were selected to participate in the at-home survey administration and to be followed up in the subsequent five waves of follow-up. To promote the validity of self-reported data in the surveys, Add Health adopted the

use of Audio Computer-Assisted Self-Interview (ACASI) and computer-assisted personal interview (CAPI) techniques.^{183,186}

My final analytic sample included n=3,884 female adolescents after exclusion of male adolescents (n=10,263), female adolescents who did not report a pregnancy that resulted in a live birth (n=6446) by 2018, female adolescents who did not report data on preterm birth for the first birth in Wave V (n=71), female adolescents who were missing information on any weighting variables (n=54), and female adolescents with missing data on race (n=13) (Figure 18).

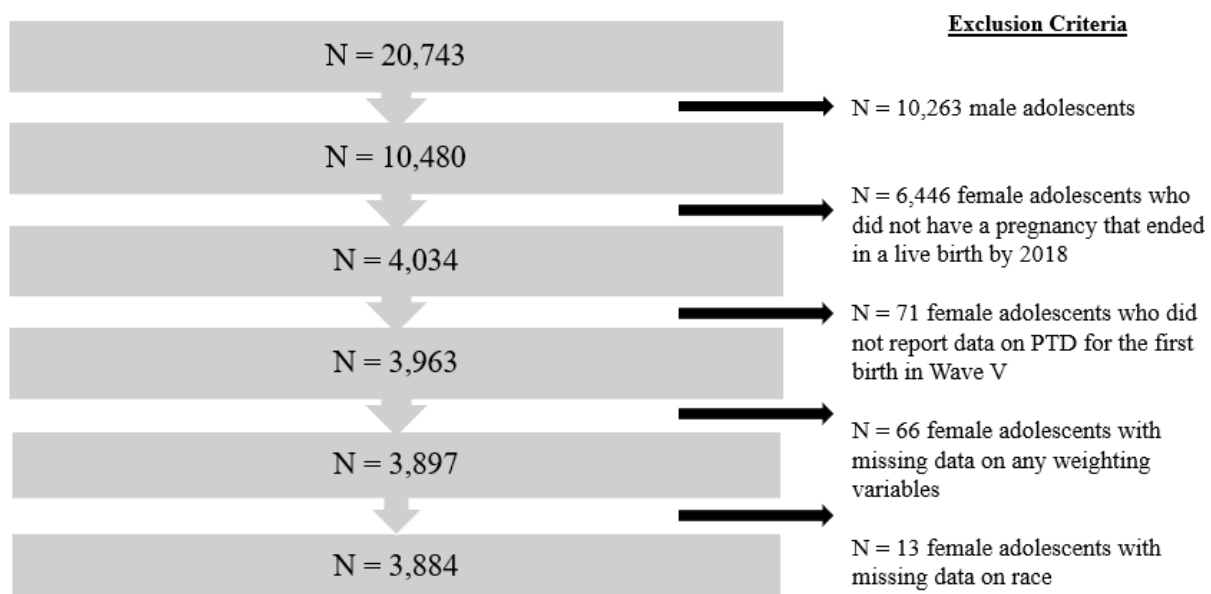


Figure 18. Analytic sample for the latent class moderation analysis among n=3,884 women in the National Longitudinal Study of Adolescent to Adult Health, 1994-2018.

6.2.2 Measures

Exposure variables. My exposure variables included six adverse childhood experiences (ACEs) across three broad ACE categories: abuse (sexual, physical, emotional), neglect, and household dysfunction (family member suicide attempt or death and foster care placement). (Appendix Table D1). The selection of these ACEs was guided by both the conventional

categories of ACEs examined in the original study on ACEs^{16,17} as well as the expanded categories of ACEs identified in recent research.⁵⁵

Sexual abuse, physical abuse, and emotional abuse (one question each) and neglect (a composite of three questions) were all modeled as binary variables (yes/no) based on the reported frequency of occurrence. If occurrence was ≥ 1 , it was categorized as “yes” and “no” if occurrence was 0. Foster care placement (one question) was modeled as a binary variable based on reported occurrence (yes/no). Suicide (two questions) was ascertained by the reported occurrence of either a family member suicide attempt or death by suicide and similarly modeled as a binary variable (yes/no). While the original Add Health survey question states the phrase “successful suicide,” I use the phrase “death by suicide” in accordance with changing protocols to avoid stigmatizing language such as “successful suicide.”^{189,190}

Outcome variable. My outcome of interest was preterm delivery (PTD), which was modeled as binary (yes/no) based on the self-reported occurrence of a birth occurring earlier than 37 weeks of gestation. Research demonstrates that the accuracy of gestational age data collected via maternal recall compares favorably to data obtained from medical records or birth certificates.^{191,192} Finally, information on PTD was collected in a different wave (Wave V: 2016-2018) than the ACEs (Waves I:1994-1995; Wave III: 2001-2002; Wave IV: 2008-2009).

Covariates. *Religiosity and Spirituality.* Religiosity (one question) was modeled as a continuous variables based on a Likert scale of responses ranging from 0 (not religious at all) to 3 (very religious). Spirituality (one question) was modeled continuously, with responses ranging from 0 (not spiritual at all) to 3 (very spiritual). An aggregate religiosity and spirituality variable (R/S), modeled as continuous, was also developed based on the sum of the scores across three

questions pertaining to spiritual and religious beliefs. The values for this variable ranged from 3-15, where a higher score indicated higher R/S.

While the Add Health survey questionnaire did not define spirituality or religiosity, I define religiosity as, “the degree which an individual believes, follows, and practices a religion” (Damiano et al., 2019, p. 5).¹⁷⁴ I define religion as “a personal set or institutionalized system of religious attitudes, beliefs, and practices, and is the service or worship of God or the supernatural.”¹⁷² Spirituality is generally a broader concept and encompasses the process of finding meaning and a sense of peace in life.^{172,174,176} It has been formally defined as, “...a dynamic and intrinsic aspect of humanity through which persons seek ultimate meaning, purpose, and transcendence, and experience relationship to self, family, others, community, society, nature, and the significant or sacred. Spirituality is expressed through beliefs, values, traditions, and practices (Puchalski et al., 2014, p. 646)”¹⁷⁷ Because of these distinctions in definition, it is important to avoid the assumption that religiosity and spirituality necessarily exert similar effects on health outcomes. Therefore, in this body of work, I examine religiosity and spirituality separately, while also examining an aggregate R/S variable comprising questions pertinent to both constructs

Religiosity, spirituality, and R/S variables were assessed during Wave III (2001-2002), when the women were young adults (ages 18-26). Thus, these variables were assessed after the period during which the occurrence of ACEs had already ended. See Appendix Table D2 for details on the assessment of religiosity and spirituality variables.

Race (White, Black, Hispanic, Asian, Pacific Islander, American Indian or Alaska Native (AIAN), Other) was collected by self-report. If a respondent self-reported as multi-racial, a follow-up question was asked to inquire about the racial category with which they best identified

with. In this study, I refer to race as the categorization of people into groups based on physically identifying characteristics such as skin color that occurs in racially stratified societies.^{193–195}

While race is a social construct and is not grounded in biology or genetics, it can significantly influence the health and well-being of people and communities.^{193–196} In addition, while I use six categories of race in this study, it is important to recognize that there is tremendous within-group heterogeneity and diversity of ethnicities and nationalities. Finally, throughout this paper, I intentionally capitalize the names for all racial subgroups to promote language that enhances grammatical racial equity.¹⁹⁷

Socioeconomic status (low SES, middle SES, high SES) was assessed as childhood socioeconomic status and captured as a construct of six socioeconomic indicators, including maternal occupation, paternal occupation, maternal education, paternal education, receipt of public assistance, and annual household income (Appendix Table D3).¹⁶⁰ Informed by the method used by Slaughter-Acey et al. (2016),¹⁶⁰ each of the six variables were dichotomized into binary categories of 0 (“low SES”) and 1 (“high SES”) (See Appendix Table D3 for operationalization details). If data was missing for any of these six variables, this was classified as low SES.¹⁶⁰ The scores for each of the six variables were then tallied to create a sum score that ranged from 0 to 6.¹⁶⁰ Based on the distribution of these scores, a composite SES variable with three categories was created whereby scores less than the lower quartile (Q1 = 1) were classified as “low SES”; scores \geq 2nd quartile (Q2=2) and \leq 3rd quartile (Q3 = 3) were classified as “middle SES,” and scores greater than the upper quartile (Q4 = 3) were classified as “high SES.”¹⁶⁰ Figure 19 provides a summary of the Add Health waves of data collection, respective ages of participants at each wave, and timing of assessment of all key variables in this study.^{182,183}

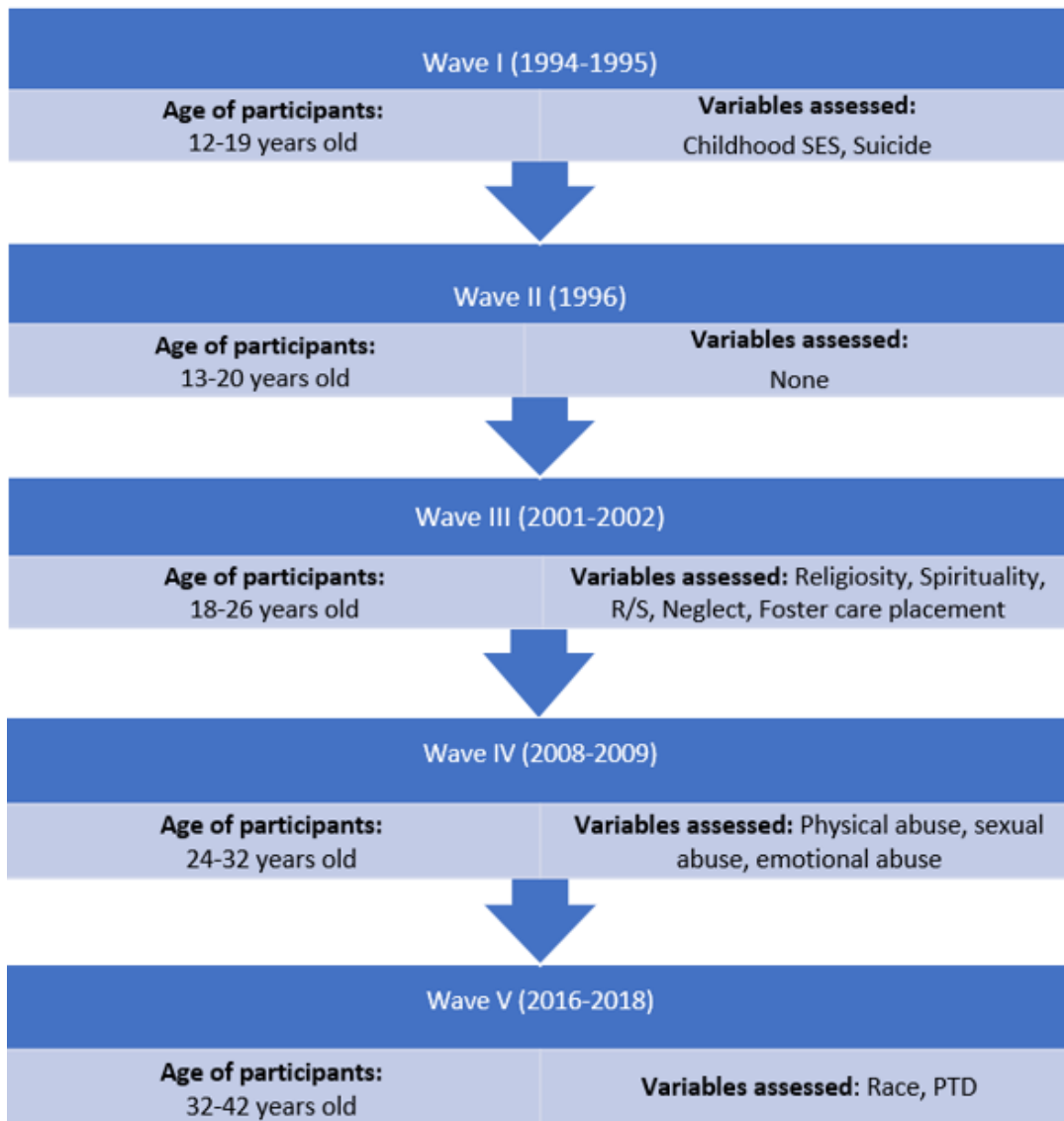


Figure 19. Timeline of Add Health data collection (1994-2018), respective ages of participants during each wave, and key Add Health variables assessed in this study.

6.2.3 Statistical Analyses

Assessment of missing data. Observations with data missing for PTSD, race, or weighting variables were excluded from the analytic sample ($n=150$; 3.7%). An assessment of bias demonstrated that removal of these observations did not significantly change the distribution of exposure variables, outcome variable, and covariates between the analytic sample ($n=3,884$) and

the reference population (n=4,034) (Appendix Table D4). Because these results indicated that there is a low likelihood for selection bias (which promotes the internal validity of the findings), this provided justification for the removal of these observations (Appendix Table D4). In addition, I used Full-information maximum likelihood (FIML) with the assumption of missing at random (MAR) to address missing data on any of the six ACEs that were used to inform the latent classes. The purpose of this technique is to retrieve any available data on indicators reported by respondents^{187,219} to maximize sample size and reduce possible selection bias. Less than 5% (n=117) of the sample was missing data on ACEs.

Descriptive analyses. I implemented survey-weighted frequency procedures to obtain the distribution for maternal characteristics, PTD, religiosity and spirituality variables, and ACEs for the analytic sample (Table 17). Survey weights were applied to account for the stratified, multistage sampling design of the Add Health study. I conducted these analyses using SAS Statistical Software Package 9.4 (Cary, NC).

The means of religiosity, spirituality, and R/S were calculated for each latent class, respectively, using survey-weighted latent class analyses in Mplus Version 8.6 (Muthén & Muthén, 1998-2017)²⁰⁸ (Table 18). A Wald Test was implemented to evaluate any statistically significant differences in the means between the two latent classes (Table 18).

Latent class moderation analysis with distal outcome. Previously (see Chapter 5), I implemented survey-weighted latent class analyses with robust maximum likelihood estimation (MLR) to derive the optimal fitting latent class model based on assessment of the following fit statistics: log-likelihood, Akaike Information Criterion (AIC), Bayesian Information Criterion (BIC), Consistent Akaike Information Criterion (CAIC), Sample Size Adjusted BIC (ssBIC), Integrated Completed Likelihood Criterion with BIC approximation (ICL-BIC), and entropy.

After identification of the optimal 2-class model comprising of the low ACEs class and the high ACEs class, I conducted Vermunt's 3-step approach to evaluate whether latent class membership predicted the distal outcome of PTSD.²¹¹ In this paper, I build upon my previous distal outcome analyses by assessing whether three potential protective factors (religiosity, spirituality, and R/S) modify the association between latent class membership and PTSD (See Figure 20 for the conceptual path diagram). This association was assessed in unadjusted models, adjusted models (for race and SES), as well as in models stratified by race and SES subgroups. Equation 1.11 depicts the adjusted overall logistic regression model for the latent class moderation analysis.

Guided by the process outlined by Bauer & Steinley (2020, p. 8.8-8.12; 8.27-8.33),²¹⁰ I conducted a survey-weighted latent class moderation analysis using Vermunt's 3-step approach as follows. In Step 1, the posterior probabilities were calculated for the model, which represents the probability that an individual belongs to a specific latent class, given their patterns of responses on the six ACE indicators.^{210,211} In Step 2, the process of modal class assignment is conducted, whereby cases are assigned to the latent class for which the individual has the highest likelihood of belonging to (i.e., largest posterior probability).^{210,211} This second step also calculates estimates of classification accuracy (i.e., level of error) based on the logits for the classification probabilities for the most likely latent class membership²¹⁰ (See Appendix Table D5 for the logits).

Finally, in Step 3 of Vermunt's, modal class assignment is designated as the proxy variable to represent latent class membership and PTSD is regressed on the religiosity, spirituality, and R/S, respectively, where the variables of race and SES are included for overall adjusted

model and excluded for the unadjusted model. For the stratified models, SES is adjusted for in the race stratified models, and race is adjusted for in the SES-stratified models.

This final step produces the findings for the Wald Test of parameter constraints, which assesses whether the regression of PTD on religiosity, spirituality, and R/S, respectively, differs significantly between the two latent classes. Because latent class membership cannot be treated as an independent variable on which the outcome variable can be regressed in Mplus, effect modification by the respective protective factors was inferred by examining whether the effect of PTD on religiosity, spirituality, and R/S, respectively, differed significantly across the classes as informed by a Wald test ($\alpha = 0.05$).

Because interaction effects are symmetric, if latent class membership moderates the relationship between religiosity and PTD, for example, this is statistically equivalent to stating that religiosity moderates the relationship between latent class membership and PTD.²¹² While these models are statistically equivalent, my conceptual framework specifically informed my selection of religiosity, spirituality, and R/S as the potential effect modifiers, not latent class membership. Therefore, while the results of the Wald test in Tables 19 and 20 specifically test for differences in the relationship between religiosity, spirituality, and R/S, respectively, and PTD by latent class membership, the interpretation is applied to religiosity, spirituality, and R/S as the effect modifiers of the relationship between latent class membership and PTD.

I conducted a Wald test to assess whether religiosity, spirituality, and R/S modified the relationship between the two latent classes and PTD in the overall sample (Table 19) as well as in stratified analyses by race and SES (Table 20).

Based on the findings of the Wald test for the regression of PTD on the potential protective factors in the overall sample, supplemental Wald test analyses were conducted to

evaluate whether the threshold for PTD differed significantly between the low ACEs and high ACEs class at each of the four levels of religiosity and spirituality, and at each of the 12 levels of R/S, in the overall sample (Table 21; Figures 21-23). Table 21 reports the thresholds of PTD, probabilities of PTD, the results of the Wald test, as well as the odds ratios (ORs) which represent the odds of PTD in the high ACEs class compared to the odds of PTD in the low ACEs class among individuals at a specific level of religiosity, spirituality, and R/S. The relationship between the threshold parameter and the probability of PTD is depicted in Equation 1.12 (Bauer and Steinley, 2020, p. 7.4).²¹⁰ Supplemental analyses were not conducted for the race and SES stratified models but will be considered as next steps for future analyses. All latent class analyses were conducted using Mplus Version 8.6 statistical software package (Muthén & Muthén, 1998-2017).²⁰⁸

6.2.4 IRB Approval

This study was determined to be exempt from human subjects research under 45 CFR 46.104(d) 4(ii) by the Institutional Review Board (IRB) at Michigan State University. Permission for use of the Add Health Restricted-Use dataset was granted by the Carolina Population Center at the University of North Carolina at Chapel Hill.

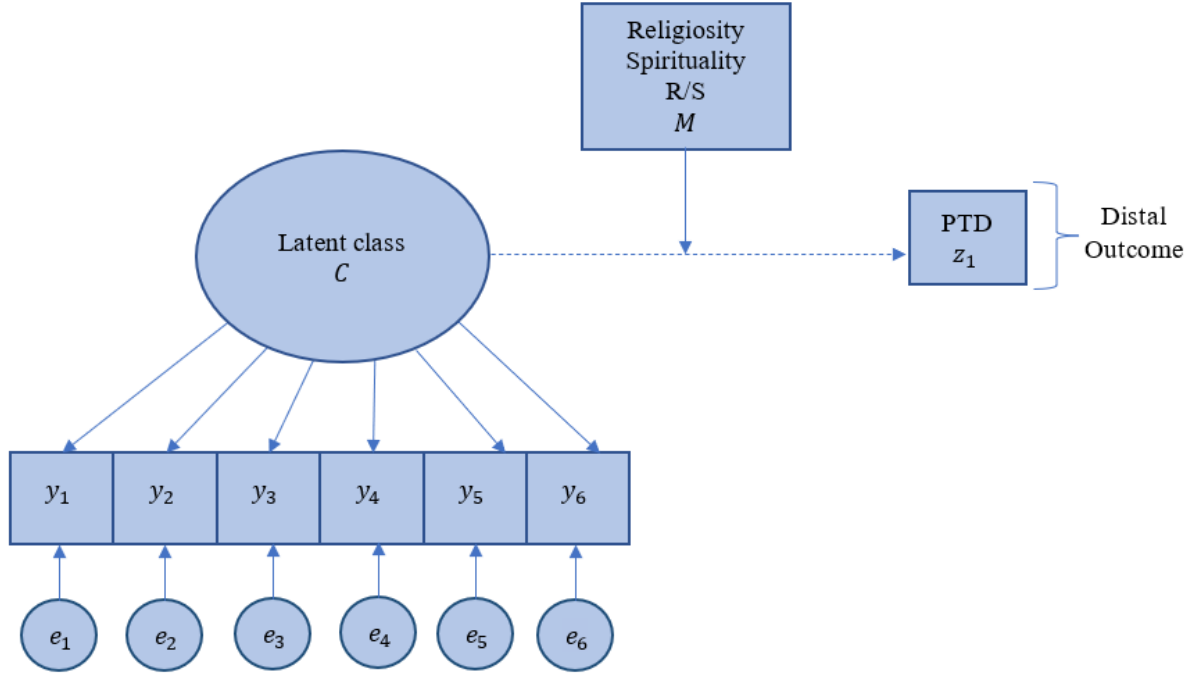


Figure 20. Conceptual diagram of the moderation analysis of the association between latent class membership and PTD by religiosity, spirituality, and R/S in the National Longitudinal Study of Adolescent to Adult Health (n=3,884), 1994-2018 where y1 (physical abuse); y2 (sexual abuse); y3 (emotional abuse); y4 (neglect); y5 (attempted suicide or death by family members); y6 (foster care placement); z1 (PTD); e1-e6 (measurement error for each indicator); and M represents each of the three potential protective factors, religiosity, spirituality, and R/S.

$$(1.11) \text{logit}(Y_i) = \beta_0 + \beta_1(x_{1,i}) + \beta_2(x_2) + \beta_3(x_3 * x_2) + \beta_4(x_4) + \beta_5(x_5) + \beta_6(x_6) + \beta_7(x_7) + \beta_8(x_8) + \beta_9(x_9) + \beta_{10}(x_{10}) + \beta_{11}(x_{11})$$

where $Y_i = 1$ for PTB, $x_1 =$ membership in class i , $x_2 =$ protective factor (i.e., religiosity, spirituality, R/S, respectively), $x_3 * x_2 =$ interaction term between class membership and protective factor; and where 'White' is the reference group for race and 'High SES' is the reference group for SES such that:

$$x_4 = \begin{cases} 1 & \text{if Black} \\ 0 & \text{if not Black} \end{cases}; x_5 = \begin{cases} 1 & \text{if Hispanic} \\ 0 & \text{if not Hispanic} \end{cases}; x_6 = \begin{cases} 1 & \text{if Asian} \\ 0 & \text{if not Asian} \end{cases};$$

$$x_7 = \begin{cases} 1 & \text{if Pacific Islander} \\ 0 & \text{if not Pacific Islander} \end{cases}; x_8 = \begin{cases} 1 & \text{if AIAN} \\ 0 & \text{if not AIAN} \end{cases}; x_9 = \begin{cases} 1 & \text{if Other} \\ 0 & \text{if not Other} \end{cases};$$

$$x_{10} = \begin{cases} 1 & \text{if low SES} \\ 0 & \text{if not low SES} \end{cases}; x_{11} = \begin{cases} 1 & \text{if middle SES} \\ 0 & \text{if not middle SES} \end{cases}$$

(1.12) $\delta_{jk} = \frac{1}{1 + e^{v_{jk}}}$ where δ_{jk} is the probability of PTD for an individual in class k at a particular level of a protective factor, and v_{jk} is the threshold parameter estimate (Bauer and Steinley, 2020, p. 7.4)²¹⁰

6.3 Results

6.3.1 Descriptive Analyses

The descriptive characteristics of the analytic sample (n=3,884) are depicted in Table 17. Most of the women in my sample were White (68.7%), from middle SES backgrounds (61.1%), and married or cohabiting (75.7%). The prevalence of PTD was roughly 12%. Approximately 48% of women reported emotional abuse, followed by neglect (42.3%), physical abuse (20.2%), sexual abuse (6.3%), family member suicide attempt or death (6.1%), and foster care placement (2.7%). On a scale from 0-3 where 0 indicates ‘not religious/spiritual at all’ and 3 indicates ‘very religious/spiritual,’ the mean score for religiosity was 1.4 (SE=.02) and the mean score for spirituality was 1.5 (SE=.02) (Table 17). The mean score for R/S was 10.4 (SE=.06), where the distribution of scores ranged from 3 -15 and higher scores indicated higher R/S (Table 17). There were no statistically significant differences in religiosity, spirituality, or R/S between the low ACEs and high ACEs class (Wald Test $p > .05$ for all means, respectively) (Table 18).

Table 17. Descriptive characteristics of the analytic sample in the National Longitudinal Study of Adolescent to Adult Health, 1994-2018 (n=3,884)

	n (Weighted %)¹
Overall	3,884
Race²	
White	2297 (68.7%)
Black	723 (14.4%)
Hispanic	517 (10.3%)
Asian	197 (2.9%)
Pacific Islander	32 (0.5%)
American Indian or Alaska Native	89 (2.4%)
Other	29 (0.8%)
Socioeconomic status (SES)	
Low	487 (12.5%)
Middle	2382 (61.1%)
High	1015 (26.4%)
Marital status³	
Married/Cohabiting	2893 (75.7%)
Not Married/Cohabiting	667 (16.1%)
Maternal age at time of delivery (years)	
≤ 19	562 (14.5%)
20-24	1048 (28.2%)
25-29	1021 (24.9%)
30-34	807 (20.7%)
≥ 35	248 (6.2%)
Preterm Delivery (PTD)	514 (11.8%)
Adverse childhood experiences (ACEs)⁴	
Sexual abuse	217 (6.3%)
Physical abuse	738 (20.2%)
Emotional abuse	1793 (47.7%)
Neglect	1614 (42.3%)
Family member suicide attempt or death	216 (6.1%)
Foster care placement	90 (2.7%)
Protective Factors	Mean (SE)
Religiosity	1.4 (.02)
Spirituality	1.5 (.02)
R/S	10.4 (.06)

¹percentages are survey-weighted to account for stratified sampling design of Add Health

²n=324 women with missing data on marital status

³n=198 women with missing data on maternal age

⁴n=117 women with missing data on any ACEs; ACEs are not mutually exclusive categories

Table 18. Means for religiosity, spirituality, and R/S by latent class in the National Longitudinal Study of Adolescent to Adult Health (n=3,884), 1994-2018

Variable	Class 1 Mean (Low ACEs Class)	Class 2 Mean (High ACEs Class)	Wald Test of Parameter Constraints
Religiosity	1.4	1.4	$X^2 = .001$, df = 1 p -value = 0.97
Spirituality	1.5	1.5	$X^2 = .85$, df=1 p -value = 0.36
R/S	10.4	10.4	$X^2 = .03$, df=1 p -value=0.85

6.3.2 Latent Class Moderation Analyses

6.3.2.1 Overall Sample

Religiosity. The results of the Wald test of parameter constraints indicated that the association between religiosity and odds of PTD differed significantly by latent class membership in both the unadjusted and adjusted models ($X^2=5.2$, $p=.02$; $X^2=5.9$, $p=.02$, respectively) (Table 19). Therefore, this provides statistical evidence that the association between latent class membership and odds of PTD differed significantly by religiosity.

Spirituality. The results of the Wald test of parameter constraints indicated that the association between spirituality and PTD differed significantly by latent class membership in both unadjusted and adjusted models ($X^2=5.5$, $p=.02$; $X^2=5.7$, $p=.02$, respectively) (Table 19). This provides statistical evidence that spirituality modifies the association between latent class membership and odds of PTD.

R/S. The Wald test of parameter constraints showed that the association between R/S and PTD differed significantly by latent class in both unadjusted and adjusted models ($X^2=6.3$,

$p=.01$; $X^2=6.6$, $p=.01$) (Table 19). This provides statistical evidence that R/S modifies the association between latent class membership and odds of PTD.

Table 19. Wald Test for the assessment of statistically significant differences in the regression of PTD on religiosity, spirituality, and R/S by latent class in the National Longitudinal Study of Adolescent to Adult Health (n=3,884), 1994-2018¹

Wald Test of Parameter Constraints	
Religiosity	
Model 1 (Unadjusted)	$X^2=5.2$, $df=1$ p-value = .02
Model 2 (Adjusted)	$X^2=5.9$, $df=1$ p-value = .02
Spirituality	
Model 1 (Unadjusted)	$X^2=5.5$, $df=1$ p-value = .02
Model 2 (Adjusted)	$X^2=5.7$, $df=1$ p-value = .02
R/S	
Model 1 (Unadjusted)	$X^2=6.3$, $df=1$ p-value = .01
Model 2 (Adjusted)	$X^2=6.6$, $df=1$ p-value = .01

¹Adjusted models control for race and SES
 Bolded values indicate statistical significance at $p<.05$

6.3.2.2 Stratified Latent Class Moderation Analyses

Table 20 presents the results for the stratified analyses by race and SES subgroups. While the majority of findings were not significant, some within-group differences are noteworthy of discussion.

Religiosity. The association between religiosity and PTD differed significantly by latent class membership among White women ($X^2=4.9$, $p=.03$), Pacific Islander women ($X^2=508.5$,

$p < .001$), and women who identified as Other ($X^2 = 24.4$, $p < .001$). The association between religiosity and odds of PTD varied by latent class membership among middle SES women only ($X^2 = 4.7$, $p = .03$). These results provide statistical evidence that religiosity significantly modifies the association between latent class membership and PTD among White women, Pacific Islander women, women who identified as Other, and middle SES women.

Spirituality. There were no statistically significant differences in the association between spirituality and the odds of PTD by latent class membership for any of the SES subgroups (p -values for all Wald Tests $> .05$, respectively) or the race subgroups, with the exception of women who identified as Other ($X^2 = 205.1$, $p < .001$). This means that spirituality significantly modified the association between latent class membership and odds of PTD only among women who identified as Other, but not for the majority of the race or SES subgroups.

R/S. In the race-stratified analyses, the association between R/S and odds of PTD varied by latent class membership among White women ($X^2 = 5.6$, $p = .02$) and Pacific Islander women only ($X^2 = 365.9$, $p < .001$). In the SES-stratified analyses, the association between R/S and the odds of PTD varied by latent class membership among high SES women only ($X^2 = 12.2$, $p < .001$). These results indicate that R/S significantly modified the association between latent class membership and odds of PTD among White women, Pacific Islander women, and high SES women, but not for the majority of race or SES subgroups.

Table 20. Race and SES-stratified Wald Test for the assessment of statistically significant differences in the regression of PTD on religiosity, spirituality, and R/S by latent class in the National Longitudinal Study of Adolescent to Adult Health (n=3,884), 1994-2018

Wald Test of Parameter Constraints	
Religiosity	
Race subgroups	
White (n=2297)	X2=4.9, df=1, p-value = .03
Black (n=723)	X2=2.6, df=1, p-value=0.1
Hispanic (n=517)	X2=.04, df=1, p-value = 0.8
Asian (n=197)	X2=.05, df=1, p-value = 0.8
Pacific Islander (n=32)	X2=508.5, df=1, p-value <.001
AI/AN (n=89)	X2=2.6, df=1, p-value = 0.1
Other (n=29)	X2=24.4, df=1, p-value<.001
SES subgroups	
Low SES (n=487)	X2=3.4, df=1, p-value=.06
Middle SES (n=2382)	X2=4.7, df=1, p-value=.03
High SES (n=1015)	X2=0.4, df=1, p-value=0.5
Spirituality	
Race subgroups	
White (n=2297)	X2=3.9, df=1, p-value = .05
Black (n=723)	X2=1.8, df=1, p-value = 0.2
Hispanic (n=517)	X2=.07, df=1, p-value = 0.8
Asian (n=197)	X2=0.8, df=1, p-value = 0.4
Pacific Islander (n=32)	X2=0.1, df=1, p-value = 0.8
AI/AN (n=89)	X2=3.6, df=1, p-value = .06
Other (n=29)	X2=205.1, df=1, p-value <.001
SES subgroups	
Low SES (n=487)	X2=3.5, df=1, p-value=.06
Middle SES (n=2382)	X2=2.5, df=1, p-value = 0.1
High SES (n=1015)	X2=3.2, df=1, p-value=0.1
R/S	
Race subgroups	
White (n=2297)	X2=5.5, df=1, p-value=.02
Black (n=723)	X2=1.7, df=1, p-value=0.2
Hispanic (n=517)	X2=0.1, df=1, p-value=0.8
Asian (n=197)	X2=0.3, df=1, p-value=0.3
Pacific Islander (n=32)	X2=365.9, df=1, p-value < .001
AI/AN (n=89)	X2=0.3, df=1, p-value=0.6
Other (n=29)	X2=0.8, df=1, p-value=0.4

Table 20 (cont'd).

SES subgroups	
Low SES (n=487)	X ² =0.6, df=1, p-value=0.5
Middle SES (n=2382)	X ² =1.6, df=1, p-value=0.2
High SES (1015)	X ² =12.2, df=1, p-value<.001

Bolded values indicate a statistically significant difference at p<.05;
AI/AN American Indian or Alaska Native

6.3.3 Supplemental Latent Class Moderation Analyses

Religiosity. Among those who indicated they were not religious at all (religiosity = 0), the odds of PTD in the high ACEs class was 80% lower than the odds of PTD in the low ACEs class (OR 0.2, 95% CI 0.1-0.9), suggesting that for women who experience high levels of ACEs, religiosity is not a protective factor against the odds of PTD (Table 21). However, among those who indicated they were very religious (religiosity = 3), the odds of PTD in the high ACEs class was 3.4 times the odds of PTD in the low ACEs class, further confirming that high levels of religiosity does not appear to be protective for those in the high ACEs class; in fact, these results suggest that religiosity may be a risk factor for PTD. Figure 21 depicts the trend that as religiosity increases for those in the low ACEs class, the probability of PTD decreases, suggesting that religiosity is protective for those in the low ACEs class. However, for those in the high ACEs class, the probability of PTD jumped significantly for those who are very religious (p(PTD) = 0.22)).

Spirituality. The findings for spirituality showed a similar trend to religiosity. Specifically, among women who indicated they were very spiritual, the odds of PTD among those in the high ACEs class was 3.4 times the odds of PTD among those in the low ACEs class (OR 3.4, 95% CI 1.4-8.4) (Table 21), suggesting that spirituality does not appear to be a protective factor against PTD among women with high levels of ACEs. Figure 22 graphically depicts these findings, showing that the probability of PTD for those in the high ACEs class was

highest at the highest level of spirituality ($p(\text{PTD}) = 0.24$), compared to $p(\text{PTD}) = .05$ for those at the lowest level of spirituality. Among those in the low ACEs class, the probability of PTD seemed to decrease overall as spirituality increased, such that the probability of PTD was lowest for those who were very spiritual ($p(\text{PTD}) = 0.10$) compared to those who were not spiritual at all ($p(\text{PTD})=0.18$).

R/S. The trend for R/S did not appear to be consistent across the 12 levels of R/S (Table 21; Figure 23). At the lowest level of R/S ($R/S = 3$), the probability of PTD among women in the low ACEs class was higher than the probability of PTD among women in the high ACEs class, but this difference in the thresholds for PTD was not statistically significant ($p=0.34$) (Table 21). However, an increase in R/S by one unit (from 3 to 4) led to a dramatic change in the trend (Figure 23), such that the probability of PTD among women in the high ACEs class who reported an R/S level of 4 was significantly higher than the probability of PT among women in the low ACEs class who reported an R/S level of 4 ($p<.01$; Table 21). This pattern remained at the highest level of R/S (15), where the probability of PTD among women in the high ACEs class who reported high levels of R/S was significantly higher than the probability of PTD among women in the low ACEs class who reported high levels of R/S ($p=.04$; Table 21). These results suggest that neither low nor high levels of R/S are necessarily protective against the risk of PTD for women in the high ACEs class.

Table 21. Threshold, probability, and odds ratio estimates of PTD comparing two latent classes across stratified levels of religiosity, spirituality, and R/S in the National Longitudinal Study of Adolescent to Adult Health (n=3,884), 1994-2018¹

	Class 1 Low ACEs Class		Class 2 High ACEs Class			
	p(PTD)	Threshold	p(PTD)	Threshold	Wald Test of Parameter Constraints ²	Odds ratio ² (95% CI)
Religiosity						
Not religious at all (n=771)	0.17	1.523	0.05	2.943	X ² =4.9, df=1 p-value = 0.03	0.2 (0.1, 0.9)
Slightly religious (n=1367)	0.11	2.280	0.17	1.764	X ² =2.7, df=1 p-value = 0.1	1.7 (0.9, 3.1)
Moderately religious (n=1321)	0.11	2.306	0.08	2.601	X ² =0.66, df=1 p-value = 0.42	0.7 (0.4, 1.5)
Very religious (n=425)	0.09	3.659	0.22	2.434	X ² =5.3, df=1 p-value = 0.02	3.4 (1.2, 9.7)
Spirituality						
Not spiritual at all (n=607)	0.18	1.084	0.05	2.590	X ² =3.5, df=1 p-value = 0.06	0.2 (.05, 1.1)
Slightly spiritual (n=1348)	0.10	2.497	0.13	2.186	X ² =0.8, df=1 p-value = 0.4	1.4 (0.7, 2.6)

Table 21 (cont'd).

Moderately spiritual (n=1287)	0.12	2.311	0.09	2.649	$X^2=0.9$, df=1 p-value = 0.3	0.7 (0.4, 1.4)
Very spiritual (n=642)	0.1	2.880	0.24	1.656	$X^2=7.1$, df=1 p-value <.01	3.4 (1.4, 8.4)
R/S						
3 (n=71)	0.35	-0.018	0.09	1.353	$X^2=0.9$, df=1 p-value = 0.34	0.3 (0.0, 4.2)
4 (n=32)	0.10	15.888	0.59	11.144	$X^2=9.4$, df=1 p-value <.01	114.9 (5.5, 2401.4)
5 (n=54)	0.23	1.289	0.16	1.543	$X^2=0.0$, df=1 p-value = 0.9	0.8 (0.0,13.8)
6 (n=172)	0.10	1.949	0.03	3.235	$X^2=1.3$, df=1 p-value = 0.2	0.3 (0.0, 2.5)
7 (n=181)	0.11	2.290	0.08	2.722	$X^2=0.1$, df=1 p-value = 0.7	0.6 (0.1, 6.1)
8 (n=260)	0.16	1.033	0.11	1.493	$X^2=0.3$, df=1 p-value = 0.6	0.6 (0.1, 3.5)
9 (n=563)	0.13	2.169	0.06	3.093	$X^2=2.0$, df=1 p-value = 0.2	0.4 (0.1, 1.4)
10 (n=530)	0.12	2.150	0.11	2.195	$X^2=0.0$, df=1 p-value = 0.9	1.0 (0.3, 2.7)
11 (n=554)	0.12	2.058	0.14	1.842	$X^2=0.2$, df=1 p-value = 0.7	1.2 (0.4, 3.6)
12 (n=663)	0.10	2.564	0.18	1.870	$X^2=2.6$, df=1 p-value = 0.10	2.0 (0.9, 4.6)
13 (n=316)	0.08	2.796	0.10	2.479	$X^2=0.2$, df=1 p-value = 0.6	1.4 (0.4, 4.6)

Table 21 (cont'd).

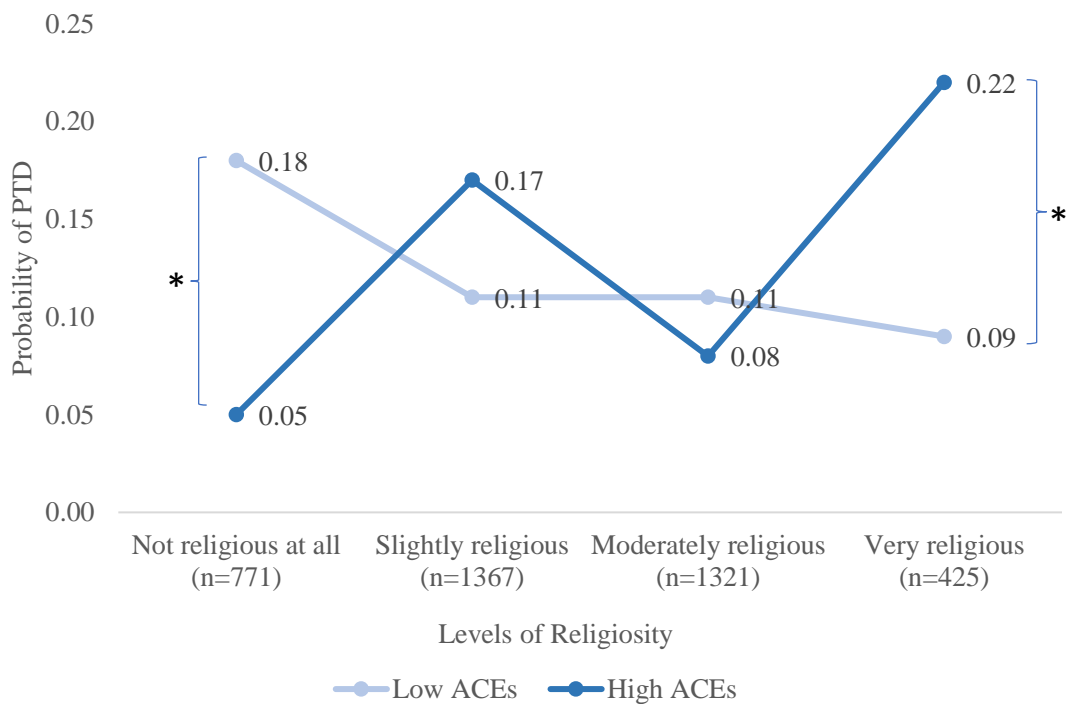
14 (n=204)	0.12	1.643	0.03	2.960	X ² =2.0, df=1 p-value = 0.2	0.3 (0.0, 1.7)
15 (n=284)	0.08	2.996	0.22	1.714	X ² =4.2, df=1 p-value = 0.04	3.6 (1.1,12.2)

¹Probability estimates are adjusted for race and SES

²OR comparing the odds of PTD in the high ACEs class compared to the low ACEs class

Bolded values indicate statistical significance at p<.05.

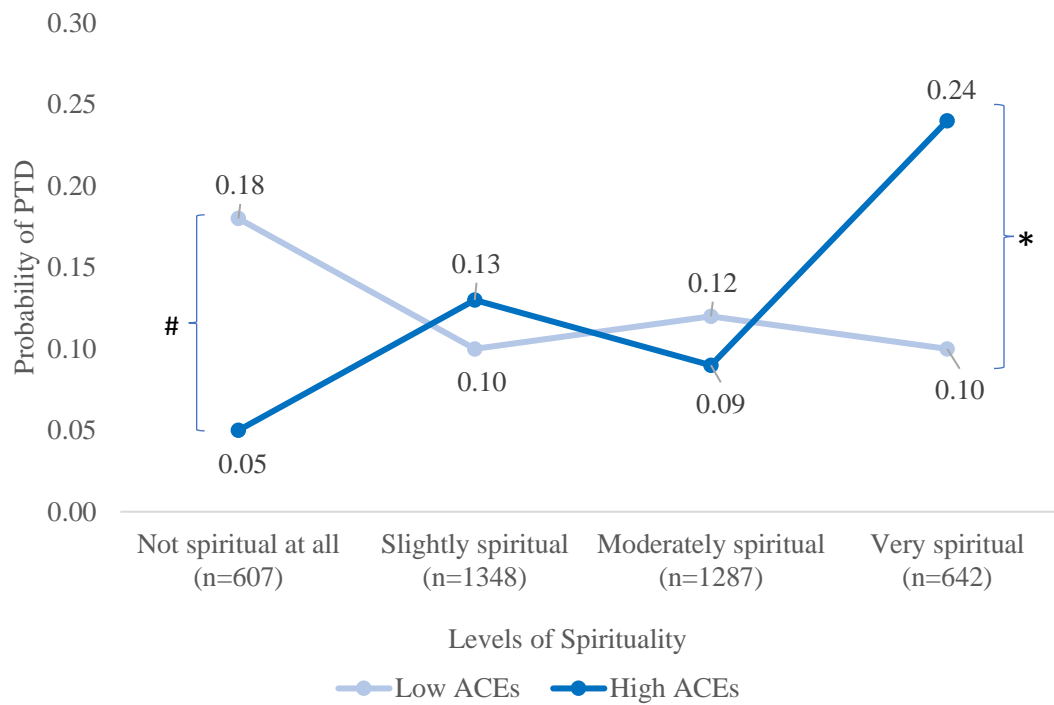
Figure 21. Comparing the probability of PTD between two the low and high ACEs class across levels of religiosity in the National Longitudinal Study of Adolescent to Adult Health (n=3,884), 1994-2018



¹Probability estimates are adjusted for race and SES

*denotes statistically significant difference at p<.05

Figure 22. Comparing the probability of PTD between the low and the high ACEs class across levels of spirituality in the National Longitudinal Study of Adolescent to Adult Health (n=3,884), 1994-2018¹

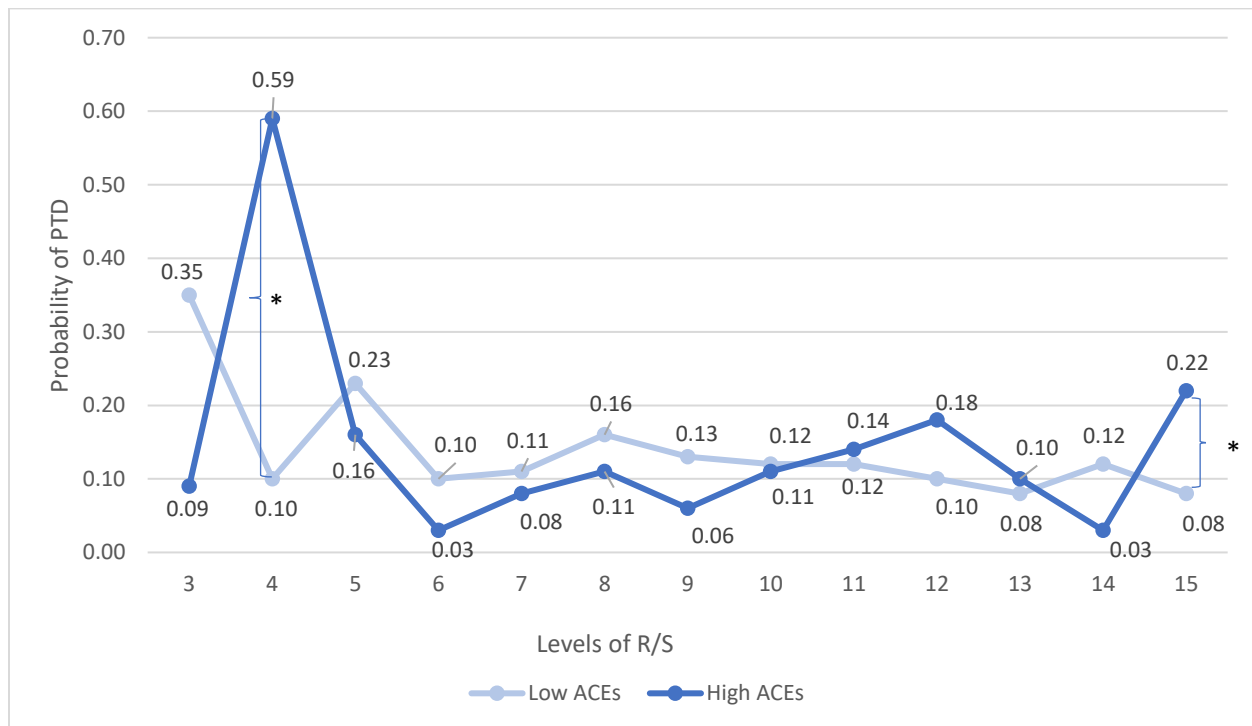


¹Probability estimates are adjusted for race and SES

*denotes statistically significant difference at $p < .05$

#denotes approaching statistically significant difference ($p = .06$)

Figure 23. Comparing the probability of PTD between the low and the high ACEs class across levels of R/S in the National Longitudinal Study of Adolescent to Adult Health (n=3,884), 1994-2018¹



¹Probability estimates adjusted for race and SES; Levels of R/S range from 3 (lowest level of R/S) to 15 (highest level of R/S)

*denotes statistically significant difference at $p < .05$

6.4 Discussion

In this study, I **1)** Assessed whether the mean levels of religiosity, spirituality, and a composite construct of religiosity and spirituality (R/S), differed across the high ACEs latent class and the low ACEs latent class; **2)** Evaluated the role that religiosity, spirituality, and R/S play in the association between ACEs and PTD and **3)** Examined whether these potential protective factors operated differently within race and SES subgroups using data on n=3,884 women who gave birth in the National Longitudinal Study of Adolescent to Adult Health (1994-2018).

The results of this study can be summarized by five key findings. **First**, the mean levels of religiosity, spirituality, and R/S did not differ significantly between the low ACEs class and the high ACEs class. **Second**, the association between latent class membership and the odds of PTD differed significantly by religiosity, spirituality, and R/S in the overall sample of women. **Third**, while religiosity, spirituality, and R/S did not modify the association between latent class membership and odds of PTD for the majority of the race and SES stratified analyses, significant differences existed for a few subgroups of women, including White women, Pacific Islander women, women who identified as Other, middle SES women, and high SES women. **Fourth**, the odds of PTD was significantly higher among women in the high ACEs class who were very religious or spiritual than the odds of PTD among women in the low ACEs class who were very religious or spiritual (OR 3.4, 95% CI 1.2-9.7; OR 3.4, 95% CI 1.4-8.4, respectively), suggesting that neither religiosity nor spirituality appear to be protective against PTD for women in the high ACEs class, but they were so for women in the low ACEs class. **Finally**, R/S was not protective against PTD among women in the high ACEs class, as women with both lower and higher levels of R/S exhibited a significantly higher probability of PTD compared to women in the low ACEs

class with the same levels of R/S, although the trend was not consistent across the range of R/S values.

As this is the first study in the literature, to my knowledge, to evaluate the relationship between ACEs, religiosity, spirituality, and PTD as the primary outcome of interest, there are no existing studies with which I can make a direct comparison of findings regarding PTD. However, I will interpret my findings in the context of a few studies in the literature which have evaluated whether religiosity and spirituality promote positive health outcomes among individuals with ACEs, as follows.

My finding that religiosity and spirituality did not differ significantly between those in the low ACEs class and the high ACEs class concurs with that of Liu et al. (2021) who did not find significant differences in spirituality among those with and without adverse childhood experiences in a sample of n=1929 adults from Singapore.²²⁷ However, this study varied from mine in study population demographics, sample size, country of study implementation, and outcomes assessed (i.e., mental health disorders).

While my study found evidence that religiosity, spirituality, and R/S moderated the relationship between latent class membership and PTD, the pattern of the moderation was not as I expected. Specifically, the odds of PTD were significantly higher for women in the high ACEs class who were very religious, very spiritual, or had high levels of R/S compared to women in the low ACEs class, suggesting that for women who experience high levels of ACEs, religiosity, spirituality, and R/S may not be protective against the occurrence of PTD, but may be protective for women in the low ACEs class. My findings concur with Mefford et al. (2021), who found that lower levels of religious/spiritual values/beliefs were associated with higher, not lower, levels of adult life satisfaction in a sample of n=132 adults who experienced childhood loss.²²⁸

Furthermore, Brockie et al. (2018) did not find evidence that spirituality moderated the association between ACEs and self-reported physical health, although social support and diabetes-specific support did, in a sample of $n=192$ American Indian adults with Type 2 diabetes.²²⁹ Similarly, Homan & Hollenberger (2021) found that religious identification, private religious practice, and religious service attendance did not moderate the association between ACEs and self-rated physical health, but religious support did ($\beta=.03$, $p<.05$) in a sample of $n=4041$ adults from the Midlife in the United States Study.²²⁵ In a sample of $n=241$ African American adolescents from Texas, Freeny et al. (2021) found that while spirituality significantly reduced the likelihood of depression ($\beta=18.5$, $p<.05$), they did not find evidence that spirituality moderated the association between ACEs and odds of depression ($\beta=1.57$, $p\text{-value}>.05$).²²⁶

While these studies differed from mine in study population, sample size, and assessment of ACEs, spirituality and religiosity, and health outcomes, these findings collectively suggest that religiosity and spirituality may not necessarily buffer against the impact of ACEs on various health outcomes. My findings build upon this body of work by providing evidence that religiosity and spirituality do not appear to be protective against PTD among women with high levels of ACEs.

The finding that higher levels of religiosity, spirituality, and R/S were associated with a significantly lower odds of PTD among women in the low ACEs class compared to women in the high ACEs class support the conceptual framework of an adversity threshold, whereby an individual has a "...a limited capacity for handling traumatic exposures. When this capacity is surpassed by the demands of adjusting to repeated or prolonged trauma exposure, disturbances emerge" (Masten, 2014, p. 123).⁷² Once this adversity threshold is exceeded, a factor's ability to confer "protection" may also be diminished.⁷³ For example, for individuals who experience

tremendously high levels of adversity, factors which are otherwise protective for low to moderate levels of adversity, may lose their ability to confer protection, due to the severity of the adversity that undermines the individual's ability to cope, even in the presence of the protective factor.

In addition, some research suggests that not all ACEs are “equal” in their impact on PTD. For example, research suggests that sexual abuse is a type of ACE that appears to be more detrimental in its health impacts than other ACEs.^{19,61,62} As sexual abuse was a more prevalent characteristic in the high ACEs class, this could play a role in the findings. Moreover, while there were no significant differences in exhibition of religiosity and spirituality between women in the high ACEs vs. low ACEs class, religiosity and spirituality may function differently for women who experience high levels of ACEs.

Indeed, in a sample of n=1800 individuals aged 15 years and older in the Czech Republic, Kosarkova et al. (2020) found that both religious (R) and nonreligious (NR) individuals who reported adverse childhood experiences such as sexual abuse perceived God in a less positive light.²³⁰ For example, they had a lower odds of viewing God as ‘loving’ (NR: OR 0.92, 95% CI 0.8-0.98; R: OR 0.86, 95% CI 0.77-0.97) or ‘forgiving’ (NR: OR 0.75, 95% CI 0.64-0.89; R: OR 0.82, 95% CI 0.68-0.99).²³⁰ Furthermore, in a nationally representative sample of n=1000 individuals aged 15 and older in the Czech Republic, Janu et al. (2022) found that five types of childhood trauma experiences (emotional abuse, physical abuse, sexual abuse, emotional neglect and physical neglect) were all associated with higher odds of religious and spiritual struggles (i.e., feelings of anger toward God, questioning religion or spirituality).²³¹ While these studies did not specifically differentiate among those who had higher versus lower levels of ACEs, these results suggest that religiosity and spirituality may function differently among those who experience ACEs, particularly higher levels of ACEs. More specifically, as spirituality and

religion can lead to feelings of guilt,²³² spirituality and religiosity may not necessarily offer solace for individuals who experience high levels of childhood adversity.

The finding that the majority of the race and SES stratified moderation analyses were not significant is unexpected, given racial and socioeconomic patterns in the exhibition of religiosity and spirituality,^{134,137} as well as the racial and SES disparities in the prevalence of ACEs.^{28,30} This lack of significant findings may be due to the small sample size for the majority of non-White women. However, as I used a large, diverse, nationally representative dataset with a plethora of data on ACEs for my study, this highlights the data gaps regarding the participation of non-White women in longitudinal studies which precludes more precise estimations of the relationships between protective factors, PTD, and ACEs.

Furthermore, our findings could be influenced by influenced by selection bias. For example, women who experience very high levels of childhood adversity may decide not to have children or may not go on to have pregnancies which end in a live birth. Indeed, as ACEs have been linked to pregnancy loss and unintended pregnancy,^{39,58,111} women who have a miscarriage or who might elect to have an abortion as a result of an unintended pregnancy may not be captured by our denominator of women with pregnancies that end in a live birth.

Strengths & Limitations

Strengths. One of the foremost strengths of this study is its contribution as the first empirical study in the literature to study the relationship between ACEs, PTD, religiosity, and spirituality. I used a large, nationally representative dataset with over 20 years of data on a variety of psychosocial, health, and demographic variables to address my research questions. My study evaluated the impacts of religiosity, spirituality, as well as an aggregate construct of both (R/S), on PTD, while previous studies have commonly focused on one or the other. This is an

important contribution because while religiosity and spirituality are related concepts, they exhibit important differences, which may manifest differently on health outcomes. Furthermore, while previous studies exploring the impacts of ACEs on PTSD have commonly operationalized ACEs as a count or sum score, my study evaluated more subtle patterns in the interactions between different ACEs using latent class methods to assess whether co-occurring patterns of ACEs may play an important role in PTSD occurrence and disparities, a methodological contribution to the perinatal literature.

Limitations. While this study entailed multiple strengths, the findings should be interpreted in the context of its limitations. First, religiosity and spirituality were each assessed by one question only. As the literature shows, these constructs are complex and multidimensional,¹⁷⁵ therefore, future studies should incorporate more detailed scales of these constructs. Furthermore, the religiosity and spirituality variables were assessed at one time point only (in Wave III). However, religiosity and spirituality are fluid constructs and may play a stronger role at different time points and periods of development throughout an individual's life. Indeed, studies show that religiosity tends to decrease in young adulthood but increase in later life.²³³ Religiosity and spirituality were captured in Wave III when the women were young adults, and this may explain the lower overall mean levels of religiosity and spirituality in the sample.

While Add Health is a nationally representative dataset, my analytic sample was not, and there were small sample sizes for non-White women in my analytic sample, which undermines generalizability of the findings to these groups. As Add Health is one of the largest and most nationally representative datasets with a diverse sample of adolescents and with a plethora of

data on environmental, psychosocial, and biological lifestyle factors, this highlights a broader challenge for participation rates of non-White women in national prospective studies.

Finally, while I was able to assess overall evidence of an interaction between latent class membership and each of the potential protective factors using the Wald test, I encountered a programming limitation as I was not able to specifically assess whether the beta coefficients for the regression of PTD on latent class membership differed across levels of religiosity, spirituality, and R/S. Therefore, I inferred effect modification of the association between latent class membership and PTD by religiosity, spirituality, and R/S by assessing for statistically significant differences in the regression of PTD on each potential protective factor between the latent classes and conducted follow-up analyses to assess how the probability of PTD for each latent class differs across levels of religiosity, spirituality, and R/S.

6.5 Conclusion

In this study, I applied latent class moderation methods using data on n=3,884 women from the National Longitudinal Study of Adolescent to Adult Health to 1) Assess whether the mean levels of religiosity, spirituality, and R/S differed between the high ACEs class and the low ACEs class; 2) Evaluate the role that religiosity, spirituality, and R/S play in the association between latent class membership and PTD and 3) Examine whether these potential protective factors operated differently within race and SES subgroups. Overall, I found evidence that 1) religiosity, spirituality, and R/S did not protect against PTD among women with high levels of ACEs but did so for women with low levels of ACEs, and 2) religiosity, spirituality, and R/S did not moderate the relationship between latent class membership and odds of PTD for the majority of race and SES subgroups of women.

These results suggest that religiosity and spirituality may be protective against PTD for women who experience lower levels of ACEs, but not for those who experience higher levels of ACEs. These findings support the argument for a adversity threshold below which protective factors like religiosity and spirituality might function optimally to reduce the risk of PTD, and above which weakens the protective influence of religiosity and spirituality on PTD.^{32,72,73} Future studies should assess whether additional protective factors at the individual, interpersonal, and community level similarly display this threshold functioning on the risk of PTD.

6.6 Acknowledgements

This research uses data from Add Health, funded by grant P01 HD31921 (Harris) from the Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD), with cooperative funding from 23 other federal agencies and foundations. Add Health is currently directed by Robert A. Hummer and funded by the National Institute on Aging cooperative agreements U01 AG071448 (Hummer) and U01AG071450 (Aiello and Hummer) at the University of North Carolina at Chapel Hill. Add Health was designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris at the University of North Carolina at Chapel Hill

CHAPTER 7: DISCUSSION

7.1 Summary of Findings

In this dissertation, I sought to investigate the relationship between adverse childhood experiences, PTSD, and two types of potential protective factors (religiosity and spirituality) using a health equity framework, person-centered methodologic approaches, interdisciplinary theories, and a large, longitudinal sample of women. Specifically, this body of work achieved three specific aims:

- **Aim 1.** Determine the association between specific adverse childhood experiences and odds of PTSD and evaluate whether this relationship differs across race and socioeconomic status.
- **Aim 2.** Identify subgroups of women characterized by early life patterns of ACEs and determine the association between subgroup membership and odds of PTSD.
- **Aim 3.** Examine the role that potential protective factors (i.e., religiosity and spirituality) play in the association between ACEs and odds of PTSD, and whether these factors operate differently by race and SES.

Using data on $n=3,884$ women from the National Longitudinal Study of Adolescent to Adult Health (Add Health, 1994-2018), I discovered the following key findings for each aim:

- **Aim 1. First**, I found that the mean ACE score differed significantly by race ($p<.001$), but not SES ($p=0.6$). **Secondly**, the prevalence of PTSD did not differ significantly by race ($p=0.3$) or SES ($p=0.6$). **Third**, there were no statistically significant associations between any of the six ACEs and odds of PTSD among women overall (all p -values $>.05$, respectively). **Fourth**, due to small sizes for some cells, I was unable to obtain precise OR estimates for the association between ACEs and

PTD for select subgroups of women, including low SES women, Pacific Islander women, American Indian or Alaska Native women, and women who identified as Other. **Finally**, while the majority of ORs were not statistically significant across all groups of women, there were a few associations between specific ACEs and PTD that differed across race subgroups, specifically for White women and Black women. Specifically, physical abuse was associated with lower adjusted odds of PTD among White women (OR 0.7, 95% CI 0.5-1.0) while sexual abuse was associated with lower adjusted odds of PTD among Black women (OR 0.3, 95% CI 0.1-0.9).

- **Aim 2. First**, two underlying subgroups (i.e., classes) of women were identified, where latent class 1 was characterized by a lower probability of experiencing ACEs (the ‘low ACEs’ class) and latent class 2 was characterized by a higher probability of experiencing ACEs (the ‘high ACEs’ class). The high ACEs class was distinguished by significantly higher probabilities of emotional abuse, physical abuse, sexual abuse, and foster care placement ($p < .05$, respectively). **Second**, I found that neither race nor SES predicted latent class membership (all OR 95% confidence intervals encompassed the null value of 1.0 for all race and SES subgroups). **Third**, I found that latent class membership did not predict PTD ($X^2=0$, $p > .05$). **Fourth**, while the majority of the race and SES stratified analyses were not statistically significant, I found that the relationship between class membership and PTD differed significantly for women who identified as Other and for low SES women, but not for other subgroups of women.
- **Aim 3. First**, I found that the mean levels of religiosity, spirituality, and R/S did not differ significantly between the low ACEs class and the high ACEs class ($X^2=.001$,

$p > .05$; $X^2 = .85$, $p > .05$; $X^2 = .03$, $p > .05$, respectively). **Secondly**, I found that religiosity, spirituality, and R/S significantly modified the relationship between latent class membership and odds of PTD in both unadjusted and adjusted models (all p -values $< .05$, respectively). **Third**, while religiosity, spirituality, and R/S did not moderate the association between latent class membership and PTD, respectively, for the majority of race and SES subgroups, a few significant associations were found among White women, Pacific Islander women, women who identified as Other, middle SES, and high SES women. **Fourth**, high levels of religiosity, spirituality, and R/S significantly increased the odds of PTD among women in the high ACEs class compared to women in the low ACEs class (OR 3.4, 95% CI 1.2-9.7; OR 3.4, 95% CI 1.4-8.4; OR 3.6, 95% CI 1.1-12.2, respectively), suggesting that religiosity, spirituality, and R/S are not protective for women who experience high levels of ACEs but may be protective at lower levels of ACEs.

7.2 Comparison of Findings with the Literature and Contextualization within Theory

7.2.1 Assessment of Findings Pertaining to the Latent Classes and Association with PTD

To my knowledge, this is the first study in the literature to apply latent class methods to assess patterns of adverse childhood experiences (ACEs) and their relationship to PTD using a large, longitudinal dataset of women. In Chapter 4, I discovered two latent classes of women—one characterized by a lower probability of experiencing ACEs and the other characterized by a higher probability of experiencing ACEs, and in Chapter 5, I assessed whether the probability of PTD differed significantly across these two classes. My findings can be best interpreted in light of two previous studies (Koning & Ehrenthal, 2019; Deichen Hansen, 2021) which uncovered

different classes of women, characterized by a diverse array of maternal characteristics and risk factors, and evaluated their association with PTD.^{60,205}

7.2.1.1 Comparison of Findings with Koning & Ehrental (2019)⁶⁰

Using hierarchical clustering on principal components analysis, Koning & Ehrental (2019) identified three clusters (designated “stressor landscapes”) of mothers characterized by varying patterns of eleven types of maternal stressful life events occurring in the year prior to childbirth, which they labeled the “Protected Landscape” (characterized by an overall low prevalence of stressful life events), “Illness/Isolated Stressor Landscape” (characterized mostly by illness-related life events), and “Toxic Stressor Landscape” (characterized by an overall high prevalence of acutely stressful life events) using data on n=111,330 women from PRAMS (32 states; 2011-2015).⁶⁰ The protected landscape comprised 62.8% of the sample, the illness/isolated stressor landscape comprised 24.5% of the sample, and the toxic cumulative landscape comprised 12.8% of the sample.⁶⁰ On the other hand, in my study, I uncovered two classes of women, one characterized by a higher risk of ACEs (which comprised 24.9% of the sample), and the other by a lower risk of ACEs (which comprised 75.1%). While the number of identified classes of women varied between my study and that of Koning & Ehrental (2019), the identification of “high risk” and “low risk” groups are consistent themes. Furthermore, the proportion of individuals in the high risk groups compare between the studies.

Next, the authors found that women in the toxic stressor landscape had a higher prevalence of preterm birth (9.6%) compared to those in the Protected (7.5%) and Illness/Isolated landscape (7.8%). However, an assessment of whether these differences were statistically significant was not included, making it difficult to decipher where these percentage differences are meaningfully different and therefore preclude an accurate comparison with my

study, which found no statistically significant differences in the probability of PTD between the low ACEs and high ACEs class (.097 vs. .098, respectively).

Furthermore, the Toxic Stressor Landscape had a higher prevalence of non-Hispanic Black (17.7%) and Hispanic (14.4%) women compared to the Protected Landscape, which included 7.1% non-Hispanic Black women and 11.9% Hispanic women, but a statistical assessment of differences was not provided.⁶⁰ In my study, neither race or SES predicted membership in the low ACEs or high ACEs class. In addition, I found that the relationship between class membership and PTD did not vary significantly within racial or SES subgroups of women, except for women who identified as Other and low SES women. Specifically, in the race-stratified analyses, the relationship between class membership and PTD differed for women who identified as Other, where women who identified as Other in the low ACEs class exhibited a higher probability of PTD than women who identified as Other in the high ACEs class. As we cannot disaggregate the identity of who comprises the women who identified as “Other,” these results are difficult to meaningfully interpret. However, it is possible that this group comprises multiracial women who could not answer the follow-up question of which group of their multiracial identity they best identified with, and therefore decided to select “Other.”

Furthermore, I found that the relationship between latent class membership and PTD differed for low SES women, as low SES women in the low ACEs class experienced a higher probability of PTD compared to low SES women in the high ACEs class. This is unexpected, as low SES women are at higher risk of experiencing more ACEs and PTD.^{3,28,29} It is possible that low SES women in the high ACEs class are more likely to seek help due to the severity of their experiences, or are more likely to be identified as “high risk” during clinical assessments and therefore connected to intervention services that may ultimately reduce their risk, whereas lower

SES women in the low ACEs class may be in denial over whether to seek assistance if they experience fewer – or isolated- incidents of ACEs.

While our findings converged in a few important ways, there are several discrepancies between my study and that of Koning & Ehrental (2019) that must be taken into consideration for a comprehensive comparison of the findings. First, Koning & Ehrental (2019) assessed an exposure during a different time period; they solely examined stressful maternal life events occurring in the year prior to childbirth,⁶⁰ whereas my study examined adverse childhood experiences occurring in the first 18 years of life. Second, there were important differences in analytic sample size and study population. Koning and Ehrental (2019) used PRAMS data on n=111,330 postpartum women across 32 states and New York City from 2011-2015.⁶⁰ While this sample size is considerably larger than ours (n=3,884), the PRAMS dataset was not nationally representative. In addition, Koning & Ehrental (2019) assessed gestational age for preterm birth based on birth records, which allows for greater accuracy of PTD reporting compared to my study, which was only able to assess PTD based on self-reported data. However, studies demonstrate that the accuracy of self-reported data on gestational age compares favorably with data obtained from medical records.^{191,192} In addition, while their analytic sample was limited to only four racial groups (non-Hispanic White, non-Hispanic Black, Hispanic, and American Indian or Alaska Native), mine included seven (White, Black, Hispanic, Asian, Pacific Islander, American Indian or Alaska Native, and Other), which enhances generalizability.

7.2.1.2 Comparison of Findings with Deichen Hansen (2021)²⁰⁵

Deichen Hansen (2021) used latent class analytic methods to identify underlying classes of women defined by patterns of various maternal demographic characteristics, behavioral health indicators, and physical health indicators, and assess whether these classes are associated with

two infant health outcomes, preterm birth (PTB) and low birthweight (LBW) among a sample of $n=4336$ women from Pennsylvania and Illinois using the Pregnancy Risk Assessment Monitoring System (PRAMS).²⁰⁵ Although Deichen Hansen (2021) uncovered three types of latent classes (high risk, moderate risk, and low risk) based on maternal characteristics as well as their risk of PTB and LBW, the theme of discovering subgroups of women with higher and lower risks is consistent with mine.

Furthermore, while overall I did not find major racial and socioeconomic differences between the low ACEs and high ACEs class, Deichen Hansen (2021) found racial and socioeconomic differences between the classes such that the high risk class included a greater proportion of Black women, women who used Medicaid, and had lower annual incomes and educational achievement, compared to women in the low risk and moderate risk classes.²⁰⁵ Furthermore, while I did not find statistically significant differences in the probability of PTD between my classes, Deichen Hansen (2021) found that the high risk class was linked to a significantly higher probability of PTD compared to women in the moderate-risk class ($p<.001$) and the low-risk class ($p<.001$).²⁰⁵

The discrepancies between my findings and those of Deichen Hansen (2021) are likely due to the following reasons. First, while I used data on $n=3,884$ women from a nationally representative Add Health dataset, Deichen Hansen (2021) used PRAMS data on $n=4336$ women from only two states (Pennsylvania and Illinois) between 2012 and 2015, which limits generalizability of their findings. Moreover, the author's analytic sample was restricted to White and Black women only, which further limits generalizability of these findings to other racial subgroups of women, as opposed to my study, which included seven categories of race. In addition, the exposures assessed in these two studies were different: my latent classes were

informed by six ACEs occurring in the first 18 years of life, whereas the latent classes of Deichen Hansen (2021) were informed by maternal demographic characteristics, and behavioral and physical health indicators during pregnancy.²⁰⁵

7.2.2 Assessment of Findings Pertaining to the Role of Religiosity and Spirituality in the Relationship between ACEs and PTD

In Chapter 6, I found evidence of effect modification by religiosity, spirituality, and R/S. Specifically, I found that religiosity, spirituality, and R/S significantly increased the odds of PTD among women in the high ACEs class, but not among women in the low ACEs class, suggesting that for women who experience low levels of ACEs, religiosity, spirituality, and R/S may be protective against the occurrence of PTD, but not for women who experience high levels of ACEs, contrary to my expectations that these variables would offer more protection against PTD for women who experienced high levels of ACEs.

While my study is the first body of work, to my knowledge, to evaluate the relationship between ACEs, religiosity and spirituality, and the health outcome of PTD, here I will interpret my findings in light of a few limited studies in the literature which have assessed the role of religiosity and/or spirituality in protecting against the risk of ACEs on other health outcomes.

My findings concur with Mefford et al. (2021), who found that lower levels of religious/spiritual values/beliefs were associated with higher, not lower, levels of adult life satisfaction in a sample of n=132 adults who experienced childhood loss.²²⁸ My findings that religiosity and spirituality did not protect against PTD among women with high levels of ACEs concur with those of Brockie et al. (2018), who did not find evidence that spirituality moderated the association between ACEs and self-reported physical health, although social support and diabetes-specific support did, in a sample of n=192 American Indian adults with Type 2

diabetes.²²⁹ Furthermore, Homan & Hollenberger (2021) found that religious identification, private religious practice, and religious service attendance did not moderate the association between ACEs and self-rated physical health, but religious support did ($\beta=.03$, $p<.05$) in a sample of $n=4041$ adults from the Midlife in the United States Study.²²⁵ Finally, in a sample of $n=241$ African American adolescents from Texas, Freeny et al. (2021) found that while spirituality was associated with a reduced odds of depression ($OR = 0.927$, 95% CI 0.881-0.976), they did not find evidence that spirituality moderated the association between ACEs and odds of depression ($\beta=1.57$, $p\text{-value}>.05$).²²⁶

While these studies differed from mine in terms of demographics of the study population, sample size, assessment of ACEs, assessment of spirituality and religiosity, and types of health outcomes assessed, the overall pattern of these findings suggest that religiosity and spirituality may not necessarily buffer against the impact of ACEs on various health outcomes. While religiosity and spirituality have been associated with improved health outcomes (Koenig, 2012),²³⁴ and as a buffer against the impacts of stressors on various health outcomes,^{134,135,148,235} few studies have specifically looked at the role of religiosity and spirituality as a buffer against ACEs, and no studies have looked at the specific role of religiosity and spirituality in the association between ACEs and PTSD. The findings from my study suggest that religiosity and spirituality may not operate in a protective manner against PTSD among those who experience the specific life stressor of ACEs, particularly high levels of ACEs. Indeed, my results even suggest that high levels of religiosity and spirituality may have adverse impacts on PTSD among those who experience higher levels of ACEs.

7.2.3 Contextualization of Findings within Theory

This body of work was heavily informed by theoretical frameworks across social epidemiology and developmental psychology, particularly lifecourse theory and psychosocial theory, to understand how biological embedding of adverse childhood experiences during an early developmental life period can impact health outcomes distally in adulthood, and whether there are psychosocial factors that may protect against the impact of early adversity on future health outcomes.

In Chapter 4, my findings showed that specific adverse childhood experiences were not significantly associated with PTD among women overall and for the majority of race and SES subgroups of women. In Chapter 5, I found that the likelihood of PTD was not significantly different between those in the low ACEs class and those in the high ACEs class among women overall. These findings were consistent in the race and SES stratified analyses with the exception of women who identified as Other and low SES women.

While taking into consideration the limitations of my study, which will be summarized in the next section, these results suggest that ACEs may not impact PTD in hypothesized ways, particularly in this sample of women. Specifically, while ACEs occur during sensitive periods in childhood and adolescence which are ripe for biological embedding of adverse (and positive) experiences, the biophysiological impacts may not ultimately impact reproductive health to the degree that would lead to adverse birth outcomes. Alternatively, because pregnancy usually occurs in an early stage of a women's life stage, it is possible that the length in time between occurrence of ACEs and time of pregnancy is not long enough for any adverse physiological effects associated with early life adversity to manifest.³² As the majority of the women in my study had a young maternal age (20-29 years), this is a possibility.

Alternatively, these results may provide support for another explanation in relation to resilience theory. It is possible that the female adolescents who experienced ACEs during childhood subsequently encountered some sort of protective factor(s) that ultimately reduced their risk of future adverse health outcomes, although the protective factor would have to be very strong. Furthermore, because of the gap in time between occurrence of ACEs and the time of occurrence of pregnancy, this could allow for a process of “resetting” of physiological systems that may have been disrupted as a result of the early life stressors.^{35,84,85}

For example, studies by Gunnar et al. (2019) and DePasquale, Donzella, & Gunnar (2019) demonstrated that institutionalized orphans who experienced early life adversity (i.e., a deprived caregiving environment) but were later adopted by nurturing families experienced a normalizing of HPA axis reactivity during later stages of puberty that compared to levels among children who did not experience early life adversity and were not adopted.^{84,85} These findings suggest that the adolescent period may represent an opportunity for “recalibration” of any potential disruptive effects of early life adversity on physiological neuroendocrine systems, particularly if a positive environment is encountered with protective resources that can buffer against the effects of early life adversity on physiological stress systems.

Building upon this framework for the potential of protective resources to buffer the impacts of early life adversity, in Chapter 6 I investigated whether two types of potential protective factors, religiosity and spirituality, play a role in reducing the risk of PTD among individuals with ACEs. I found that high levels of religiosity, spirituality, and R/S (a composite construct of religiosity and spirituality) significantly decreased the odds of PTD among women in the low ACEs class, but not among women in the high ACEs class. These findings suggest that for women who experience low levels of ACEs, religiosity, spirituality, and R/S may be

protective against the occurrence of PTSD, but not for women who experience high levels of ACEs.

The broader implication of these findings is relevant to the conceptual framework of adversity thresholds, whereby an individual's ability to cope with adversity can become undermined after a severe or acute level of adversity has been surpassed.⁷² Furthermore, once a threshold of severe adversity has been exceeded, factors which may otherwise be protective might lose their effectiveness to buffer against the adversity.⁷³ For example, for individuals who experience tremendously high levels of adversity, factors which may otherwise be protective in the context of low to moderate levels of adversity, may lose their ability to confer protection, due to the severe or prolonged nature of the adversity that undermines the individual's ability to cope.

In addition, some research suggests that not all ACEs are "equal" in their impact on health outcomes.⁶¹ For example, sexual abuse is an ACE that appears to be more detrimental in its health impacts compared to other types of adverse experiences.^{19,61,62} Margerison-Zilko et al. (2016) found that sexual abuse during childhood and adulthood significantly increased the odds of PTSD among women, but not physical abuse.¹⁹ Briggs et al. (2021) found that sexual abuse formed the highest number of synergistic pairings of ACEs, meaning its co-occurrence with another ACE contributed to a greater proportion of the attributable risk for clinical behavioral problems among children and adolescents than the sum of the effects of the ACEs individually.⁶¹ Putnam et al. (2013) similarly identified sexual abuse as the most detrimental ACE on adult psychopathology, particularly for women.⁶² As sexual abuse was a more prevalent characteristic in the high ACEs class, this could play a role in my findings.

Furthermore, while there were no significant differences in mean religiosity and spirituality between women in the high ACEs vs. low ACEs class, religiosity and spirituality may function differently for women who experience high levels of ACEs. For example, in a sample of n=1800 individuals aged 15 years and older in the Czech Republic, Kosarkova et al. (2020) found that both religious (R) and nonreligious (NR) individuals with childhood trauma experiences such as sexual abuse viewed the image of God in a less positive light, exhibiting a lower odds of viewing God as Loving (NR: OR 0.92, 95% CI 0.8-0.98; R: OR 0.86, 95% CI 0.77-0.97) or Forgiving (NR: OR 0.75, 95% CI 0.64-0.89; R: OR 0.82, 95% CI 0.68-0.99).²³⁰

In a nationally representative sample of n=1000 individuals aged 15 and older in the Czech Republic, Janu et al. (2022) found that five types of childhood trauma experiences (emotional abuse, physical abuse, sexual abuse, emotional neglect and physical neglect) were all associated with higher odds of religious and spiritual struggles (i.e., feelings of anger toward God; questioning religion or spirituality).²³¹ While these studies did not specifically differentiate among those who had higher versus lower levels of ACEs, these results suggest that religiosity and spirituality may function differently among those who experience ACEs, particularly higher levels of ACEs. More specifically, as spirituality and religion can lead to feelings of guilt,²³² spirituality and religiosity may not necessarily offer solace for individuals who experience high levels of childhood adversity.

7.3 Summary of Limitations

The findings of this dissertation work must be contextualized within its limitations. First, while I used a diverse, nationally representative dataset, my analytic sample was not nationally representative, as it disproportionately comprised of White women (~70%) and women from middle and high SES backgrounds (~90%). This limits generalizability of my findings to other

racial and SES groups. These groups of women are also at lower risk for PTD, which may have contributed to the overall non-significant findings. Furthermore, as discussed in my findings across Chapters 4-6, due to limited cell sizes for some groups of women, I was unable to derive precise estimates for some race stratified analyses.

In addition, as most of the ACEs were collected when the female adolescents were in adulthood, this could have introduced the potential for retrospective recall bias, particularly underreporting due to stigma, which could have underestimated the true relationship between ACEs and PTD.^{56,57} While Add Health used ACASI/CAPI methods to enhance the reporting accuracy of sensitive information, this possibility cannot be fully ruled out. Furthermore, there is some evidence that women may “minimize or deny” occurrence of ACEs assessed during pregnancy and the postpartum period.²³⁶

Furthermore, women who experience high levels of ACEs may be more likely to 1) decide not to have children¹⁰⁶ 2) have difficulties becoming pregnant due to physical trauma (i.e., physical and/or sexual abuse) or other health conditions as a result of the ACEs (i.e., depression, anxiety); 3) experience a pregnancy loss⁵⁸ or 4) possibly have an abortion as a result of an unintended pregnancy,^{39,111} and therefore may not be captured in my denominator of women who have pregnancies that end in a live birth, resulting in a potential selection bias that can underestimate the association between ACEs and PTD.

Furthermore, I was only able to assess six types of ACEs, which may limit generalizability of findings to other types of ACEs (i.e., parental death or incarceration). Also, ACEs were operationalized as binary variables based on presence or absence of occurrence (yes/no), which does not account for severity or frequency of occurrence. Hypothetically, the

more frequently an adverse experience occurs and the longer it occurs, the opportunity for physiological stress systems to recover or recalibrate may be diminished.

In addition, religiosity and spirituality were captured by one question only. As the literature shows, these constructs are complex and multidimensional, therefore, future studies should incorporate more detailed scales of these constructs. Furthermore, religiosity and spirituality variables were assessed at one time point only. As religiosity and spirituality are fluid constructs, their significance and meaning may wax and wane over an individual's lifecourse. For example, studies show that religiousness tends to decrease in young adulthood but increase in later life.²³³ Religiosity and spirituality were captured in Wave III when the women were young adults, and this may explain the lower overall mean levels of religiosity and spirituality in the sample.

Finally, while my study incorporated a lifecourse study design, it does not account for macro-level factors at the socioecological level^{216,237} that may contribute to, and perpetuate, disparities in both ACEs and PTD, such as institutional racism, and unequal access to education, health care, insurance coverage, and comprehensive mental health and counseling services. Furthermore, it is important to acknowledge that factors at the individual (i.e., religiosity) interpersonal (i.e., social support), and community levels (i.e., access to recreational areas) are interrelated, and macro-level factors such as institutional racism have the ability to impact individuals at all socio-ecological levels.

7.4 Summary of Strengths

Despite the limitations, this body of work represents a significant contribution to the literature in several domains. Specifically, this is the first study in the literature to evaluate the relationship between ACEs, religiosity and spirituality, and PTD. Furthermore, this is the first

study in the perinatal literature to apply latent class methods to evaluate patterns of ACEs and their association to PTD and whether this relationship differs by race and socioeconomic status. I used latent class modeling as a person-based methodological approach to determine whether co-occurring patterns of ACEs are important predictors for PTD, instead of the common methodological approach of using a sum or count of ACEs, which fails to account for potential interactions between multiple risk factors for PTD among women who give birth.

My dissertation's application of a racial and socioeconomic health equity framework is an important contribution as it enhances understanding of whether specific groups of women may be at greater risk of experiencing PTD as a result of early adverse life experiences, which is important for clinical intervention. Furthermore, the development of my research aims was heavily informed by interdisciplinary theoretical frameworks, particularly across developmental psychology and lifecourse epidemiology, which contributes to a paradigm shift in thinking about the importance of women's health not just from the perspective of the prenatal period, but from the preconception period as well, building off of foundational literature that underscores the importance of the preconception period as a "critical means of identifying, managing, and treating risk factors originating prior to pregnancy that can harm fetal development" (Margerison-Zilko et al., 2020, p. 1).²³⁸

These frameworks influenced my study design and selection of Add Health as the dataset for my dissertation, as it uniquely captures detailed information on life events occurring during childhood and adolescence and follows these individuals for 20 years to capture health outcomes during adulthood, including pregnancy and birth outcomes. This dataset allowed me to tailor my research questions to encompass a lifecourse study design.^{37,80}

7.5 Public Health Implications

The findings of my dissertation carry several implications for public health practice and clinical intervention. First, as the evidence for an association between ACEs and PTD was weak, this could suggest that ACEs do not impact PTD in hypothesized ways. To this point, the literature on stressor hypotheses and preterm delivery has been mixed.⁵ It is possible that the biological embedding of ACEs during childhood and adolescence may not impact reproductive outcomes to the degree that they impact cases of chronic disease, risky health behaviors, and mental health conditions. Furthermore, as women have been carrying pregnancies for millennia, the human body has most likely evolved some inherently resilient adaptive mechanisms to buffer against environmental stressors for the purposes of survival.

Alternatively, these findings may suggest the involvement of a very strong protective factor, or factors, that buffer against the effects of early life adversity on preterm delivery. Furthermore, the length of time between occurrence of ACEs during childhood and occurrence of pregnancy may allow for a process of resetting or recalibration of any physiological disruptions influenced by ACEs during late adolescence particularly in the presence of protective factors (i.e., nurturing caretaking environment),^{84,85} therefore reducing the risk of future adverse birth outcomes. In addition, the length of time between occurrence of ACEs and occurrence of pregnancy may not be long enough for any adverse physiological effects triggered by early life adversity to manifest,³² particularly for women who become pregnant at a younger age.

The length of time between ACE exposure and time of pregnancy offers a unique opportunity period for clinical intervention and counseling services to help “identify, affirm, and build” the repertoire of protective factors among children exposed to ACEs, especially during the peripubertal period (Walsh, 2016, p. 140).⁴³ Underscoring the importance of this period and the

potential for intervention, Gunnar states, "...intervention efforts to improve outcomes for children who have experienced early life adversity should include a focus on the prepubertal and peripubertal period in order to maximize their impact on recalibrating systems like the HPA axis" (Gunnar et al., 2019, p. 23984).⁸⁵ Health assessments at primary care visits and school-based assessments may identify children with a history of ACE exposures and connect them to appropriate community-based services to promote a trajectory of positive health outcomes.²³⁹

Furthermore, I found that religiosity, spirituality, and R/S significantly reduced the odds of PTD for women in the low ACEs class, but not for those in the high ACEs class. As previously discussed in Section 7.2.3, religiosity and spirituality may not be "protective factors" for women with acute, high levels of ACEs. This suggests the possibility of a type of threshold effect, whereby specific factors may lose their potential to serve as protective resources in the face of acute and prolonged adversity.⁷³ This underscores the concept that not all factors may be considered protective for all groups of women, which has important implications for clinical interventions. For example, preconception counseling services and prenatal assessments offer the opportunity to conduct a thorough life history assessment to identify early life adversity risk factors and needs assessments that identify and assesses the availability of an individual's protective factors that can help promote resilience, such as the Walsh Family Resilience Questionnaire.⁴³

7.6 Directions for Future Research

The findings of this dissertation inform several directions for future research. First and foremost, future research on this important topic needs to include larger sample sizes for non-White groups of women to enhance generalizability and allow for a better assessment of whether these

relationships between ACEs, PTD, and protective factors operate differently among heterogeneous groups of women by race and SES.

Secondly, while I focused exclusively on preterm birth in this dissertation study, life stressors such as ACEs may influence various birth outcomes differently. Therefore, future research should assess whether adverse childhood experiences impact other adverse birth outcomes such as low birthweight (i.e., indicators of fetal growth restriction) differently.

Furthermore, as a next step, it will be important to evaluate whether the prevalence of ACEs among women who do not have a pregnancy that ends in a live birth differs significantly from those who do have a live birth pregnancy to assess the potential for selection bias. In addition, it will be interesting to assess the prevalence of women who experience ACEs yet decide to have children compared to women who experience ACEs but decide *not* to have children.

Furthermore, future research should incorporate assessments of ACE severity and frequency, not only binary indicators of occurrence, which may result in having a different association with PTD. As a next step, I plan to incorporate frequency of occurrence into the latent class models to assess whether the makeup of the latent classes change, and whether these potentially alternative classes have a different association with PTD. As my dissertation only captured six ACEs, future research should include additional categories of ACEs (i.e., parental incarceration, parental loss) to maximize generalizability of findings. In addition, the findings of Chapter 6 invoke the question of whether other potentially protective factors similarly exhibit a threshold effect, whereby they offer protection in the context of lower to moderate levels of adversity, but not for severe levels of adversity.

Furthermore, as previously discussed, because the majority of women in my study had a young maternal age at birth (20-29 years), the length in time between occurrence of ACEs and time of pregnancy may not be long enough for any potential adverse health effects associated with early life adversity to manifest. Therefore, future studies could look at the relationship between ACEs and PTD stratified by maternal age to see if the relationship differs for women of advanced maternal age (>35 years), who would have a longer time between ACE occurrence and time of pregnancy.

7.7 Conclusion

My dissertation uses data on n=3,884 women from the National Longitudinal Study of Adolescent to Adult Health to conduct the first study in the literature to investigate the relationship between adverse childhood experiences, PTD, and two potential protective factors, religiosity and spirituality, among women who have pregnancies and assess whether these relationships operate differently across race and SES. This body of work contributes to the literature through an improved understanding of whether early life adversity during the preconception period may impact future birth outcomes in women, and the role of religiosity and spirituality to buffer against PTD among women with varying levels of ACEs. My dissertation did not find strong evidence that specific individual ACEs, or latent classes of ACEs, were associated with increased odds of PTD among women overall, and within race and SES subgroups. In future research, it will be important for the field to expand beyond individual-level stressors and focus on macro-level factors that may be contributing to disparities in PTD such as nationwide policies like the ACA, health insurance coverage, environmental factors, and importantly, institutional, and structural racism. Although protective factors are important to health, “....without sound policies, individual attributes, involved families, and supportive

communities will have limited effectiveness” (Seccombe, 2002, p. 389).²⁴⁰ Furthermore, my results suggest that protective factors like religiosity and spirituality may not operate the same for all groups of women. This underscores the concept that not all factors may be considered protective for all groups of women, and therefore context is critical for assessing whether a factor may be beneficial for intervention purposes. The findings of this dissertation can provide useful insight for the conduction of needs assessments in the context of counseling and development of tailored clinical interventions that aim to help women “identify, affirm, and build” (Walsh, 2016, p. 140)⁴³ their repertoire of specific and unique protective factors that can be mobilized to promote optimal preconception and pregnancy health in spite of experiences of early life adversity.

BIBLIOGRAPHY

1. Martin JA, Hamilton BE, Sutton PD, Ventura SJ, Mathews TJ, Osterman MJ. Births: Final Data for 2008. *Natl Vital Stat Reports*. 2010;59(1):1-72.
2. Osterman MJ, Hamilton BE, Martin JA, Driscoll AK, Valenzuela CP. Births: Final Data for 2020. *Natl Vital Stat Reports*. 2022;70(17):1-50.
3. Blumenshine P, Egerter S, Barclay CJ, Cubbin C, Braveman PA. Socioeconomic disparities in adverse birth outcomes: A systematic review. *Am J Prev Med*. 2010;39(3):263-272. doi:10.1016/j.amepre.2010.05.012
4. Kramer MS, Séguin L, Lydon J, Goulet L. Socio-economic disparities in pregnancy outcome: Why do the poor fare so poorly? *Paediatr Perinat Epidemiol*. 2000;14(3):194-210. doi:10.1046/j.1365-3016.2000.00266.x
5. Behrman RE, Butler AS, Institute of Medicine (U.S.). Committee on Understanding Premature Birth and Assuring Healthy Outcomes. *Preterm Birth : Causes, Consequences, and Prevention*. National Academies Press; 2007.
6. Ionio C, Colombo C, Brazzoduro V, et al. Mothers and fathers in nicu: The impact of preterm birth on parental distress. *Eur J Psychol*. 2016;12(4):604-621. doi:10.5964/ejop.v12i4.1093
7. Carson C, Redshaw M, Gray R, Quigley MA. Risk of psychological distress in parents of preterm children in the first year: Evidence from the UK Millennium Cohort Study. *BMJ Open*. 2015;5(12). doi:10.1136/bmjopen-2015-007942
8. Centers for Disease Control and Prevention (CDC). Preterm birth. <https://www.cdc.gov/reproductivehealth/maternalinfanthealth/pretermbirth.htm>. Published 2021. Accessed August 7, 2022.
9. de Paula Eduardo JAF, de Rezende MG, Menezes PR, Del-Ben CM. Preterm birth as a risk factor for postpartum depression: A systematic review and meta-analysis. *J Affect Disord*. 2019;259(April):392-403. doi:10.1016/j.jad.2019.08.069
10. Trumello C, Candelori C, Cofini M, et al. Mothers' depression, anxiety, and mental representations after preterm birth: A study during the infant's hospitalization in a neonatal intensive care unit. *Front Public Heal*. 2018;6(DEC):1-9. doi:10.3389/fpubh.2018.00359
11. Lu MC, Halfon N. Racial and ethnic disparities in birth outcomes: a life-course perspective. *Matern Child Health J*. 2003;7(1):13-30. doi:10.1023/A:1022537516969

12. Dunkel Schetter C, Schafer P, Lanzi RG, Clark-kauffman E, Raju TNK, Marianne M. Shedding Light on the Mechanisms Underlying Health Disparities Through Community Participatory Methods : The Stress Pathway. *Perspect Psychol Sci.* 2013;8(6):613-633. doi:10.1177/1745691613506016
13. Geronimus AT, Hicken M, Keene D, Bound J. “Weathering” and age patterns of allostatic load scores among blacks and whites in the United States. *Am J Public Health.* 2006;96(5):826-833. doi:10.2105/AJPH.2004.060749
14. Strutz KL, Hogan VK, Siega-Riz AM, Suchindran CM, Halpern CT, Hussey JM. Preconception stress, birth weight, and birth weight disparities among us women. *Am J Public Health.* 2014;104(8):125-132. doi:10.2105/AJPH.2014.301904
15. Christian LM. At the forefront of psychoneuroimmunology in pregnancy: Implications for racial disparities in birth outcomes PART 1: Behavioral risks factors. *Neurosci Biobehav Rev.* 2019;1-8. doi:10.1016/j.neubiorev.2019.04.009
16. Petrucci K, Davis J, Berman T. Adverse childhood experiences and associated health outcomes: A systematic review and meta-analysis. *Child Abus Negl.* 2019;97(August):104127. doi:10.1016/j.chiabu.2019.104127
17. Felitti VJ, Anda RF, Nordenberg DF, et al. Relationship of Childhood abuse and household dysfunction to many of the leading causes of deaths in adults. *Am J Prev Med.* 1998;14(4):245-258.
18. Sulaiman S, Premji SS, Tavangar F, et al. Total Adverse Childhood Experiences and Preterm Birth: A Systematic Review. *Matern Child Health J.* 2021;25(10):1581-1594. doi:10.1007/s10995-021-03176-6
19. Margerison-Zilko CE, Strutz KL, Li Y, Holzman C. Stressors Across the Life-Course and Preterm Delivery: Evidence From a Pregnancy Cohort. *Matern Child Health J.* 2017;21(3):648-658. doi:10.1007/s10995-016-2151-5
20. Olsen JM. Integrative review of pregnancy health risks and outcomes associated with adverse childhood experiences. *JOGNN - J Obstet Gynecol Neonatal Nurs.* 2018;47:783-794.
21. Witt WP, Cheng ER, Wisk LE, et al. Preterm birth in the United States: The impact of stressful life events prior to conception and maternal age. *Am J Public Health.* 2014;104(SUPPL. 1):73-80. doi:10.2105/AJPH.2013.301688
22. Hamilton BE, Martin JA, Osterman MJ. *Births: Provisional Data for 2021.* Hyattsville, MD; 2022.

23. Martin JA, Hamilton BE, Osterman MJ, Drisco. Births: Final Data for 2018. *Natl Vital Stat Reports*. 2019;68(13):1-47.
24. Rossen LM, Schoendorf KC. Trends in racial and ethnic disparities in infant mortality rates in the united states, 1989-2006. *Am J Public Health*. 2014;104(8):1549-1556. doi:10.2105/AJPH.2013.301272
25. Parker JD, Schoendorf KC, Kiely JL. Associations between measures of socioeconomic status and low birth weight, small for gestational age, and premature delivery in the United States. *Ann Epidemiol*. 1994;4(4):271-278. doi:10.1016/1047-2797(94)90082-5
26. Braveman PA, Dominguez TP, Burke W, et al. Explaining the Black-White Disparity in Preterm Birth: A Consensus Statement from a Multidisciplinary Scientific Work Group Convened by the March of Dimes. *Front Reprod Heal*. 2021;3:1-24.
27. Slaughter-Acey JC, Sealy-Jefferson S, Helmkamp L, et al. Racism in the form of micro aggressions and the risk of preterm birth among black women. *Ann Epidemiol*. 2016;26:7-13.
28. Giano Z, Wheeler DL, Hubach RD. The frequencies and disparities of adverse childhood experiences in the U.S. *BMC Public Health*. 2020;20(1):1-12. doi:10.1186/s12889-020-09411-z
29. Walsh D, McCartney G, Smith M, Armour G. Relationship between childhood socioeconomic position and adverse childhood experiences (ACEs): A systematic review. *J Epidemiol Community Health*. 2019;73(12):1087-1093. doi:10.1136/jech-2019-212738
30. Merrick MT, Ford DC, Ports KA, Guinn AS, Chen J. Vital signs: estimated proportion of adult health problems attributable to adverse childhood experiences and implications for prevention-25 states, 2015-2017. *Morb Mortal Wkly Rep*. 2019;68(44):999-1005.
31. Sacks V, Murphey D. *The Prevalence of Adverse Childhood Experiences, Nationally, by State, and by Race or Ethnicity*. Bethesda, Maryland; 2018. <https://www.childtrends.org/publications/prevalence-adverse-childhood-experiences-nationally-state-race-ethnicity>.
32. Ehrlich K, Miller G, Chen E. Childhood adversity and adult physical health. In: Cicchetti D, ed. *Developmental Psychopathology, Volume 4: Risk, Resilience, and Intervention*. 3rd ed. John Wiley & Sons; 2016:1-73.
33. Hostinar CE, Gunnar MR. The Developmental Effects of Early Life Stress: An Overview of Current Theoretical Frameworks. *Curr Dir Psychol Sci*. 2013;22(5):400-406. doi:10.1177/0963721413488889

34. Danese A, McEwen BS. Adverse childhood experiences, allostasis, allostatic load, and age-related disease. *Physiol Behav.* 2012;106(1):29-39. doi:10.1016/j.physbeh.2011.08.019
35. Gunnar MR. Chapter 6 Stress Effects on the Developing Brain. In: Romer D, Walker EF, eds. *Adolescent Psychopathology and the Developing Brain: Integrating Brain and Prevention Science.* Oxford University Press; 2007:127-147.
36. Cicchetti D, Walker EF. Editorial: Stress and development: Biological and psychological consequences. *Dev Psychopathol.* 2001;13(3):413-418. doi:10.1017/S0954579401003017
37. Misra DP, Guyer B, Allston A. Integrated perinatal health framework: A multiple determinants model with a life span approach. *Am J Prev Med.* 2003;25(1):65-75. doi:10.1016/S0749-3797(03)00090-4
38. Frankenberger DJ, Clements-nolle K, Yang W. The Association between Adverse Childhood Experiences and Alcohol Use during Pregnancy in a Representative Sample of Adult Women. *Women's Heal Issues.* 2015;25(6):688-695. doi:10.1016/j.whi.2015.06.007
39. Hall KS, Beauregard JL, Rentmeester ST, Livingston M, Harris KM. Adverse life experiences and risk of unintended pregnancy in adolescence and early adulthood: Implications for toxic stress and reproductive health. *SSM - Popul Heal.* 2019;7(June 2018):1-10. doi:10.1016/j.ssmph.2018.100344
40. Young-Wolff KC, Alabaster A, McCaw B, et al. Adverse childhood experiences and mental and behavioral health conditions during pregnancy: The role of resilience. *J Women's Heal.* 2019;28(4):452-461. doi:10.1089/jwh.2018.7108
41. Bublitz M, Parade S, Stroud L. The effects of childhood sexual abuse on cortisol trajectories in pregnancy are moderated by current family functioning. *Biol Psychol.* 2014;103:152-157.
42. Thomas JC, Magel C, Tomfohr-Madsen L, et al. Adverse childhood experiences and HPA axis function in pregnant women. *Horm Behav.* 2018;102:10-22.
43. Walsh F. *Strengthening Family Resilience.* Third. New York: The Guilford Press; 2016.
44. Blencowe H, Cousens S, Oestergaard MZ, et al. National, regional, and worldwide estimates of preterm birth rates in the year 2010 with time trends since 1990 for selected countries: A systematic analysis and implications. *Lancet.* 2012;379(9832):2162-2172. doi:10.1016/S0140-6736(12)60820-4
45. What are the risk factors for preterm labor and birth? | NICHD - Eunice Kennedy Shriver National Institute of Child Health and Human Development.

https://www.nichd.nih.gov/health/topics/preterm/conditioninfo/who_risk. Published 2017. Accessed December 1, 2018.

46. Kramer MS, Wilkins R, Goulet L, et al. Investigating socio-economic disparities in preterm birth: Evidence for selective study participation and selection bias. *Paediatr Perinat Epidemiol*. 2009;23(4):301-309. doi:10.1111/j.1365-3016.2009.01042.x
47. Pickett KE, Ahern JE, Selvin S, Abrams B. Neighborhood socioeconomic status, maternal race and preterm delivery: A case-control study. *Ann Epidemiol*. 2002;12(6):410-418. doi:10.1016/S1047-2797(01)00249-6
48. Kramer MR, Hogue CR. What causes racial disparities in very preterm birth? a biosocial perspective. *Epidemiol Rev*. 2009;31(1):84-98. doi:10.1093/ajerev/mxp003
49. Luthar SS. Resilience in development: a synthesis of research across five decades. In: Cicchetti D, Cohen D, eds. *Developmental Psychopathology: Risk, Disorder, and Adaptation*. 2nd ed. New York: Wiley; 2006.
50. Danese A, Moffitt TE, Harrington HL, et al. Adverse childhood experiences and adult risk factors for age-related disease: Depression, inflammation, and clustering of metabolic risk markers. *Arch Pediatr Adolesc Med*. 2009;163(12):1135-1143. doi:10.1001/archpediatrics.2009.214
51. Berens AE, Jensen SKG, Nelson CA. Biological embedding of childhood adversity: From physiological mechanisms to clinical implications. *BMC Med*. 2017;15(1):1-12. doi:10.1186/s12916-017-0895-4
52. Centers for Disease Control and Prevention (CDC). Adverse Childhood Experiences (ACEs). CDC Vital Signs. <https://www.cdc.gov/vitalsigns/aces/index.html>. Published 2021. Accessed August 7, 2022.
53. Centers for Disease Control and Prevention (CDC). Fast Facts: Preventing Childhood Adverse Experiences. <https://www.cdc.gov/violenceprevention/aces/fastfact.html>. Published 2022. Accessed August 13, 2022.
54. Centers for Disease Control and Prevention (CDC); Kaiser Permanente. About the CDC-Kaiser ACE Study. <https://www.cdc.gov/violenceprevention/aces/about.html>. Published 2021. Accessed August 10, 2022.
55. Cronholm PF, Forke CM, Wade R, et al. Adverse Childhood Experiences: Expanding the Concept of Adversity. *Am J Prev Med*. 2015;49(3):354-361. doi:10.1016/j.amepre.2015.02.001
56. Marcus JL, Bernstein KT, Philip S, Klausner JD. Evidence of underreporting of adverse

- childhood experiences, san francisco municipal STD clinic, 2007. *Sex Transm Dis*. 2009;36(7):422-424. doi:10.1097/OLQ.0b013e31819d72ad
57. Dill DL, Chu JA, Grob MC, Eisen S V. The reliability of abuse history reports: A comparison of two inquiry formats. *Compr Psychiatry*. 1991;32(2):166-169. doi:10.1016/0010-440X(91)90009-2
 58. Mersky JP, Lee CP. Adverse childhood experiences and poor birth outcomes in a diverse, low-income sample. *BMC Pregnancy Childbirth*. 2019;19(1):1-7. doi:10.1186/s12884-019-2560-8
 59. Patwardhan I, Hurley KD, Thompson RW, Mason WA, Ringle JL. Child maltreatment as a function of cumulative family risk: Findings from the intensive family preservation program. *Child Abus Negl*. 2017;70(May):92-99. doi:10.1016/j.chiabu.2017.06.010
 60. Koning SM, Ehrental DB. Stressor landscapes, birth weight, and prematurity at the intersection of race and income: Elucidating birth contexts through patterned life events. *SSM - Popul Heal*. 2019;8(100460):1-10. doi:10.1016/j.ssmph.2019.100460
 61. Briggs EC, Amaya-Jackson L, Putnam KT, Putnam FW. All Adverse Childhood Experiences Are Not Equal: The Contribution of Synergy to Adverse Childhood Experience Scores. *Am Psychol*. 2021;76(2):243-252. doi:10.1037/amp0000768
 62. Putnam K, Harris W, Putnam F. Synergistic childhood adversities and complex adult psychopathology. *J Trauma Stress*. 2013;26(4):435-442.
 63. Putnam F, Amaya-Jackson L, Putnam K, Briggs E. Synergistic adversities and behavioral problems in traumatized children and adolescents. *Child Abus Negl*. 2020;104(492):1-10.
 64. Nurius P, Logan-Greene P, Green S. ACEs within a social disadvantage framework: distinguishing, unique, cumulative, and moderated contributions to adult mental health. *J Prev Interv Community*. 2012;40(4):278-290.
 65. Berkman L, Kawachi I, Glymour M. *Social Epidemiology*. 2nd ed. New York: Oxford University Press; 2014.
 66. Marmot M, Wilkinson RG, eds. *Social Determinants of Health*. 2nd ed. Oxford University Press; 2005.
 67. Social Determinants of Health. U.S. Department of Health and Human Services, Office of Disease Prevention and Health Promotion. Healthy People 2030. <https://health.gov/healthypeople/priority-areas/social-determinants-health>. Published 2022. Accessed August 7, 2022.

68. Centers for Disease Control and Prevention (CDC). About Social Determinants of Health (SDOH). <https://www.cdc.gov/socialdeterminants/about.html>. Published 2021. Accessed August 7, 2022.
69. Braveman P. What are health disparities and health equity? we need to be clear. *Public Health Rep.* 2014;129(SUPPL. 2):5-8. doi:10.1177/00333549141291s203
70. Krieger N. Theories for social epidemiology in the 21st century: An ecosocial perspective. *Int J Epidemiol.* 2001;30(4):668-677. doi:10.1093/ije/30.4.668
71. Luthar SS, Cicchetti D, Becker B. The Construct of Resilience : A Critical Evaluation and Guidelines for Future Work. *Soc Res Child Dev.* 2000;71(3):543-562.
72. Masten AS. *Ordinary Magic*. New York: The Guilford Press; 2014.
73. Masten AS, Best KM, Garmezy N. Resilience and development contributions from the study of children who overcome adversity. *Dev Psychopathol.* 1990;2:425-444.
74. Solivan AE, Wallace ME, Kaplan KC, Harville EW. Use of a Resiliency Framework to Examine Pregnancy and Birth Outcomes Among Adolescents : A Qualitative Study. *Fam Syst Heal.* 2015;33(4):349-355.
75. Kramer MS, Lydon J, Séguin L, et al. Stress pathways to spontaneous preterm birth: The role of stressors, psychological distress, and stress hormones. *Am J Epidemiol.* 2009;169(11):1319-1326. doi:10.1093/aje/kwp061
76. Wakeel F, Wisk LE, Gee R, Chao SM, Witt WP. The balance between stress and personal capital during pregnancy and the relationship with adverse obstetric outcomes: findings from the 2007 Los Angeles Mommy and Baby (LAMB) study. *Arch Womens Ment Heal.* 2013;16:435-451.
77. Appleton AA, Kiley K, Holdsworth EA, Schell LM. Social Support During Pregnancy Modifies the Association Between Maternal Adverse Childhood Experiences and Infant Birth Size. *Matern Child Health J.* 2019;23(3):408-415. doi:10.1007/s10995-018-02706-z
78. Hetherington E, Doktorchik C, Premji SS, McDonald SW, Tough SC, Sauve RS. Preterm Birth and Social Support during Pregnancy: A Systematic Review and Meta-Analysis. *Paediatr Perinat Epidemiol.* 2015;29(6):523-535. doi:10.1111/ppe.12225
79. Adler NE, Stewart J. Health disparities across the lifespan: Meaning, methods, and mechanisms. *Ann N Y Acad Sci.* 2010;1186:5-23. doi:10.1111/j.1749-6632.2009.05337.x
80. Pollitt RA, Rose KM, Kaufman JS. Evaluating the evidence for models of life course socioeconomic factors and cardiovascular outcomes: A systematic review. *BMC Public*

Health. 2005;5:1-13. doi:10.1186/1471-2458-5-7

81. Zamani-Hank Y, Margerison CE, Talge NM, Holzman C. Differences in Psychosocial Protective Factors by Race/Ethnicity and Socioeconomic Status and Their Relationship to Preterm Delivery. *Women's Heal Reports*. 2022;3(1):243-255. doi:10.1089/whr.2021.0049
82. Cicchetti D. Neural plasticity, sensitive periods, and psychopathology. *Dev Psychopathol*. 2015;27(2):319-320. doi:10.1017/S0954579415000012
83. Nelson CA, Gabard-Durnam LJ. Early Adversity and Critical Periods: Neurodevelopmental Consequences of Violating the Expectable Environment. *Trends Neurosci*. 2020;43(3):133-143. doi:10.1016/j.tins.2020.01.002
84. DePasquale CE, Donzella B, Gunnar MR. Pubertal recalibration of cortisol reactivity following early life stress: a cross-sectional analysis. *J Child Psychol Psychiatry Allied Discip*. 2019;60(5):566-575. doi:10.1111/jcpp.12992
85. Gunnar MR, DePasquale CE, Reid BM, Donzella B. Pubertal stress recalibration reverses the effects of early life stress in postinstitutionalized children. *Proc Natl Acad Sci U S A*. 2019;116(48):23984-23988. doi:10.1073/pnas.1909699116
86. Schneiderman N, Ironson G, Siegel SD. Stress and health: Psychological, behavioral, and biological determinants. *Annu Rev Clin Psychol*. 2005;1(Lacey 1967):607-628. doi:10.1146/annurev.clinpsy.1.102803.144141
87. Carrion VG, Weems CF, Ray RD, Glaser B, Hessel D, Reiss AL. Diurnal salivary cortisol in pediatric posttraumatic stress disorder. *Biol Psychiatry*. 2002;51(7):575-582. doi:10.1016/S0006-3223(01)01310-5
88. De Bellis M, Baum A, Birmaher B, Keshavan M, CH E. Developmental traumatology, Part 1: Biological stress systems. *Biol Psychiatry*. 1999;45(10):1259-1270.
89. Bowlby J. *Attachment and Loss, Volume 1*. New York: Basic Books; 1969.
90. Dettling A, Gunnar M, Donzella B. Cortisol levels of young children in full-day childcare centers: relations with age and temperament. *Psychoneuroendocrinology*. 1999;24:505-518.
91. Ahnert L, Gunnar M, Lamb M, Barthel M. Transition to childcare: associations of infant-mother attachment, infant negative emotion, and cortisol elevations. *Child Dev*. 2004;75:639-650.
92. Dunkel Schetter C. Psychological Science on Pregnancy: Stress Processes,

- Biopsychosocial Models, and Emerging Research Issues. *Annu Rev Psychol.* 2011;62:531-558. doi:10.1146/annurev.psych.031809.130727
93. Shapiro GD, Fraser WD, Frasch MG, Séguin JR. Review article Psychosocial stress in pregnancy and preterm birth : associations and mechanisms. 2013;41(6):631-645. doi:10.1515/jpm-2012-0295
 94. Wadhwa PD, Entringer S, Buss C, Lu MC. The contribution of maternal stress to preterm birth: Issues and considerations. *Clin Perinatol.* 2011;38(3):351-384. doi:10.1016/j.clp.2011.06.007
 95. Wadhwa PD, Sandman CA, Porto M, Dunkel-Schetter C, Garite TJ. The association between prenatal stress and infant birth weight and gestational age at birth: A prospective investigation. *Am J Obstet Gynecol.* 1993;169(4):858-865. doi:10.1016/0002-9378(93)90016-C
 96. Giurgescu C. Are maternal cortisol levels related to preterm birth? *JOGNN - J Obstet Gynecol Neonatal Nurs.* 2009;38(4):377-390. doi:10.1111/j.1552-6909.2009.01034.x
 97. Hobel CJ, Dunkel-Schetter C, Roesch SC, Castro LC, Arora CP. Maternal plasma corticotropin-releasing hormone associated with stress at 20 weeks' gestation in pregnancies ending in preterm delivery. *Am J Obstet Gynecol.* 1999;180(1):S257-S263. doi:10.1016/S0002-9378(99)70712-X
 98. Phocas I, Sarandakou A, Rizos D. Maternal serum total cortisol levels in normal and pathologic pregnancies. *Int J Gynecol Obstet.* 1990;31(1):3-8. doi:10.1016/0020-7292(90)90173-I
 99. Mazor M, Chaim W, Hershkowitz R, Levy J, Leiberman J, Glezerman M. Association between preterm birth and increased maternal plasma cortisol concentrations. *Obs Gynecol.* 1994;84(4):521-524.
 100. Sandman CA, Glynn L, Schetter CD, et al. Elevated maternal cortisol early in pregnancy predicts third trimester levels of placental corticotropin releasing hormone (CRH): Priming the placental clock. *Peptides.* 2006;27(6):1457-1463. doi:10.1016/j.peptides.2005.10.002
 101. Himes KP, Simhan HN. Plasma corticotropin-releasing hormone and cortisol concentrations and perceived stress among pregnant women with preterm and term birth. *Am J Perinatol.* 2011;28(6):443-448. doi:https://dx.doi.org/10.1055/s-0030-1270119
 102. Hoffman MC, Mazzoni SE, Wagner BD, Laudenslager ML, Ross RG. Measures of Maternal Stress and Mood in Relation to Preterm Birth. *Obstet Gynecol.* 2016;127(3):545-552. doi:10.1097/AOG.0000000000001287

103. Erickson K, Thorsen P, Chrousos G, Grigoriadis DE, McGregor J. Preterm Birth : Associated Neuroendocrine , Medical , and Behavioral Risk Factors. *J Clin Endocrinol Metab.* 2001;86(6):2544-2552.
104. Kramer MS, Lydon J, Goulet L, et al. Maternal stress/distress, hormonal pathways and spontaneous preterm birth. *Paediatr Perinat Epidemiol.* 2013;27(3):237-246. doi:10.1111/ppe.12042
105. Bandoli G, Jelliffe-Pawłowski LL, Feuer SK, et al. Second trimester serum cortisol and preterm birth: an analysis by timing and subtype. *J Perinatol.* 2018:1-9. doi:10.1038/s41372-018-0128-5
106. Belsky J, Steinberg L, Draper P. Childhood Experience, Interpersonal Development, and Reproductive Strategy: An Evolutionary Theory of Socialization. *Child Dev.* 1991;62(4):647-670. doi:10.1111/j.1467-8624.1991.tb01558.x
107. Moffitt TE, Caspi A, Belsky J, Silva PA. Childhood Experience and the Onset of Menarche: A Test of a Sociobiological Model. *Child Dev.* 1992;63(1):47-58. doi:10.1111/j.1467-8624.1992.tb03594.x
108. Jorm AF, Christensen H, Rodgers B, Jacomb PA, Easteal S. Association of Adverse Childhood Experiences, Age of Menarche, and Adult Reproductive Behavior: Does the Androgen Receptor Gene Play a Role? *Am J Med Genet - Neuropsychiatr Genet.* 2004;125 B(1):105-111. doi:10.1002/ajmg.b.20114
109. Colich NL, Platt JM, Keyes KM, Sumner JA, Allen NB, McLaughlin KA. Earlier age at menarche as a transdiagnostic mechanism linking childhood trauma with multiple forms of psychopathology in adolescent girls. *Psychol Med.* 2020;50(7):1090-1098. doi:10.1017/S0033291719000953
110. Li H, Song L, Shen L, et al. Age at menarche and prevalence of preterm birth: Results from the Healthy Baby Cohort study. *Sci Rep.* 2017;7(1):1-7. doi:10.1038/s41598-017-12817-2
111. Young-Wolff KC, Wei J, Varnado N, Rios N, Staunton M, Watson C. Adverse Childhood Experiences and Pregnancy Intentions among Pregnant Women Seeking Prenatal Care. *Women's Heal Issues.* 2021;31(2):100-106. doi:10.1016/j.whi.2020.08.012
112. Valles SA. *Philosophy of Population Health.* 1st ed. Routledge Taylor and Francis Group; 2018. doi:10.4324/9781315163307
113. Antonovsky A. The salutogenic model as a theory to guide health promotion 1. *Health Promot Int.* 1996;11(1):11-18. doi:10.1093/heapro/11.1.11

114. Masten AS, Tellegen A. Resilience in developmental psychopathology: Contributions of the Project Competence Longitudinal Study. *Dev Psychopathol.* 2012;24(2):345-361. doi:10.1017/S095457941200003X
115. Olsson CA, Bond L, Burns JM, Vella-brodrick DA, Sawyer SM. Adolescent resilience : a concept analysis. *J Adolesc.* 2003;26:1-11.
116. Bauer GR. Incorporating intersectionality theory into population health research methodology: Challenges and the potential to advance health equity. *Soc Sci Med.* 2014;110:10-17. doi:10.1016/j.socscimed.2014.03.022
117. Gariépy G, Honkaniemi H, Quesnel-Vallée A. Social support and protection from depression: Systematic review of current findings in western countries. *Br J Psychiatry.* 2016;209(4):284-293. doi:10.1192/bjp.bp.115.169094
118. Stevens E, Jason LA, Ram D, Light J. Investigating Social Support and Network Relationships in Substance Use Disorder Recovery. *Subst Abus.* 2015;36(4):396-399. doi:10.1080/08897077.2014.965870
119. Verheijden MW, Bakx JC, van Weel C, Koelen MA, van Staveren WA. Role of social support in lifestyle-focused weight management interventions. *Eur J Clin Nutr.* 2005;59(SUPPL. 1). doi:10.1038/sj.ejcn.1602194
120. Stopford R, Winkley K, Ismail K. Social support and glycemic control in type 2 diabetes: A systematic review of observational studies. *Patient Educ Couns.* 2013;93(3):549-558. doi:10.1016/j.pec.2013.08.016
121. Carlson LE, Goodey E, Bennett MH, Taenzer P, Koopmans J. The addition of social support to a community-based large-group behavioral smoking cessation intervention: Improved cessation rates and gender differences. *Addict Behav.* 2002;27(4):547-559. doi:10.1016/S0306-4603(01)00192-7
122. Hamilton MM, Razzano LA, Martin NB. The relationship between type and quality of social support and HIV medication adherence. *HIV Treat Adherence Challenges Soc Serv.* 2013;1501:39-64. doi:10.1300/J187v06n01_04
123. Kawachi I, Colditz GA, Ascherio A, et al. A prospective study of social networks in relation to total mortality and cardiovascular disease in men in the USA. *J Epidemiol Community Health.* 1996;50(3):245-251. doi:10.1136/jech.50.3.245
124. Barth J, Schneider S, Von Känel R. Lack of social support in the etiology and the prognosis of coronary heart disease: A systematic review and meta-analysis. *Psychosom Med.* 2010;72(3):229-238. doi:10.1097/PSY.0b013e3181d01611

125. Abu HO, Ulbricht C, Ding E, et al. Association of religiosity and spirituality with quality of life in patients with cardiovascular disease: a systematic review. *Qual Life Res.* 2018;27(11):2777-2797. doi:10.1007/s11136-018-1906-4
126. Koenig HG, George LK, Hays JC, Larson DB, Cohen HJ, Blazer DG. The relationship between religious activities and blood pressure in older adults. *Int J Psychiatry Med.* 1998;28(2):189-213. doi:10.2190/75JM-J234-5JKN-4DQD
127. Bonelli R, Dew RE, Koenig HG, Rosmarin DH, Vasegh S. Religious and spiritual factors in depression: Review and integration of the research. *Depress Res Treat.* 2012;2012. doi:10.1155/2012/962860
128. Koenig HG. Research on religion, spirituality, and mental health: A review. *Can J Psychiatry.* 2009;54(5):283-291. doi:10.1177/070674370905400502
129. Darvyri P, Christodoulakis S, Galanakis M, Avgoustidis AG, Thanopoulou A, Chrousos GP. On the Role of Spirituality and Religiosity in Type 2 Diabetes Mellitus Management—A Systematic Review. *Psychology.* 2018;09(04):728-744. doi:10.4236/psych.2018.94046
130. Rivera-Hernandez M. Religiosity, Social Support and Care Associated with Health in Older Mexicans with Diabetes. *J Relig Health.* 2016;55(4):1394-1410. doi:10.1007/s10943-015-0105-7
131. Miller AS, Hoffmann JP. Risk and Religion: An Explanation of Gender Differences in Religiosity. *J Sci Study Relig.* 1995;34(1):63. doi:10.2307/1386523
132. Miller AS, Stark R. Gender and religiousness: Can socialization explanations be saved? *Am J Sociol.* 2002;107(6):1399-1423. doi:10.1086/342557
133. Levin JS, Taylor RJ, Chatters LM. Race and gender differences in religiosity among older adults: Findings from four national surveys. *Journals Gerontol.* 1994;49(3):137-145. doi:10.1093/geronj/49.3.S137
134. Chatters LM, Taylor RJ, Jackson JS, Lincoln KD. Religious coping among African Americans, Caribbean Blacks and Non-Hispanic Whites. *J Community Psychol.* 2008;36(3):371-386.
135. Gregg G. I ' m A Jesus Girl : Coping Stories of Black American Women Diagnosed with Breast Cancer. *J Relig Health.* 2011;50(4):1040-1053.
136. Chen E. Protective Factors for Health Among Low-Socioeconomic-Status Individuals. *Curr Dir Psychol Sci.* 2012;21(3):189-193. doi:10.1177/0963721412438710

137. Joshi P, Hardy E, Hawkins S. *Role of Religiosity in the Lives of Low-Income Population: A Comprehensive Review of the Evidence*. Washington, D.C.; 2009.
138. McDonald SW, Kingston D, Bayrampour H, Dolan SM, Tough SC. Cumulative psychosocial stress, coping resources, and preterm birth. *Arch Womens Ment Health*. 2014;17(6):559-568. doi:10.1007/s00737-014-0436-5
139. Holzman C, Bullen B, Fisher R, Paneth N, Reuss L. Pregnancy outcomes and community health: the POUCH study of preterm birth. *Paediatr Perinat Epidemiol*. 2001;15(s2):136-158. doi:10.1046/j.1365-3016.2001.00014.x
140. Jesse DE, Walcott-McQuigg J, Mariella A, Swanson MS. Risks and protective factors associated with symptoms of depression in low-income African American and Caucasian women during pregnancy. *J Midwifery Women's Heal*. 2005;50(5):405-410. doi:10.1016/j.jmwh.2005.05.001
141. Jesse DE, Swanson MS, Newton ER, Morrow J. Racial Disparities in Biopsychosocial Factors and Spontaneous Preterm Birth Among Rural Low-Income Women. *J Midwifery Women's Heal*. 2009;54(1):35-42. doi:10.1016/j.jmwh.2008.08.009
142. Taylor RJ, Chatters LM, Woodward AT, Brown E. Racial and ethnic differences in extended family, friendship, fictive kin, and congregational informal support networks. *Fam Relat*. 2013;62(4):609-624. doi:10.1111/fare.12030
143. Stephens NM, Cameron JS, Townsend SSM. Lower Social Class Does Not (Always) Mean Greater Interdependence: Women in Poverty Have Fewer Social Resources Than Working-Class Women. *J Cross Cult Psychol*. 2014;45(7):1061-1073. doi:10.1177/0022022114534768
144. Chatters LM, Taylor RJ, Lincoln KD. African American Religious Participation : A Multi-Sample Comparison. *J Sci Study Relig*. 1999;38(1):132-145.
145. Taylor RJ. Structural Determinants of Religious Participation among Black Americans. *Rev Relig Res*. 1988;30(2):114-125.
146. Taylor RJ, Chatters LM, Levin J. *Religion in the Lives of African Americans: Social, Psychological, and Health Perspectives*. Thousand Oaks: SAGE Publications; 2004. doi:http://dx.doi.org/10.4135/9781452229782
147. Taylor RJ, Chatters LM, Brown RK. African American religious participation. *Rev Relig Res*. 2014;56(4):513-538. doi:10.1007/s13644-013-0144-z.
148. Lipira L, Williams EC, Nevin PE, et al. Religiosity, Social Support, and Ethnic Identity: Exploring “resilience Resources” for African-American Women Experiencing HIV-

- Related Stigma. *J Acquir Immune Defic Syndr*. 2019;81(2):175-183. doi:10.1097/QAI.0000000000002006
149. Mann RJ, Abercrombie PD, DeJoseph J, Norbeck JS, Smith RT. The Personal Experience of Pregnancy for African-American Women. *J Transcult Nurs*. 1999;10(4):297-305. doi:10.1177/104365969901000408
 150. Jesse DEl, Schoneboom C, Blanchard A. The Effect of Faith or Spirituality in Pregnancy: A Content Analysis. *J Holist Nurs*. 2007;25(3):151-158. doi:10.1177/0898010107300882
 151. Reed TD, Neville HA. The Influence of Religiosity and Spirituality on Psychological Well-Being Among Black Women. *J Black Psychol*. 2014;40(4):384-401. doi:10.1177/0095798413490956
 152. Van Olphen J, Schulz A, Israel B, et al. Religious involvement, social support, and health among African-American women on the east side of Detroit. *J Gen Intern Med*. 2003;18(7):549-557. doi:10.1046/j.1525-1497.2003.21031.x
 153. Cyphers NA, Clements AD, Lindseth G. The Relationship Between Religiosity and Health-Promoting Behaviors in Pregnant Women. *West J Nurs Res*. 2017;39(11):1429-1446. doi:10.1177/0193945916679623
 154. Burdette AM, Weeks J, Hill TD, Eberstein IW. Maternal religious attendance and low birth weight. *Soc Sci Med*. 2012;74(12):1961-1967. doi:10.1016/j.socscimed.2012.02.021
 155. Page RL, Ellison CG, Lee J. Does Religiosity Affect Health Risk Behaviors in Pregnant and Postpartum Women ? *Matern Child Health J*. 2009;13:621-632. doi:10.1007/s10995-008-0394-5
 156. Holzman C, Senagore P, Tian Y, et al. Maternal catecholamine levels in midpregnancy and risk of preterm delivery. *Am J Epidemiol*. 2009;170(8):1014-1024. doi:10.1093/aje/kwp218
 157. Tiedje L, Holzman CB, De Vos E, et al. Hostility and anomie: links to preterm delivery subtypes and ambulatory blood pressure at mid-pregnancy. *Soc Sci Med*. 2008;66(6):1310-1321.
 158. Chetty R, Hendren N, Kline P, Saez E. Where is the land of opportunity? The geography of intergenerational mobility in the United States. *Q J Econ*. 2014;129(4):1553-1623.
 159. Tian Y, Holzman C, Slaughter-Acey J, Margerison-Zilko C, Luo Z, Todem D. Maternal Socioeconomic Mobility and Preterm Delivery: A Latent Class Analysis. *Matern Child Health J*. 2018;22(11):1647-1658. doi:10.1007/s10995-018-2562-6

160. Slaughter-Acey JC, Holzman C, Calloway D, Tian Y. Movin' on Up: Socioeconomic Mobility and the Risk of Delivering a Small-for-Gestational Age Infant. *Matern Child Health J.* 2016;20(3):613-622. doi:10.1007/s10995-015-1860-5
161. Misra DP, O'Campo P, Strobino D. Testing a sociomedical model for preterm delivery. *Paediatr Perinat Epidemiol.* 2001;15(2):110-122. doi:10.1046/j.1365-3016.2001.00333.x
162. Buka SL, Brennan RT, Rich-Edwards JW, Raudenbush SW, Earls F. Neighborhood Support and the Birth Weight of Urban Infants. *Am J Epidemiol.* 2003;157(1):1-8.
163. Tendulkar SA, Koenen KC, Dunn EC, Buka S, Subramanian S V. Neighborhood influences on perceived social support among parents: Findings from the project on human development in Chicago neighborhoods. *PLoS One.* 2012;7(4):1-9. doi:10.1371/journal.pone.0034235
164. Giurgescu C, Misra DP, Sealy-Jefferson S, et al. The impact of neighborhood quality, perceived stress, and social support on depressive symptoms during pregnancy in African American women. *Soc Sci Med.* 2015;130:172-180. doi:10.1016/j.socscimed.2015.02.006
165. Mair C, Diez Roux A V., Morenoff JD. Neighborhood stressors and social support as predictors of depressive symptoms in the Chicago Community Adult Health Study. *Heal Place.* 2010;16(5):811-819. doi:10.1016/j.healthplace.2010.04.006
166. Mann JR, Mckeown RE. Religiosity , Spirituality , and Tobacco Use by Pregnant Women. *South Med J.* 2007;100(9):867-872.
167. Mann JR, Mannan J, Quiñones LA, Palmer AA, Torres M. Religion, spirituality, social support, and perceived stress in pregnant and postpartum hispanic women. *JOGNN - J Obstet Gynecol Neonatal Nurs.* 2010;39(6):645-657. doi:10.1111/j.1552-6909.2010.01188.x
168. Manlove J, Logan C, Moore KA, Ikramullah E. Pathways from Family Religiosity to Adolescent Sexual Activity and Contraceptive Use. *Perspect Sex Reprod Health.* 2008;40(2):105-117. doi:10.1363/4010508
169. McCree DH, Wingood GM, DiClemente R, Davies S, Harrington KF. Religiosity and risky sexual behavior in African-American adolescent females. *J Adolesc Heal.* 2003;33(1):2-8. doi:10.1016/S1054-139X(02)00460-3
170. Najman JM, Williams GM, Keeping JD, Morrison J, Anderson MJ. Religious values, practices and pregnancy outcomes: A comparison of the impact of sect and mainstream christian affiliation. *Soc Sci Med.* 1988;26(4):401-407. doi:10.1016/0277-9536(88)90308-5

171. Page RL, Perez-Patron M, Han G, Burdette AM, Badejo M. Religiosity, Acculturation, and Preterm Birth in Mexican-Origin Women: A Pilot Study. *Hisp Heal Care Int*. 2021;19(3):155-162. doi:10.1177/1540415320976643
172. Paul Victor CG, Treschuk J V. Critical Literature Review on the Definition Clarity of the Concept of Faith, Religion, and Spirituality. *J Holist Nurs*. 2020;38(1):107-113. doi:10.1177/0898010119895368
173. Sessanna L, Finnell D, Jezewski M. Spirituality in Nursing and Health-Related Literature A Concept Analysis. *J Holist Nurs*. 2007;25(4):252-262.
174. Damiano R, Peres M, Sena M. Conceptualizing Spirituality and Religiousness. In: Lucchetti G, Peres M, Damiano R, eds. *Spirituality, Religiousness, and Health*. Cham, Switzerland: Springer, Cham; 2019:3-10.
175. Fetzer Institute, National Institute on Aging Working Group. *Multidimensional Measurement of Religiousness/Spirituality for Use in Health Research.*; 1999.
176. Arrey AE, Bilsen J, Lacor P, Deschepper R. Spirituality/religiosity: Acultural and psychological resource among sub-saharan african migrant women with HIV/AIDS in Belgium. *PLoS One*. 2016;11(7):1-22. doi:10.1371/journal.pone.0159488
177. Puchalski CM, Vitillo R, Hull SK, Reller N. Improving the spiritual dimension of whole person care: Reaching national and international consensus. *J Palliat Med*. 2014;17(6):642-656. doi:10.1089/jpm.2014.9427
178. Breilsford GM, Doheny KK, Nestler L. Parents' post-traumatic growth and spirituality post-neonatal intensive care unit discharge. *J Psychol Theol*. 2020;48(1):34-43. doi:10.1177/0091647119856468
179. Alvarenga W de A, de Montigny F, Zeghiche S, Polita NB, Verdon C, Nascimento LC. Understanding the spirituality of parents following stillbirth: A qualitative meta-synthesis. *Death Stud*. 2021;45(6):420-436. doi:10.1080/07481187.2019.1648336
180. Cowchock FS, Ellestad SE, Meador KG, Koenig HG, Hooten EG, Swamy GK. Religiosity is an Important Part of Coping with Grief in Pregnancy After a Traumatic Second Trimester Loss. *J Relig Health*. 2011;50(4):901-910. doi:10.1007/s10943-011-9528-y
181. Masten AS, Roisman GI, Long JD, et al. Developmental cascades: Linking academic achievement and externalizing and internalizing symptoms over 20 years. *Dev Psychol*. 2005;41(5):733-746. doi:10.1037/0012-1649.41.5.733
182. Harris KM, Halpern CT, Whitsel EA, et al. Cohort Profile: The National Longitudinal Study of Adolescent to Adult Health (Add Health). *Int J Epidemiol*. 2019;48(5):1-12.

doi:10.1093/ije/dyz115

183. Harris KM. The Add Health Study : Design and Accomplishments. *Carolina Popul Cent Univ North Carolina Chapel Hill*. 2013:1-22.
184. Carolina Population Center. The Add Health Research Design PowerPoint— National Longitudinal Study of Adolescent to Adult Health. https://addhealth.cpc.unc.edu/wp-content/uploads/docs/documentations/2020_Study_Design.pdf. Published 2019. Accessed December 8, 2019.
185. Harris KM. Correcting for Design Effects and Selecting Weights. Lecture presented at: Add Health Users Online Workshop; July 26, 2021.
186. Turner CF, Ku L, Rogers SM, Lindberg LD, Pleck JH, Sonenstein FL. Adolescent sexual behavior, drug use, and violence: Increased reporting with computer survey technology. *Science* (80-). 1998;280(5365):867-873. doi:10.1126/science.280.5365.867
187. Collins L, Lanza ST. *Latent Class and Latent Transition Analysis with Applications in the Social, Behavioral, and Health Sciences*. Hoboken: John Wiley & Sons; 2010.
188. Jakobsen JC, Gluud C, Wetterslev J, Winkel P. When and how should multiple imputation be used for handling missing data in randomised clinical trials - A practical guide with flowcharts. *BMC Med Res Methodol*. 2017;17(1):1-10. doi:10.1186/s12874-017-0442-1
189. Volkow ND, Gordon JA, Koob GF. Choosing appropriate language to reduce the stigma around mental illness and substance use disorders. *Neuropsychopharmacology*. 2021;46(13):2230-2232. doi:10.1038/s41386-021-01069-4
190. Beaton S, Forster P, Maple M. Suicide and language: why we shouldn't use the "C" word. *InPsych*. 2013:29-31.
191. Carter EB, Stuart JJ, Farland L V., et al. Pregnancy Complications as Markers for Subsequent Maternal Cardiovascular Disease: Validation of a Maternal Recall Questionnaire. *J Women's Heal*. 2015;24(9):702-712. doi:10.1089/jwh.2014.4953
192. Wise LA, Wang TR, Wesselink AK, et al. Accuracy of self-reported birth outcomes relative to birth certificate data in an Internet-based prospective cohort study. *Paediatr Perinat Epidemiol*. 2021;35(5):590-595. doi:10.1111/ppe.12769
193. Jones CP. Invited commentary: "Race," racism, and the practice of epidemiology. *Am J Epidemiol*. 2001;154(4):299-304. doi:10.1093/aje/154.4.299
194. American Association of Physical Anthropologists. Executive summary: AAPA statement on Race and Racism.

- https://bioanth.org/documents/199/AAPA_Race_statement_March_2019.pdf. Published 2019. Accessed February 21, 2021.
195. Zuberi T, Bonilla-Silva E. *White Logic, White Methods: Racism and Methodology*. Lanham: Rowman & Littlefield Publishers, Inc.; 2008.
 196. Krieger N. Refiguring “race”: epidemiology, racialized biology, and biological expressions of race relations. *Int J Heal Serv*. 2000;30(1):211-216.
 197. Mack K, Palfrey J. Capitalizing Black and White: Grammatical Justice and Equity. MacArthur Foundation. <https://www.macfound.org/press/perspectives/capitalizing-black-and-white-grammatical-justice-and-equity>. Published 2020. Accessed August 13, 2022.
 198. U.S. Bureau of the Census. *Income, Poverty, and Valuation of Noncash Benefits: 1994*. Washington, D.C.; 1996. <https://www2.census.gov/library/publications/1996/demographics/p60-189.pdf>.
 199. Lucchetti G, Peres MFP, Damiano RF, eds. *Spirituality, Religiousness and Health*. Cham, Switzerland: Springer International Publishing
 200. Lanza ST, Flaherty BP, Collins LM. *Latent Class and Latent Transition Analysis*. (Weiner IB, ed.); 2003. doi:10.1002/0471264385.wei0226
 201. Lanza ST, Cooper BR. Latent Class Analysis for Developmental Research. 2016;10(1):59-64. doi:10.1111/cdep.12163
 202. Weller BE, Bowen NK, Faubert SJ. Latent Class Analysis: A Guide to Best Practice. *J Black Psychol*. 2020;46(4):287-311. doi:10.1177/0095798420930932
 203. Bogat GA, Von Eye A, Bergman LR. Person-Oriented Approaches. In: Cicchetti D, ed. *Developmental Psychopathology, Volume 1: Theory and Method*. Third. John Wiley & Sons; 2016:797-845.
 204. Meehl P. A Most Peculiar Paradox. *Philos Stud*. 1950;1:47-48.
 205. Deichen Hansen ME. Predictors of preterm birth and low birth weight: A person-centered approach. *SSM - Popul Heal*. 2021;15:1-10. doi:10.1016/j.ssmph.2021.100897
 206. Bauer D, Shanahan M. Modeling complex interactions: person-centered and variable-centered approaches. In: *Modeling Contextual Effects in Longitudinal Studies*. Lawrence Erlbaum Associates, Inc.; 2007:255-283.
 207. Raykov T, Marcoulides GA. *A First Course in Structural Equation Modeling*. Mahwah, NJ: Lawrence Erlbaum Associates, Inc.; 2000.


208. Muthén L, Muthén O. (1998-2017). *Mplus User's Guide. Eighth Edition*. Los Angeles, CA: Muthen & Muthen
209. Bauer D, Steinley D. *Latent Class/Cluster Analysis and Mixture Modeling: Course Notes*. Chapel Hill, North Carolina: Curran-Bauer Analytics; June 1-5, 2020: p. 1.3-10.25.
210. Bauer D, Steinley D. *Latent Class/Cluster Analysis and Mixture Modeling Demonstration Notes: Mplus*. Chapel Hill, North Carolina: Curran-Bauer Analytics; June 1-5, 2020: p. 5.3-9.18.
211. Vermunt JK. Latent class modeling with covariates: Two improved three-step approaches. *Polit Anal*. 2010;18(4):450-469. doi:10.1093/pan/mpq025
212. Keele L, Stevenson RT. Causal interaction and effect modification : same model , different concepts. *Polit Sci Res Methods*. 2021;9:641-649. doi:10.1017/psrm.2020.12
213. Gillespie SL, Christian LM, Alston AD, Salsberry PJ. Childhood stress and birth timing among African American women: Cortisol as biological mediator. *Psychoneuroendocrinology*. 2017;84:32-41. doi:10.1016/j.psyneuen.2017.06.009
214. Smith M V., Gotman N, Yonkers KA. Early Childhood Adversity and Pregnancy Outcomes. *Matern Child Health J*. 2016;20(4):790-798. doi:10.1007/s10995-015-1909-5
215. McDonnell CG, Valentino K. Intergenerational Effects of Childhood Trauma: Evaluating Pathways Among Maternal ACEs, Perinatal Depressive Symptoms, and Infant Outcomes. *Child Maltreat*. 2016;21(4):317-326. doi:10.1177/1077559516659556
216. Bronfenbrenner U. *The Ecology of Human Development: Experiments by Nature and Design*. Cambridge, MA: Harvard University Press; 1979.
217. Christian LM. At the forefront of psychoneuroimmunology in pregnancy: Implications for racial disparities in birth outcomes: PART 2: Biological mechanisms. *Neurosci Biobehav Rev*. 2019;(July 2018):0-1. doi:10.1016/j.neubiorev.2019.03.010
218. Bronstein JM, Wingate MS, Brisendine AE. Why Is the U.S. Preterm Birth Rate So Much Higher Than the Rates in Canada, Great Britain, and Western Europe? *Int J Heal Serv*. 2018;48(4):622-640. doi:10.1177/0020731418786360
219. Cham H, Reshetnyak E, Rosenfeld B, Breitbart W. Full information maximum likelihood estimation for latent variable interactions with incomplete indicators. *Multivar Behav Res*. 2017;52(1):12-30.
220. Crowther S, Stephen A, Hall J. Association of psychosocial–spiritual experiences around childbirth and subsequent perinatal mental health outcomes: an integrated review. *J*

221. Vitorino LM, Chiaradia R, Low G, et al. Association of spiritual/religious coping with depressive symptoms in high- and low-risk pregnant women. *J Clin Nurs.* 2018;27:e635-e642. doi:10.1111/jocn.14113
222. Jesse DE, Graham M, Swanson M. Psychosocial and spiritual factors associated with smoking and substance use during pregnancy in African American and white low-income women. *JOGNN - J Obstet Gynecol Neonatal Nurs.* 2006;35(1):68-77. doi:10.1111/j.1552-6909.2006.00010.x
223. Salinas-Miranda A, King L, Salihu H, et al. Protective Factors using the Life Course Perspective in Maternal and Child Health. *Engage.* 2020;1(3):69-86. doi:10.18060/24205
224. Davis CE, Lamson AL, Meier EM, Pratt GA. The Intersect of Pregnancy Loss, Trauma, and Spirituality: A Dyadic Perspective. *Matern Child Health J.* 2022;26(8):1709-1718. doi:10.1007/s10995-022-03452-z
225. Homan KJ, Hollenberger JC. Religious Support Moderates the Long Reach of Adverse Childhood Events on Physical Health in Middle to Late Adulthood. *J Relig Health.* 2021;60(6):4186-4208. doi:10.1007/s10943-021-01259-w
226. Freeny J, Peskin M, Schick V, et al. Adverse Childhood Experiences, Depression, Resilience, & Spirituality in African-American Adolescents. *J Child Adolesc Trauma.* 2021;14(2):209-221. doi:10.1007/s40653-020-00335-9
227. Liu J, Abdin E, Vaingankar JA, et al. Positive mental health framework of transdiagnostic protective factors in elucidating the association between adverse childhood experiences and severe mental disorders. *Aust N Z J Psychiatry.* 2021;00(0):1-12. doi:10.1177/00048674211053568
228. Mefford LC, Phillips KD, Chung ML. Childhood Loss Experiences, Religiousness/Spirituality, and Hope as Predictors of Adult Life Satisfaction. *Issues Ment Health Nurs.* 2021;42(7):649-659. doi:10.1080/01612840.2020.1828514
229. Brockie TN, Elm JHL, Walls ML. Examining protective and buffering associations between sociocultural factors and adverse childhood experiences among American Indian adults with type 2 diabetes: a quantitative, community-based participatory research approach. *BMJ Open.* 2018;8(9):1-8. doi:10.1136/bmjopen-2018-022265
230. Kosarkova A, Malinakova K, van Dijk JP, Tavel P. Childhood trauma and experience in close relationships are associated with the god image: Does religiosity make a difference? *Int J Environ Res Public Health.* 2020;17(23):1-13. doi:10.3390/ijerph17238841

231. Janů A, Malinakova K, Kosarkova A, Tavel P. Associations of childhood trauma experiences with religious and spiritual struggles. *J Health Psychol.* 2022;27(2):292-304. doi:10.1177/1359105320950793
232. Brewer-Smyth K, Koenig HG. Could spirituality and religion promote stress resilience in survivors of childhood trauma? *Issues Ment Health Nurs.* 2014;35(4):251-256. doi:10.3109/01612840.2013.873101
233. Bengtson VL, Silverstein M, Putney NM, Harris SC. Does Religiousness Increase with Age? Age Changes and Generational Differences Over 35 Years. *J Sci Study Relig.* 2015;54(2):363-379. doi:10.1111/jssr.12183
234. Koenig HG. Religion, Spirituality, and Health: The Research and Clinical Implications. *ISRN Psychiatry.* 2012;2012:1-33. doi:10.5402/2012/278730
235. Gill CS, Minton CAB, Myers JE. Spirituality and Religiosity: Factors Affecting Wellness Among Low-Income, Rural Women. *J Couns Dev.* 2010;88:293-302.
236. Schmidt MR, Narayan AJ, Atzl VM, Rivera LM, Lieberman AF. Childhood Maltreatment on the Adverse Childhood Experiences (ACEs) Scale versus the Childhood Trauma Questionnaire (CTQ) in a Perinatal Sample. *J Aggress Maltreatment Trauma.* 2020;29(1):38-56. doi:10.1080/10926771.2018.1524806
237. Bronfenbrenner U. Ecology of the family as a context for human development: Research perspectives. *Dev Psychol.* 1986;22(6):723-742.
238. Margerison CE, MacCallum CL, Chen J, Zamani-Hank Y, Kaestner R. Impacts of Medicaid Expansion on Health Among Women of Reproductive Age. *Am J Prev Med.* 2020;58(1):1-11. doi:10.1016/j.amepre.2019.08.019
239. Dubowitz H, Feigelman S, Lane W, Jeongeun K. Pediatric primary care to help prevent child maltreatment: the safe environment for every kid (SEEK). *Pediatrics.* 2009;123(3):858-864.
240. Seccombe K. "Beating the odds" versus "changing the odds": poverty, resilience, and family policy. *J Marriage Fam.* 2002;64(2):384-394.

APPENDIX A: IRB DETERMINATION

Figure A1. IRB Determination Letter

<div><p>Office of Regulatory Affairs Human Research Protection Program</p><p>4000 Collins Road Suite 136 Lansing, MI 48910</p><p>517-355-2180 Fax: 517-432-4503 Email: irb@msu.edu www.hrpp.msu.edu</p><p><small>MSU is an affirmative-action, equal-opportunity employer.</small></p></div>		<div><p>MICHIGAN STATE UNIVERSITY</p><p>EXEMPT DETERMINATION Revised Common Rule</p><p>February 10, 2020</p><p>To: Claire Margerison</p><p>Re: MSU Study ID: STUDY00003945 Principal Investigator: Claire Margerison Category: Exempt 4(ii) Exempt Determination Date: 2/10/2020 Limited IRB Review: Not Required.</p><p>Title: The Role of Resilience in Pregnancy Health and Birth Outcomes using a Lifecourse Approach</p><p>This study has been determined to be exempt under 45 CFR 46.104(d) 4(ii).</p><p>Principal Investigator (PI) Responsibilities: The PI assumes the responsibilities for the protection of human subjects in this study as outlined in Human Research Protection Program (HRPP) Manual Section 8-1, Exemptions.</p><p>Continuing Review: Exempt studies do not need to be renewed.</p><p>Modifications: In general, investigators are not required to submit changes to the Michigan State University (MSU) Institutional Review Board (IRB) once a research study is designated as exempt as long as those changes do not affect the exempt category or criteria for exempt determination (changing from exempt status to expedited or full review, changing exempt category) or that may substantially change the focus of the research study such as a change in hypothesis or study design. See HRPP Manual Section 8-1, Exemptions, for examples. If the study is modified to add additional sites for the research, please note that you may not begin the research at those sites until you receive the appropriate approvals/permissions from the sites.</p><p>Please contact the HRPP office if you have any questions about whether a change must be submitted for IRB review and approval.</p><p>New Funding: If new external funding is obtained for an active study that had been determined exempt, a new initial IRB submission will be required, with limited exceptions. If you are unsure if a new initial IRB submission is required, contact the HRPP office. IRB review of the new submission must be completed before new funds can be spent on human research activities, as the new funding source may have additional or different requirements.</p></div>
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APPENDIX B: MANUSCRIPT 1

Table B1. Adverse childhood experiences assessed in the National Longitudinal Study of Adolescent to Adult Health (Add Health)
(n=3,767), 1994-2018.¹

ACEs	Period of data collection	Survey question(s)	Operationalization
Abuse			
Physical	Wave IV	Before your 18 th birthday, how often did a parent or adult caregiver hit you with a fist, kick you, or throw you down on the floor, into a wall or down stairs?	Yes/No
Sexual	Wave IV	Before your 18 th birthday, how often did a parent or other adult caregiver touch you in a sexual way, force you to touch him or her in a sexual way, or force you to have sexual relations?	Yes/No
Emotional	Wave IV	Before your 18 th birthday, how often did a parent or other adult caregiver say things that really hurt your feelings or made you feel like you were not wanted or loved?	Yes/No
Neglect	Wave III	By the time you started 6 th grade, how often had your parents or other adult caregivers left you home alone when an adult should have been with you? How often had your parents or other adult caregivers not taken care of your basic needs, such as keeping you clean or providing food or clothing? How often had social services investigated how you were taken care of or tried to take you out of your living situation?	Yes/No

Table B1 (cont'd).

Household Dysfunction			
Suicide	Wave I	Have any of your family members tried to kill themselves during the past 12 months?	Yes/No
		Have any of your family members succeeded in killing themselves during the past 12 months?	
Foster care placement	Wave III	Did you ever live in a foster home?	Yes/No

¹These questions are from Add Health, funded by grant P01 HD31921 (Harris) from the Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD), with cooperative funding from 23 other federal agencies and foundations. Add Health is currently directed by Robert A. Hummer and funded by the National Institute on Aging cooperative agreements U01 AG071448 (Hummer) and U01AG071450 (Aiello and Hummer) at the University of North Carolina at Chapel Hill. Add Health was designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris at the University of North Carolina at Chapel Hill. Information on how to obtain the Add Health data files is available on the Add Health website (<https://addhealth.cpc.unc.edu>). No direct support was received from grant P01-HD31921 for this project.

Table B2. Socioeconomic variables from Wave I of the National Longitudinal Study of Adolescent to Adult Health (1994) used to operationalize childhood socioeconomic status.¹

Socioeconomic variables	Low SES (0)	High SES (1)
Maternal occupation	Sales worker (i.e., insurance agent, store clerk)	Professional (i.e., doctor, lawyer, scientist)
Paternal occupation	Restaurant worker or personal service (i.e., waitress, housekeeper)	Professional (i.e., teacher, librarian, nurse)
	Craftsperson (i.e., toolmaker, woodworker)	Manager (i.e., executive, director)
	Construction worker (i.e., carpenter, crane operator)	Technical (i.e., computer specialist, radiologist)
	Mechanic (i.e., electrician, plumber, machinist)	
	Factory worker or laborer (i.e., assembler, janitor)	
	Transportation (i.e., bus driver, taxi driver)	
	Military or security (i.e., police officer, soldier, fire fighter)	
	Farm or fishery worker	
	Other	
	None	
	Missing	

Table B2 (cont'd).

Maternal education Paternal education	Eighth grade or less	Went to a business, trade, or vocational school after high school
	More than eighth grade, but did not graduate from high school	Went to college, but did not graduate
	Went to a business, trade, or vocational school	Graduated from a college or university
	High school graduate	Professional training beyond a four-year college or university
	Completed a GED	
	Never went to school	
	Missing	
Gross household income in 1994²	≤\$32,000	>\$32,000
	Missing	
Receipt of public assistance	Yes	No
	Missing	

¹Informed by the Slaughter-Acey method (Slaughter-Acey et al., 2016)¹⁶⁰

²Income threshold informed by the national gross median household income in the United States in 1994 (U.S. Census Bureau, 1996).¹⁹⁸

Table B3. Assessment of bias between the reference population and two analytic samples on sociodemographic and health-related characteristics among women in the National Longitudinal Study of Adolescent to Adult Health (1994-2018).

	Reference Population (Women with live birth pregnancies) Max N = 4,034		Manuscripts 2 & 3 Analytic Sample Max N = 3,884		Manuscript 1 Analytic Sample Max N = 3,767	
	N	(Wt%)	N	(Wt%)	N	(Wt%)
Race						
White	2346	(68.1)	2297	(68.7)	2226	(68.5)
Black	773	(14.6)	723	(14.4)	702	(14.4)
Hispanic	538	(10.5)	517	(10.3)	502	(10.4)
Asian	205	(2.9)	197	(2.9)	194	(3.0)
Pacific Islander	35	(0.5)	32	(0.5)	29	(0.5)
American Indian or Alaska Native	93	(2.4)	89	(2.4)	86	(2.4)
Other	31	(0.8)	29	(0.8)	28	(0.8)
Socioeconomic status (SES)						
Low	513	(12.6)	487	(12.5)	473	(12.7)
Middle	2476	(61.0)	2382	(61.1)	2309	(61.1)
High	1045	(26.4)	1015	(26.4)	985	(26.2)
Marital Status						
Missing	356	(8.7)	324	(8.2)	314	(8.0)
Married/Cohabiting	2986	(75.3)	2893	(75.7)	2807	(76.0)
Not Married/Cohabiting	692	(16.0)	667	(16.1)	646	(16.0)
Maternal age at time of delivery (years)						
Missing	229	(6.2)	198	(5.4)	190	(5.4)
≤19	597	(14.5)	562	(14.5)	537	(14.3)
20-24	1090	(28.3)	1048	(28.2)	1021	(28.3)
25-29	1043	(24.6)	1021	(24.9)	996	(25.1)
30-34	818	(20.2)	807	(20.7)	783	(20.8)
>35	257	(6.1)	248	(6.2)	240	(6.1)
Preterm Delivery (PTD)						

Table B3 (cont'd).

Yes	520	(11.6)	514	(11.8)	501	(11.7)
Adverse Childhood Experiences (ACEs)						
Sexual abuse	225	(6.2)	217	(6.3)	212	(6.2)
Physical abuse	765	(20.1)	738	(20.2)	721	(20.4)
Emotional abuse	1865	(47.6)	1793	(47.7)	1750	(48.2)
Neglect	1674	(42.0)	1614	(42.3)	1585	(42.8)
Family Member suicide attempt or death	229	(6.1)	216	(6.1)	209	(6.0)
Foster care placement	94	(2.7)	90	(2.7)	87	(2.7)
Protective Factors	Mean (SE)		Mean (SE)			
Religiosity	1.4	(.02)	1.4	(.02)	NA ¹	NA ¹
Spirituality	1.5	(.02)	1.5	(.02)	NA ¹	NA ¹
R/S	10.4	(.06)	10.4	(.06)	NA ¹	NA ¹

¹Variables not assessed in Manuscript 1

Table B4. Adjusted odds ratios for the association between six ACEs and odds of PTD among race subgroups of women in the National Longitudinal Study of Adolescent to Adult Health (n=3,767), 1994-2018.¹

	White (n=2226)	Black (n=702)	Hispanic (n=502)	Asian (n=194)	PI (n=29)	AI/AN (n=86)	Other (n=28)
	aOR (95%CI)	aOR (95%CI)	aOR (95%CI)	aOR (95%CI)	aOR (95%CI)	aOR (95%CI)	aOR (95%CI)
Sexual abuse	1.4 (0.7,3.0)	0.3 (0.1, 0.9)	96.7 (12.5, 750.1)	0.8 (0.1, 6.8)	*	*	*
Physical abuse	0.7 (0.5, 1.0)	0.7 (0.3, 1.4)	1.6 (0.4, 6.4)	1.3 (0.3, 5.3)	*	*	*
Emotional abuse	1.3 (0.9, 1.8)	1.6 (0.9, 3.1)	1.5 (0.6, 4.1)	0.4 (0.1, 1.4)	*	1.4 (0.4, 4.7)	0.2 (0.0, 3.5)
Neglect	0.9 (0.6, 1.2)	1.0 (0.5, 1.8)	1.0 (0.4, 2.6)	1.4 (0.3, 5.9)	*	1.5 (0.0, 6.9)	0.4 (0.0, 6.3)
Suicide	1.0 (0.5, 1.8)	2.6 (0.5, 14.8)	0.7 (0.2, 2.2)	39.2 (6.4, 241.2)	*	0.1 (0.0, 15.2)	*
Foster	5.0 (0.9, 29.2)	5.1 (0.5, 48.5)	0.2 (0.0, 1.3)	0.2 (0.0, 3.9)	*	*	*

¹OR estimates are adjusted for SES; all results are survey-weighted to account for the Add Health stratified study design

*Precise estimates could not be derived due to small cell sizes

Table B5. Adjusted odds ratios for the association between six ACEs and odds of PTD among SES subgroups of women in the National Longitudinal Study of Adolescent to Adult Health (n=3,767), 1994-2018.¹

	Low SES (n=473)	Middle SES (n=2309)	High SES (n=985)
	aOR (95% CI)	aOR (95% CI)	aOR (95% CI)
Sexual abuse	1.5 (0.4, 4.7)	1.4 (0.7, 3.1)	0.7 (0.2, 1.9)
Physical abuse	1.4 (0.5, 3.8)	0.8 (0.5, 1.2)	0.7 (0.4, 1.4)
Emotional abuse	2.0 (0.9, 4.8)	1.1 (0.6, 1.9)	1.1 (0.6, 1.9)
Neglect	0.7 (0.4, 1.4)	0.9 (0.6, 1.3)	1.0 (0.6, 1.7)
Suicide	0.5 (0.2, 1.4)	1.4 (0.7, 3.2)	0.9 (0.3, 2.6)
Foster	*	1.0 (0.4, 2.7)	1.6 (0.2, 13.4)

¹OR estimates are adjusted for race; all results are survey-weighted to account for Add Health stratified study design

*Precise estimates could not be derived due to small cell sizes

APPENDIX C: MANUSCRIPT 2

Table C1. Adverse childhood experiences assessed in the National Longitudinal Study of Adolescent to Adult Health (Add Health)
(n=3,884), 1994-2018.¹

ACEs	Period of data collection	Survey question(s)	Operationalization
Abuse			
Physical	Wave IV	Before your 18 th birthday, how often did a parent or adult caregiver hit you with a fist, kick you, or throw you down on the floor, into a wall or down stairs?	Yes/No
Sexual	Wave IV	Before your 18 th birthday, how often did a parent or other adult caregiver touch you in a sexual way, force you to touch him or her in a sexual way, or force you to have sexual relations?	Yes/No
Emotional	Wave IV	Before your 18 th birthday, how often did a parent or other adult caregiver say things that really hurt your feelings or made you feel like you were not wanted or loved?	Yes/No
Neglect	Wave III	By the time you started 6 th grade, how often had your parents or other adult caregivers left you home alone when an adult should have been with you? How often had your parents or other adult caregivers not taken care of your basic needs, such as keeping you clean or providing food or clothing? How often had social services investigated how you were taken care of or tried to take you out of your living situation?	Yes/No

Table C1 (cont'd).

Household Dysfunction			
Suicide	Wave I	Have any of your family members tried to kill themselves during the past 12 months?	Yes/No
		Have any of your family members succeeded in killing themselves during the past 12 months?	
Foster care placement	Wave III	Did you ever live in a foster home?	Yes/No

¹These questions are from Add Health, funded by grant P01 HD31921 (Harris) from the Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD), with cooperative funding from 23 other federal agencies and foundations. Add Health is currently directed by Robert A. Hummer and funded by the National Institute on Aging cooperative agreements U01 AG071448 (Hummer) and U01AG071450 (Aiello and Hummer) at the University of North Carolina at Chapel Hill. Add Health was designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris at the University of North Carolina at Chapel Hill. Information on how to obtain the Add Health data files is available on the Add Health website (<https://addhealth.cpc.unc.edu>). No direct support was received from grant P01-HD31921 for this project.

Table C2. Socioeconomic variables from Wave I of the National Longitudinal Study of Adolescent to Adult Health (1994) used to operationalize childhood socioeconomic status.¹

Socioeconomic variables	Low SES (0)	High SES (1)
Maternal occupation	Sales worker (i.e., insurance agent, store clerk)	Professional (i.e., doctor, lawyer, scientist)
Paternal occupation	Restaurant worker or personal service (i.e., waitress, housekeeper)	Professional (i.e., teacher, librarian, nurse)
	Craftsperson (i.e., toolmaker, woodworker)	Manager (i.e., executive, director)
	Construction worker (i.e., carpenter, crane operator)	Technical (i.e., computer specialist, radiologist)
	Mechanic (i.e., electrician, plumber, machinist)	
	Factory worker or laborer (i.e., assembler, janitor)	
	Transportation (i.e., bus driver, taxi driver)	
	Military or security (i.e., police officer, soldier, fire fighter)	
	Farm or fishery worker	
	Other	
	None	
	Missing	

Table C2 (cont'd).

Maternal education Paternal education	Eighth grade or less	Went to a business, trade, or vocational school after high school
	More than eighth grade, but did not graduate from high school	Went to college, but did not graduate
	Went to a business, trade, or vocational school	Graduated from a college or university
	High school graduate	Professional training beyond a four-year college or university
	Completed a GED	
	Never went to school	
	Missing	
Gross household income in 1994²	<\$32,000	≥\$32,000
	Missing	
Receipt of public assistance	Yes	No
	Missing	

¹Informed by the Slaughter-Acey method (Slaughter-Acey et al., 2016)¹⁶⁰

²Income threshold informed by the national gross median household income in the United States in 1994.¹⁹⁸

Table C3. Assessment of bias between the reference population and two analytic samples on sociodemographic and health-related characteristics among women in the National Longitudinal Study of Adolescent to Adult Health (1994-2018).

	Reference Population (Women with live birth pregnancies) Max N = 4,034		Manuscripts 2 & 3 Analytic Sample Max N = 3,884		Manuscript 1 Analytic Sample Max N = 3,767	
	N	(Wt%)	N	(Wt%)	N	(Wt%)
Race						
White	2346	(68.1)	2297	(68.7)	2226	(68.5)
Black	773	(14.6)	723	(14.4)	702	(14.4)
Hispanic	538	(10.5)	517	(10.3)	502	(10.4)
Asian	205	(2.9)	197	(2.9)	194	(3.0)
Pacific Islander	35	(0.5)	32	(0.5)	29	(0.5)
American Indian or Alaska Native	93	(2.4)	89	(2.4)	86	(2.4)
Other	31	(0.8)	29	(0.8)	28	(0.8)
Socioeconomic status (SES)						
Low	513	(12.6)	487	(12.5)	473	(12.7)
Middle	2476	(61.0)	2382	(61.1)	2309	(61.1)
High	1045	(26.4)	1015	(26.4)	985	(26.2)
Marital Status						
Missing	356	(8.7)	324	(8.2)	314	(8.0)
Married/Cohabiting	2986	(75.3)	2893	(75.7)	2807	(76.0)
Not Married/Cohabiting	692	(16.0)	667	(16.1)	646	(16.0)
Maternal age at time of delivery (years)						
Missing	229	(6.2)	198	(5.4)	190	(5.4)
≤19	597	(14.5)	562	(14.5)	537	(14.3)
20-24	1090	(28.3)	1048	(28.2)	1021	(28.3)
25-29	1043	(24.6)	1021	(24.9)	996	(25.1)
30-34	818	(20.2)	807	(20.7)	783	(20.8)
>35	257	(6.1)	248	(6.2)	240	(6.1)

Table C3 (cont'd).

Preterm Delivery (PTD)					
Yes	520	(11.6)	514	(11.8)	501 (11.7)
Adverse Childhood Experiences (ACEs)					
Sexual abuse	225	(6.2)	217	(6.3)	212 (6.2)
Physical abuse	765	(20.1)	738	(20.2)	721 (20.4)
Emotional abuse	1865	(47.6)	1793	(47.7)	1750 (48.2)
Neglect	1674	(42.0)	1614	(42.3)	1585 (42.8)
Family Member suicide attempt or death	229	(6.1)	216	(6.1)	209 (6.0)
Foster care placement	94	(2.7)	90	(2.7)	87 (2.7)
Protective Factors	Mean (SE)		Mean (SE)		
Religiosity	1.4	(.02)	1.4	(.02)	NA ¹ NA ¹
Spirituality	1.5	(.02)	1.5	(.02)	NA ¹ NA ¹
R/S	10.4	(.06)	10.4	(.06)	NA ¹ NA ¹

¹Variables not assessed in Manuscript 1

APPENDIX D: MANUSCRIPT 3

Table D1. Adverse childhood experiences assessed in the National Longitudinal Study of Adolescent to Adult Health (Add Health)
(n=3,884), 1994-2018.¹

ACEs	Period of data collection	Item(s) in measure	Operationalization
Abuse			
Physical	Wave IV	Before your 18 th birthday, how often did a parent or adult caregiver hit you with a fist, kick you, or throw you down on the floor, into a wall or down stairs?	Yes/No
Sexual	Wave IV	Before your 18 th birthday, how often did a parent or other adult caregiver touch you in a sexual way, force you to touch him or her in a sexual way, or force you to have sexual relations?	Yes/No
Emotional	Wave IV	Before your 18 th birthday, how often did a parent or other adult caregiver say things that really hurt your feelings or made you feel like you were not wanted or loved?	Yes/No
Neglect	Wave III	By the time you started 6 th grade, how often had your parents or other adult caregivers left you home alone when an adult should have been with you? How often had your parents or other adult caregivers not taken care of your basic needs, such as keeping you clean or providing food or clothing? How often had social services investigated how you were taken care of or tried to take you out of your living situation?	Yes/No
Household Dysfunction			

Table D1 (cont'd).

Suicide	Wave I	Have any of your family members tried to kill themselves during the past 12 months?	Yes/No
		Have any of your family members succeeded in killing themselves during the past 12 months?	
Foster care placement	Wave III	Did you ever live in a foster home?	Yes/No

¹These questions are from Add Health, funded by grant P01 HD31921 (Harris) from the Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD), with cooperative funding from 23 other federal agencies and foundations. Add Health is currently directed by Robert A. Hummer and funded by the National Institute on Aging cooperative agreements U01 AG071448 (Hummer) and U01AG071450 (Aiello and Hummer) at the University of North Carolina at Chapel Hill. Add Health was designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris at the University of North Carolina at Chapel Hill. Information on how to obtain the Add Health data files is available on the Add Health website (<https://addhealth.cpc.unc.edu>). No direct support was received from grant P01-HD31921 for this project.

Table D2. Religiosity and spirituality variables captured in Wave III (2001-2002) of the National Longitudinal Study of Adolescent to Adult Health, 1994-2018.¹

Variable	Item(s) in measure	Possible responses	Operationalization of variable
Religiosity	To what extent are you a religious person?	0- Not religious at all 1- Slightly religious 2- Moderately religious 3- Very religious	Continuous (0-3); higher score means higher religiosity
Spirituality	To what extent are you a spiritual person?	0- Not religious at all 1- Slightly religious 2- Moderately religious 3- Very religious	Continuous (0-3); higher score means higher spirituality

Table D2 (cont'd).

Combined religiosity and spirituality variable (R/S)	To what extent do you agree with the following statement? Angels are present to help or watch over me	1- Strongly disagree 2- Disagree 3- Neither agree nor disagree 4- Agree 5- Strongly agree	Continuous (3-15); higher score means higher spirituality/religiosity
	To what extent do you agree with the following statement? I employ my religious or spiritual beliefs as a basis for how to act and live on a daily basis.	1- Strongly disagree 2- Disagree 3- Neither agree or disagree 4- Agree 5- Strongly agree	
	To what extent do you agree with the following statement? What seem to be coincidences in my life are not really coincidences...I am being led “spiritually”	1- Strongly disagree 2- Disagree 3- Neither agree or disagree 4- Agree 5- Strongly agree	

¹These questions are from Add Health, funded by grant P01 HD31921 (Harris) from the Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD), with cooperative funding from 23 other federal agencies and foundations. Add Health is currently directed by Robert A. Hummer and funded by the National Institute on Aging cooperative agreements U01 AG071448 (Hummer) and U01AG071450 (Aiello and Hummer) at the University of North Carolina at Chapel Hill. Add Health was designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris at the University of North Carolina at Chapel Hill. Information on how to obtain the Add Health data files is available on the Add Health website (<https://addhealth.cpc.unc.edu>). No direct support was received from grant P01-HD31921 for this project.

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Socioeconomic variables	Low SES (0)	High SES (1)
Maternal occupation	Sales worker (i.e., insurance agent, store clerk)	Professional (i.e., doctor, lawyer, scientist)
Paternal occupation	Restaurant worker or personal service (i.e., waitress, housekeeper)	Professional (i.e., teacher, librarian, nurse)
	Craftsperson (i.e., toolmaker, woodworker)	Manager (i.e., executive, director)
	Construction worker (i.e., carpenter, crane operator)	Technical (i.e., computer specialist, radiologist)
	Mechanic (i.e., electrician, plumber, machinist)	
	Factory worker or laborer (i.e., assembler, janitor)	
	Transportation (i.e., bus driver, taxi driver)	
	Military or security (i.e., police officer, soldier, fire fighter)	
	Farm or fishery worker	
	Other	
	None	
	Missing	

Table D3 (cont'd).

Maternal education Paternal education	Eighth grade or less	Went to a business, trade, or vocational school after high school
	More than eighth grade, but did not graduate from high school	Went to college, but did not graduate
	Went to a business, trade, or vocational school	Graduated from a college or university
	High school graduate	Professional training beyond a four-year college or university
	Completed a GED	
	Never went to school	
Gross household income in 1994²	Missing	
	<\$32,000	≥\$32,000
Receipt of public assistance	Missing	
	Yes	No
	Missing	

¹Informed by the Slaughter-Acey method (Slaughter-Acey et al., 2016)¹⁶⁰

²Income threshold informed by the national gross median household income in the United States in 1994.¹⁹⁸

Table D4. Assessment of bias between the reference population and two analytic samples on sociodemographic and health-related characteristics among women in the National Longitudinal Study of Adolescent to Adult Health (1994-2018).

	Reference Population (Women with live birth pregnancies) Max N = 4,034		Manuscripts 2 & 3 Analytic Sample Max N = 3,884		Manuscript 1 Analytic Sample Max N = 3,767	
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Socioeconomic status (SES)						
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Marital Status						
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Maternal age at time of delivery (years)						
Missing	229	(6.2)	198	(5.4)	190	(5.4)
≤19	597	(14.5)	562	(14.5)	537	(14.3)
20-24	1090	(28.3)	1048	(28.2)	1021	(28.3)
25-29	1043	(24.6)	1021	(24.9)	996	(25.1)
30-34	818	(20.2)	807	(20.7)	783	(20.8)
>35	257	(6.1)	248	(6.2)	240	(6.1)
Preterm Delivery (PTD)						

Table D4 (cont'd).

Yes	520	(11.6)	514	(11.8)	501	(11.7)
Adverse Childhood Experiences (ACEs)						
Sexual abuse	225	(6.2)	217	(6.3)	212	(6.2)
Physical abuse	765	(20.1)	738	(20.2)	721	(20.4)
Emotional abuse	1865	(47.6)	1793	(47.7)	1750	(48.2)
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Foster care placement	94	(2.7)	90	(2.7)	87	(2.7)
Protective Factors	Mean (SE)		Mean (SE)			
Religiosity	1.4	(.02)	1.4	(.02)	NA ¹	NA ¹
Spirituality	1.5	(.02)	1.5	(.02)	NA ¹	NA ¹
R/S	10.4	(.06)	10.4	(.06)	NA ¹	NA ¹

¹Variables not assessed in Manuscript 1

Table D5. Logits for the classification probabilities for the two-class model derived from Vermunt's 3-step approach in the National Longitudinal Study of Adolescent to Adult Health (n=3,884), 1994-2018

	Modal Class Assignment 1	Modal Class Assignment 2
Latent Class 1	-4.008	0.000
Latent Class 2	1.121	0.000