CHILDHOOD ADVERSITY AND ITS EFFECTS ON HEALTH OVER THE LIFESPAN: ANALYSIS OF THE ALLEGHENY COUNTY HEALTH SURVEY

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ABSTRACT

It has been estimated that 75% of the U.S. adult population has experienced some type of childhood adversity (CA), such as child maltreatment, parental divorce or violence. Evidence continues to mount that exposure to CA can lead to serious mental and physical health consequences that extend well into late life. The life course perspective (LCP) is a theoretical perspective often used to explain how early life exposures influence health and behavior across the lifespan. Using the LCP and secondary data collected from the 2009-2010 Allegheny County Health Survey (N=5442), this study describes the prevalence and disparities in CA in terms of social, demographic, and geographic characteristics. A series of bivariate and multivariate logistic regressions are conducted to determine which adult health indicators (e.g., smoking, perceived social support, serious mental illness, cancer, cardiovascular disease) are most associated with CA and to what extent the prevalence of these health issues could be reduced if CA was prevented. Furthermore, social, behavioral, and environmental pathways are evaluated in statistical models to determine which factors moderate and mediate the relationship between CA and adult health and behavior. Results indicate that CA is prevalent in the adult population of Allegheny County, Pennsylvania with an estimated 59.8% reporting at least one adverse childhood event. CA disparities were observed by gender, race, socioeconomic status,

unemployment status and disability status. Population Attributable Risk (PAR) fractions were calculated and revealed that approximately 42% of serious mental illness and 26% of cardiovascular disease in the population could be eliminated if ACEs were prevented. Social and behavioral factors that mediated the CA adult health relationships included adult socioeconomic status, social support, smoking, and body mass index. The effects of CA on adult health appeared stronger and more independent for mental health outcomes than physical health outcomes. Public Health Significance: For those at-risk of or those who have experienced CA, programs and policies that teach positive strategies for coping with stress and help families to increase human and social capital may greatly decrease adult morbidly associated with CA and reduce current and intergenerational CA disparities by race and socioeconomic status.

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1.0 INTRODUCTION

Childhood adversity (CA) is a broad concept that "refers to the challenges to development created by stressors outside of the family (such as poverty, warfare, and stranger assaults) and within it (such as neglect, sexual, physical and emotional abuse)."(1 p. 195) CA is often measured retrospectively by asking adults about the negative experiences and circumstances they may have experienced during childhood. These negative experiences and circumstances are commonly referred to as Adverse Childhood Experiences or ACEs. ACEs are defined as "the perception of negative events that have occurred during childhood […] are outside the control of the child, have the potential to impede or alter normal development, and cause harm or the potential for harm along with stress and suffering."(2 p.164) Examples of ACEs studied include, but are not limited to, the loss of a parental figure, parental substance abuse, child maltreatment, parental divorce, poverty, exposure to domestic and non-domestic violence, and parental incarceration.(3, 4). A recent national survey of youth, aged 0 -17 years old, revealed that 60.6% of children in the United States have directly experienced or witnessed victimization (e.g., maltreatment, violence, sexual assault) in a 12 month period prior to assessment.(5) This high prevalence of ACEs coupled with the immediate and long-term detrimental health effects ACEs can have over the lifespan make ACEs a serious public health issue that demands attention and strategies for primary and secondary prevention.

It is no surprise that children who experience adversities are at greater risk for emotional, cognitive, behavioral, and physical health problems. Research has shown that children who have been maltreated are more likely to be depressed and use illicit drugs and alcohol (6), have learning and behavioral problems (7), and have health complaints and illnesses that require a doctor (8). What is less obvious is the lifelong damage that ACEs can have on health and development. ACEs have been shown to be associated with a host of mental health outcomes well into adulthood, including depression, anxiety disorders, and Post Traumatic Stress Disorder.(9) It has also been argued that childhood trauma may also contribute to psychosis in adulthood (10). In addition, over the past decade evidence has been mounting that supports a relationship between CA and a number of adult physical health outcomes including heart disease (11), asthma (12), obesity (13), and cancer (14). Furthermore, recent evidence suggests that CA increases the risk of premature mortality. Brown et al. found that those who reported experiencing 6 or more ACEs were 1.7 times more likely to die before age 75 and 2.4 times more likely to die before age 65 compared to those who did not report experiencing ACEs. (15)

It can be said, then, with some confidence that both the mental and physical health of adults have foundations set in early life experiences. Accordingly, public health professionals and clinicians interested in disease prevention and treatment should consider both proximal and distal risk factors when investigating the etiological causes of disease with a particular focus on ACEs. Although the link between ACEs and adult health is well established, what remains unclear are the pathways or mechanisms by which ACEs impact health across the lifespan and for whom and under what social and environmental circumstances ACEs exact their greatest toll on health and well-being. How is it that the experience of violence in childhood manifests into cardiovascular disease or cancer in adulthood? Are the effects of ACEs on health the same for

those in higher versus lower socio-economic strata or different across race and gender? What proportion of mental, physical, and social disorders are attributable to ACEs? Are there pathways that can be identified and altered to reduce the burdens of disease caused by ACEs?

Many of the debilitating and chronic diseases prevalent in society today have their roots in early life. ACEs as social determinants of health affect health over the lifespan and across generations and must be studied both nationally and at the local level so that appropriate, targeted and effective prevention and intervention strategies can be implemented. In the review and study that follows, I describe CA in terms of child maltreatment (CM) and household dysfunction, two major types of CA, and discuss what is known about these adversities and how they impact adult health. I also describe the life course perspective (LCP), its major concepts, and timing models. The LCP is a theoretical framework that is often used to explain how the interplay of social, behavioral, biological and environmental exposures and contexts in early life impact health in adulthood.(16)

I use data collected from the 2009-2010 Allegheny County Health Survey (ACHS) to describe the lifetime prevalence of CA in a local population and in terms of social, demographic, and geographic characteristics. The overarching aims of this secondary analysis are to: 1) use behavioral risk and health surveillance data to describe and identify sub-populations at greatest risk for exposure to CA and determine what, if any, CA disparities exist in the population; 2) determine the *attributable risk* (AR) and *population attributable risk* (PAR) of adult disease and behavioral risk factors associated with CA; and 3) test behavioral and social pathways to determine which environmental, behavioral, and social factors are likely mediating and moderating the relationship between CA and adult health.

Results and findings are discussed using concepts of LCP to illustrate how CA affects adult health. The information derived from these analyses is meant to inform public health researchers and practitioners about: what health outcomes, as a result of CA exposure, are most likely to occur in the population; what level of reduction in adult morbidity could be expected if we prevent and treat CA; and what services or programs are most needed to prevent and mitigate the negative long-term health effects of CA. Moreover, the methods of data collection, analyses, and interpretation of findings presented in this study may serve as a model to be replicated in other states and localities. Through better surveillance and comprehensive analysis of ACE data, we can come to understand how CA is contributing to local trends and patterns of morbidity, mortality, and health disparities across the lifespan and generationally.

2.0 BACKGROUND

The impetus for much of the research on childhood adversity (CA) can be traced back to Henry Kemp's seminal article "The Battered Child Syndrome" which at the time placed child physical abuse into the realm of public concern. (17-19) Prior to Kemp's article, child abuse and other family adversities (e.g., domestic violence, or parental drug abuse or mental illness) were viewed largely as private matters of the family or as rare events typically involving families with severe mental illness.(18) Kemp's work helped to expose childhood maltreatment (CM) as a relatively common occurrence committed often by stable and mentally healthy parents. Kemp's research and its resulting impact on public discourse ultimately culminated into the first federal legislation aimed at the prevention and treatment of child abuse and neglect, namely the Child Abuse Prevention and Treatment Act (CAPTA) which was enacted in 1974. CAPTA included provisions for government financial support to investigate child abuse and neglect as well as fund activities and grants aimed at the identification, surveillance, and prevention and treatment of child abuse and neglect.

In the early 1990's Ronald Kessler et al. moved beyond the study of child abuse and neglect, and encompassing more types of childhood trauma and adversity (e.g. parental death or divorce, natural disasters, witness to violence) studied the long-term effects CA had on adult mental health.(4, 20) Using data from the National Comorbidity Study (NCS), Kessler et al. found that a large percentage of the general population, nearly 75%, had experienced at least one CA and approximately 51% had experienced more than one CA.(4) More interesting though was their finding that indicated an association between reported CA and adult depression such that those who had been recently diagnosed with major depression were approximated 2 times

more likely to have reported CA than those without major depression. Using data from the same study, Kessler et al. later showed that CA were consistently associated with adult mental health outcomes including mood disorders, anxiety disorders, and conduct and addictive disorders.(20) Kessler et al. concluded that the relationship between CA and adult health outcomes proved to be complex with multiple causal mechanisms. For example, many of the bivariate associations were attenuated and became non-significant when controlling for other CAs or for a history of mental illness. This suggested that CAs cluster together and interact with one another to produce health outcomes across the life course. Thus, studies that attempt to uncover the effects of a single adversity on a single health outcome are likely misguided given the interrelatedness and co-occurrence of adversity and illness. An example of this complexity and how adversities interact synergistically to cause health outcomes is apparent in the findings of a study conducted by Afifi et al.(21) They found that when both parental divorce and child abuse co-occurred, their joint effect on mental health outcomes was much stronger than the independent effects of each adversity alone.

One of the first and most cited studies supporting the relationship between childhood adversity and adult physical health was the Adverse Childhood Experiences Study (ACE Study) conducted by Felitti et al. in 1998.(3) In this prospective, longitudinal study, Felitti et al. examined the effects of adverse childhood experiences (ACEs) on a number of leading causes of death in a cohort of Kaiser Permanente patients. The ACEs examined in this study included family dysfunction and child maltreatment (including physical, mental, and sexual abuse). Results indicated that over half of the more than 9,500 participants reported at least one ACE and more than a quarter reported experiencing two of more ACEs. Moreover, there was a graded positive relationship between the number of ACEs reported and many of the adult health risk

factors studied, including smoking, alcohol use, and attempted suicide. The risk for "ever attempting suicide" for example, increased by a factor of 12 for participants who reported 4 or more ACEs compared to those who reported no ACEs. Similar graded associations were found between the number of ACEs reported and adult morbidities including cancer and heart, lung, and liver diseases.(3) Compared to those who reported no ACEs, participants who experienced 4 or more ACEs had a 2-fold increase in the odds for ischemic heart disease and nearly a 4-fold increase in the odds for chronic bronchitis/emphysema.(3)

Kessler's and Felitti's early work on childhood adversity demonstrated at least three things: first, childhood adversity is prevalent in our society with nearly 60-75% of the population having reported experiencing at least one adversity during childhood; second, many of the leading causes of death have their origins, at least in part, in early life experiences; and third, the relationship between childhood adversity and adult health is complex such that adversities cluster together and interact with health and other adversities to produce health effects that can span the life course. Since Kessler's work, numerous studies have been conducted, repeatedly showing a link between CA and adult mental and physical health. In addition to depression, CA is known to be associated with adult cardiovascular outcomes (22), obesity (23), suicide ideation and attempts (21), PTSD (24), substance abuse and dependence (25), chronic fatigue syndrome (26), and decreased cognitive functioning (27).

2.1 PREVALENCE OF CHILDHOOD ADVERSITY

Recent work suggests that ACEs continue to be quite prevalent in the U.S. and across the country. Estimates of lifetime exposure to CA were derived from the Centers for Disease

Control and Prevention (CDC) Behavioral Risk Factor Surveillance System (BRFSS). In 2009, five states administered questions inquiring about lifetime exposure to ACEs. The analysis of the combined dataset included responses from 26,229 adults and indicated that 59% of the sample experienced at least 1 ACE and nearly 9% experienced 5 or more ACEs.(28) The ACEs measured in this study included physical, mental, and verbal abuse, incarceration of household member, mental illness and substance abuse of household member, parental separation or divorce, and witness of domestic violence. The highest prevalence amongst the ACEs measured was household substance abuse at 29.1% and the lowest was incarceration of a household member at 7.2%. CM was reported by type with 25.9% reporting verbal abuse, 14.8% reporting physical abuse, and 12% reporting sexual abuse. This study, according to the authors, was the first population-based study to document the prevalence of ACEs in the general population across several states.

Whereas the estimates from the CDC BRFSS were based on adults reporting about childhood experience which may be difficult for adults to recall or remember accurately and which represent the prevalence of ACEs across many generations, the estimates provided by Finkelhor et al. (5) were reported by children and family members and represent current estimates of CA. Using data from a national population-based survey of children and adolescents, Finkelhor et al. found staggeringly high estimates of childhood exposure to violence; 86.6% of children age 0-17 had been exposed to violence, abuse, or crime at some point in their lifetime and 60.6% were exposed in the past year. Some types of victimizations reported in the past year included physical assault 46.3%, child maltreatment 10.2%, and sexual victimization 6.1%. (5) Given that this data was collected closer in time to CA exposure and

reflect data on a specific cohort, these estimates may be more reliable and indicative of current trends.

Outside of the studies described above, there is very limited data in the U.S on the prevalence of ACEs. Several states have begun to collect ACE data as an optional module when conducting their annual BRFSS surveys. For example Pennsylvania administered the ACE module in 2010, but has not done so since then (See Table 2-1). Much of what we know about the incidence and prevalence of CA comes from surveillance efforts measuring child maltreatment (CM). In the sections that follow the focus shifts to CM as this is the most commonly studied childhood adversity and thus allows for a comprehensive discussion about the effects of CA on health over the life course and across generations.

Table 2-1 Estimates and 95 Percent Confidence Limits of Adverse Childhood Events in the Adult Population:

Pennsylvania Behavioral Risk Factors Surveillance Survey, 2010

ACE measured	**Percent**	**Lower CL**	**Upper CL**
During Childhood They Lived with Someone Who was Depressed, Mentally Ill, or Suicidal	16	15	18
During Childhood They Lived with Someone Who Was a Problem Drinker or Alcoholic	19	18	21
During Childhood They Lived with Someone Who Used Illegal Street Drugs or Abused Prescription Meds	7	6	8
During Childhood They Lived with Someone Who Served Time or was Sentenced to Prison/Jail/Correction Facility	5	4	6
During Childhood Their Parents were Separated or Divorced	21	19	22
As a Child, Their Parents/Adults Slapped/Hit/Kicked/Punched/Beat Each Other Up 1+ Times in Their Home	15	14	17
Before Age 18, Their Parents/Adults Hit/Beat/Kicked/Phys. Hurt Them in Any Way 1+ Times in Their Home	14	13	16
As a Child, Their Parents or Adults Swore, Insulted or Put Them Down Once or More in Their Home	33	31	35
As a Child, They Had Someone At Least 5 Years Older Touch Them Sexually At Least One Time	9	8	10
As a Child, An Adult or Someone 5+ Years Older Tried to Make Them Touch Them Sexually	6	5	7
As a Child, They Were Forced to Have Sex 1+ Times With an Adult or Someone 5+ Years Older Than Them	4	3	4

2.2 CHILD MALTREATMENT DEFINITIONS

The terms “child maltreatment” and “child abuse” are often used interchangeably in the literature. For example, the World Health Organization defines both together. “Child abuse or maltreatment constitutes all forms of physical and/or emotional ill-treatment, sexual abuse, neglect or negligent treatment or commercial or other exploitation, resulting in actual or potential harm to the child’s health, survival, development or dignity in the context of a relationship of responsibility, trust or power.”(29) Although the two terms are used interchangeably, child abuse is sometimes used only to refer to acts of commission against a child such as verbal,

physical or sexual abuse and not acts of omission or child neglect. For this reason the more general and encompassing term, child maltreatment (CM), will be used here instead of child abuse.

Child maltreatment is defined broadly as "[A]ny act or series of acts of commission or omission by a parent or other caregiver that results in harm, potential for harm, or threat of harm to a child."(30) Acts of commission are what people most commonly understand to be child abuse. These acts of abuse are intentional and deliberate, even if injury is not a result, and cause harm, potential of harm, or threat to the child. Physical, sexual, and psychological abuse are all considered acts of commission. Similarly, acts of omission result in harm, potential for harm, or present a threat to a child, but in these cases it is not actions, per se, that cause harm or potential for harm to the child, rather it is the failure to act on behalf of the child's welfare. By failing to provide adequate supervision, medical needs, shelter, food, and/or education, harm or potential harm to the child is likely. All forms of neglect are considered to be acts of omission. Together acts of commission and acts of omission that result in harm or potential of harm or threat to the child constitute CM.

2.3 CHILD MALTREATMENT SURVEILLANCE AND DATABASES

United States surveillance data on CM comes typically from two sources, the National Incident Study (NIS) and the National Child Abuse and Neglect Data System (NCANDS) of the Children's Bureau. Prevalence and incidence rates provided below come from these two data sources, namely the NIS-IV 2006 and the NCANDS 2010. These datasets represent the most current data collection efforts. Both studies are national in scope, mandated by federal law, and

are conducted periodically or yearly so that trends and patterns in CM can be tracked. Briefly, the methods for the NIS-IV entail a representative sample of 122 counties across the U.S. from which a total of 126 Child Protection Service (CPS) agencies provided data on both reported and investigated cases of CM. In addition to CPS reports, a total of 10,791 professionals from 1,094 agencies provided 6,208 completed forms on cases of potential CM. These agencies include hospitals, police departments, schools, day care centers, and social service agencies and were selected because they likely have professionals that regularly or frequently come in contact with infants, children, and adolescents. The information gathered from these agencies and professionals adds to the CPS reports by providing information that might not otherwise be available given that many cases of CM are not reported to CPS agencies.

The methods for the NCANDS are based on the fact that all 50 states, mandated by federal law, require certain institutions and professionals to report suspected cases of CM to the local CPS agency. Reports on referral cases of CM are collected from CPS agencies across all 50 states on a yearly basis and provide aggregate and case level data including type of maltreatment, perpetrator and victim characteristics, and whether or not services were provided. Datasets are then compiled based on these records and analyzed to produce annual reports entitled Child Maltreatment which provide a national profile of child maltreatment.(31)

2.4 CHILD MALTREATMENT PREVALENCE AND INCIDENCE

According to the NIS-4, the estimated incidence for CM ranged from approximately 1.26 million (1 in every 58 children) to nearly 3 million (1 in every 25 children) depending on the how abuse and neglect were defined.(32) The lower estimate represents the Harm Standard, a strict

standard including only cases where harm to the child is evident. The higher rate represents the Endangerment Standard and includes all cases from the Harm Standard but also includes cases where harm was not yet evident. These cases are often identified and substantiated by child protection service providers, yet because harm to the child is not evident, they are not counted under the Harm Standard.

Since the NIS-3, conducted in 1993, the overall incidence rate of CM has decreased using the Harm Standard, and over the same time period no statistical change has been observed using the Endangerment Standard. The change in rate of CM using the Harm standard is significant, decreasing by 32%. Under this Harm Standard, specific types of abuse and neglect also showed significant decreases. There were significant decreases in the rate of sexual abuse, physical abuse, and emotional abuse; decreases of 23%, 27%, and 44% were observed respectively. Similar results were found using the Endangerment Standard; however there was a counterbalancing effect in that there were significant decreases in all types of abuse but significant increases in emotional neglect. Higher rates of CM, regardless of which standard, were observed amongst girls. Boys' rates of abuse decreased more than the girls' rates, and boys' rates of emotional neglect increased less so than the girls' rate.

The NIS-4 showed clear racial differences in the incidence of CM, with Blacks experiencing CM more than Whites and Hispanics. These racial differences were new and not present in any of the previous three NIS studies. Children with disability had lower rates of physical abuse and moderate harm resulting from abuse, but higher rates of emotional neglect and serious harm using the Harm Standard. Children with no parent in the labor force had 2 to 3 times higher rates of child abuse and neglect, respectively, compared to children with an employed parent.

Data from the NCANDS 2010 offers a slightly different picture.(33) Of 5.9 million alleged reports of CM in FY 2009, 3.3 million referrals were made to CPS. Of those, approximately 60.7% were screened in to be further investigated resulting in 436,321 substantiated claims of CM to at least one child. Another 24,976 cases of CM were suspected but either the investigation did not result in a substantiated case because the state criteria, developed via law and policy, was not met or evidence outside on any investigation determined that a child was in fact a victim of CM. In total approximately 461,297cases of CM were either substantiated or highly suspected in 2010. Given that reports to CPS could involve more than one child, the total number of victims of CM is much higher. The total number of unique victims in 2010 was 695,000 or a rate of 9.2 per 1,000 children. Decreasing by less than 1% from 2005 to 2010, this figure has remained relatively stable over the past 6 years indicating that not much progress has been made in prevention effort to curb CM. Furthermore the number of fatalities resulting from substantiated claims of CM has increased over the past 6 years from 1.94 per 100,000 children in 2005 to 2.07 per 100,000 children in 2010, indicating an increase in CM severity.

Neglect is clearly the most prevalent form of CM at 78.3 percent, followed by physical abuse at 17.6. Sexual abuse is next at 9.2 percent followed by psychological maltreatment at 8.1 percent and medical neglect at 2.4%.(33)

Child victim characteristics: Children age < 1 year had the highest rate of victimization at 20.6 per 1,000. In general the rate declines with increasing age, such that children age 1, 2, and 3, had victimization rates of 11.9, 11.3, and 10.6, respectively. African Americans had the highest rate of victimization at 15.1 per 1,000 and Asian had the low rate at 2.0 per 1,000. Whites had the second lowest rate at 7.8 per 1,000. Children with a disability (e.g., mental

retardation, emotional disturbance, medical condition, physical disability, or behavioral problems) are overrepresented and accounted for 11% of unique victims.

Regardless of the statistics used, child maltreatment (and more broadly childhood adversity) in the U.S. amounts to a serious and significant public health issue. The financial costs associated with CM in the U.S. are estimated to be $128 billion for 2008 and in sensitivity analyses could be as high as $585 billion.(34) The cost per new case of CM is estimated at approximately $210,000 for non-fatal cases and $1.3 million for fatal cases. (34) The intangible costs associated with the pain, emotional suffering, and a reduced quality of life as a result of CM are not represented in the statistics above, but should not be overlooked in planning CM prevention and treatment interventions as they represent a real cost to victims, their families, and to society as a whole.(35)

2.5 CHILD MALTREATMENT RISK FACTORS & DISPARITIES

Risk factors for CM exist at multiple levels starting with the child and extending up to the family, community, and society. Although the child victim is not to blame for the abuse, there are meaningful associations between characteristics of the child and CM. For example, age of the child and maltreatment are associated such that children under the age of 4 are at greater risk for maltreatment.(33) Having special needs which increase caregiver burden (e.g., mental illness, disability, or chronic health conditions) also places children at greater risk.(36) Gender is a risk factor for sexual abuse with adolescent females being at a much greater risk for sexual abuse than adolescent males.(33) Gay and lesbian youth may also be at greater risk for CM.(37)

Most CM risk factors pertain to characteristics of the family and/or the perpetrators of the abuse. The Centers for Disease Control identifies the following risk and protective factors for CM.(36) Risk factors pertaining to perpetrators of child maltreatment include: a of lack of knowledge regarding the needs of children and parenting skills; a history of being abused themselves as children; substance abuse and/or mental health issues; and socio-demographics such as young age, low income and/or education, single parenthood, and/or a large number of dependent children. Family risk factors include social isolation, family disorganization and violence, parenting stress, and poor parent child interactions. Risk factors at the community level include community violence, high poverty, and low social capital. Some protective factors that may buffer children from abuse and neglect have also been identified and include: supportive family environments, large social networks, nurturing parenting skills, parental employment, and access to health care and social services.

Many of the risk factors for CM involve sociodemographic characteristics of the victim and perpetrator. For this reason and others the proportion of maltreatment is not evenly distributed across the population. Most notably is the disproportionate amount of maltreatment experienced by children in low socioeconomic conditions. According to the NIS-IV, children in in low socioeconomic status (SES), categorized using household income, parental education, and participation in federal aid programs, had rates of CM 5 times that of those in the highest SES category. Disparities can also be found with regards to race, with a greater incidence of CM experienced by Black children when compared to White children. According to the NIS-IV, the rate of CM using the Harm Standard was 12.6 per 1000 for White children and 24.0 per 1,000 for Black children. The rates for Blacks were nearly twice that of Whites. As this was the first time in all of the National Incidence Studies that racial differences were found, a follow up analysis was conducted in an attempt to explain the racial disparities. The authors of this follow-up study

concluded that the disparities could be explained in part by the fact that Whites improved their SES more so than Blacks since the NIS-III, and that because CM is so strongly correlated with SES, SES differences between races were likely contributing, more so than race, to the observed disparities.(32) However, there were significant interactions effects such that CM racial disparities were non-existent in the low SES households, but pronounced in the "not-low SES households." The interaction between race and SES indicates that other factors besides SES, such as racism and discrimination may account for the observed disparities.

2.6 INTERGENERATIONAL EFFECTS OF CHILD MALTREATMENT

Intergenerational effects of CM are known to exist so much so that that a major risk factor for CM is a history of parental or caregiver CM.(36) Berlin, Appleyard, and Dodge studying prospective 499 mother infant dyads, found that a mother's physical abuse was related to the child maltreatment of their offspring.(38) Of children who had mothers that reported experiencing CM, 16.7% of children were maltreated compared to 7.1% of children with mothers who did not report histories of CM. Valentino et al. found similar results in a longitudinal study of 70 women followed from the third trimester of pregnancy until their child turned 18 years old. Children of mothers who had been abused as children themselves were nearly twice as likely to experience CM as compared to children with mothers who did not report being abused as children, 54.3%, versus 29.2%, respectively.(39) In both studies there was approximately a two-fold increase in child maltreatment associated with parental child maltreatment.

Studies of the intergenerational pathway of CM indicate that factors across multiple ecological levels play a role in the transmission and continuity of CM and its negative effects on

health and behavior. Braveman and Barclay propose a broad ecological model for understanding how health is shaped by social advantages and disadvantages over the life course and across generations.(40) This model has relevance to the transmission and continuity of CM in that CM can be viewed as a social disadvantage that can negatively impact health, education, and employment trajectories which in turn affect the next generation's risk for CA and CM. The main ideas in their model are: 1) health over the life course is dependent, to some extent , on health at other preceding stages of life; 2) health is determined by factors at multiple levels of the socio-ecological model (e.g., environmental conditions and economic and health policies); and 3) health and socio-ecological factors interact to produce social stratification by class and race, resulting in differential exposures, vulnerabilities, and consequences that lead to negative social and health problems that further stratify vulnerable populations. Exposed to the same level of adversity, children who live in high SES families with two parental figures with higher SES have better health outcomes and health trajectories then children growing up in low SES environments or divorced single mother families(41). Higher SES groups often have more resources and support (e.g., better schools, cohesive neighborhoods) to help cope with and overcome adversity.

Berlin et al. studied the relationship between parental CM and CM in their offspring and proposed that intergenerational CM was mediated by maternal mental health, social isolation, and social information processing patterns (e.g., aggressive cognitions). They found support for maternal social isolation as a mediator.(38) The explanation, they provide, is that women with little perceived social support or a small social network have less resources to help raise their children, resulting in an increased risk of CM. Because social isolation is a consequence of CM, it is likely a factor that explains, in part, transmission of CM across generations.

2.7 THE LIFE COURSE PERSPECTIVE

2.7.1 Life course perspective: definition, approach, and key concepts

The view of the social context interacting together with biological, behavioral, psychosocial, and genetic processes to affect health over the lifespan and across generations is best captured by the Life Course Perspective (LCP). Kun and Shlomo define the LCP as it relates to chronic disease epidemiology as "[…] the study of long-term effects on chronic disease risk of physical and social exposures during gestation, childhood, adolescence, young adult hood and later life. It included studies of the biological, behavioral, and psychosocial pathways that operate across an individual's life course, as well as across generations, to influence the development of chronic disease."(16 p.285) Developed in the 1960's by Glen Elder and others (42), the LCP "builds on recent social science and public health literature that posits that each life stage influences the next and that social, economic, and physical environments interacting across the life course have a profound impact on individual and community health. "(43 p.4) Health, from this perspective is not a status but rather a developmental process occurring over the lifespan.

Over the past two decades there has been renewed interest in the effects that early life environments, events, and experiences have on health and wellbeing over the lifespan.(44) Advances in genetics, biology, and social sciences have given researchers the tools and theoretical frameworks to better formulate and test hypotheses that include distal as well as proximal risk factors in explaining disease etiologies. Made prominent by Barker's work in the 1980's and 1990's (45, 46) on what would become known as the "fetal origins hypothesis" the idea that adult cardiovascular morbidity and mortality could be explained, in part, by in utero

environments and birth weight appealed to researchers from a multitude of disciplines as they struggled to explain the causes of disease from strictly an adult lifestyles approach.(47)

The purpose of the LCP "is to build and test theoretical models that postulate pathways linking exposures across the life course to later life health outcomes."(47 p.778) What are the pathways by which CA impacts adult health? How does neglect and emotional, physical, and sexual abuse "get under the skin" to produce heart disease, diabetes, and major depressive disorders in adulthood? How can we account for the social patterning of CA and resulting health disparities within and across generations? As indicated above there is a plethora of research that shows associations between CA and a variety of health outcomes, yet the complex ways in which early adversity interacts with social and environmental factors to produce health remains unclear.

Some hypothesize that physiological and biological pathways are responsible for the long-term effects of CA. (48, 49) For example, traumatic brain injury (TBI) which can result directly from "shaken baby syndrome" or other types of physical abuse is associated with numerous psychiatric and conduct disorders later in adolescence. Youth who have experienced TBI are more likely to be diagnosed with a psychiatric disorder, use alcohol and illicit substances, have criminal backgrounds, or attempt suicide.(50) These psychosocial issues can often lead to poorer academic performance, workforce placement issues, and lower SES status in adulthood. Taken together these social circumstances increase the risk for a broad spectrum of maladaptive behaviors and disease.

Others have studied behavioral and psychosocial pathways are mediators of long term health effects related to CA.(51, 52) From this perspective CA exerts great emotional and psychological distress resulting in chronic stress, emotional and psychiatric problems, and maladaptive behaviors, such as illicit drug use, alcohol use, overeating, and smoking which then

impact health. This perspective offers an explanation as to how non-physical types of abuse, such as neglect, produce health issues downstream.

Although proponents for these different pathways present competing arguments for the specific mechanisms responsible for the CM and adult health relationship, it is most likely that biological, psychological, and genetic mechanisms are working independently but also interactively and synergistically to produce health outcomes over the lifespan. In addition, because CM often occurs in low SES environments, additional and cumulative insults and risk factors e.g., violent and poor neighborhoods, risky families, deficient schools likely contribute to poor health trajectories.(53)

2.7.2 Key life course perspective concepts

Given that the LCP has roots in several disciplines, including epidemiology, sociology, developmental psychology, public health, and biology, terminology is not always used consistently or correctly in the literature. Furthermore, it is through a clear understanding of these LCP concepts that a framework for understanding how health is shaped over time and in the context of social and physical environments emerges.

Lifespan, lifespan development, and life course: The term lifespan is often meant to refer to the longevity of an individual organism and sometimes to mean "life expectancy" or the maximum number of years a person is expected to live.(54) *Lifespan Development* is a more complex term and is used to mean the "[m]ultidimensional and multidirectional processes of growth involving gains and losses , embedded in multilayered social and cultural contexts, involving dynamic processes of interaction of the developing organism with the social and physical environment." (54 p.3) Life course is defined by Clausen (cited by Alwin (54)) as a

progression through time from birth to death. It implies age related statuses and roles, life events, life transitions and trajectories, and the relation to adult disease. Changing social and physical environments as well as human agency are involved in the process of shaping the life course.

Birth cohort effects: based on the year of birth, the concept of birth cohort conveys the effect time and historical events have on social and physical environments which in turn can affect the health of a cohort years later.(16) Birth cohorts that experienced the Great Depression in their early years of life may show ill effects decades later due to increased exposure to impoverished physical and social environments.(42)

Trajectory: is used to mean the long term development of some aspect of a person's life over the lifespan.(16) For example, the trajectory of one's health refers to how health develops over the lifespan. A life trajectory is then the long term development of one life as specified by the sequence of life events, transitions, and roles experienced over the lifespan.

Life transitions: refer to short term changes that are embedded in trajectories and have effects on the physiological, psychological, or social state of the individual. (16) Life transitions include leaving home for the first time, becoming employed or retired, and taking on the role of a parent. Life transitions are often marked by *life events* such as the death of a spouse, graduation, or adopting a child.

Embodiment: "Describes how extrinsic factors experienced at different life stages are inscribes into an individual's body functions or structures." (16 p.778) Biological programming or "embedding" are related terms.

Plasticity: is the potential to change intrinsic characteristics when exposed to environmental stimuli. (16)

Susceptibility vs. vulnerability: Although often used interchangeably, the term susceptibility and vulnerability have slightly different meanings. Susceptibility refers to the situation where one embodies one of two interacting factors which make them more susceptible to the other factor. Vulnerability is a process of maladaptation in the face of adversity.(16) One is more susceptible to infection if one has a condition that decreases the immune system. One is more vulnerable to infection if one fails to practice good hygiene or safe sex practices.

Linked lives: Refers to the interrelatedness of lives through both synchronization and role sequencing.(42) Related to social networks and social support, the concept of linked lives expresses how relationships harbor both positive and negative effects. For example, the ill effects of racism experienced by a mother, directly and indirectly affect the child in negative ways via the relationship they share. The term "linked lives" expresses the idea that events and experiences of one person affect those of another person in the same network.(55) Parents' experienced racism and discrimination can lead to decreased opportunities in life such as lower salaries and wages, financial distress, and interpersonal and marital stress. This, in turn, can decrease the ability of parents to effectively care for their child and can increase the likelihood of maltreatment.

2.8 LIFE COURSE PERSPECTIVE MODELS

Three models are general discussed under the LCP: the critical/sensitive period model, the accumulation model, and the pathways or "chain of risks" model. Each model provides an explanation of how early biological, social, and environmental insults affect health across the lifespan. The models are presented here separately, and although the models are distinct in many

ways, they are also complementary and likely co-occur in etiological pathways to adult health outcomes.

2.8.1 Critical/sensitive period model

The critical periods model (also known as the latency model) is based on the premise that there exist periods in human development (critical periods) during which exposures to environmental or social toxins can have permanent effects on the trajectory of one's health. A critical period is defined as "a limited time window in which an exposure can have adverse or protective effects on development and subsequent disease outcome. Outside this developmental window there is no excess disease risk associated with exposure."(16 p.778) For example exposure to lead during infancy and early life can have grave consequences on the developing brain resulting in permanent damage and suboptimal trajectories in health. Similar levels of exposure during adolescence and adulthood may have no detrimental health effects as exposure occurrs outside the critical period. Power and Hertzman describe this model as "emphasizing the strong independent effects on health status late in life, of discrete events that tend to occur early in life."(56) From a critical periods model, then, the relationship between CA and adult health should largely be independent of both extrinsic events and conditions and intrinsic characteristics of the child. There is little to no *plasticity* from a critical period model perspective meaning there is little room for secondary prevention efforts.

The LCP has expanded the idea of critical periods to include sensitive periods which like critical periods are windows of time where exposures has a profound and lasting effect, but the effects are to some extent modifiable. That is, there exists some plasticity in the effects of exposures in sensitive periods, whereas there is no plasticity in the effects of exposures during

critical periods. Critical and sensitive periods are time windows where physical, emotional, or social exposures can alter the health trajectory of the individual. The expansion to include sensitive time periods has important policy implications as it shifts the focus back to primary, secondary, and tertiary prevention efforts occurring over the course of adolescence and adulthood rather than focusing primarily on in utero and early life when critical periods are many.

Cicchetti et al. provides evidence for adopting a critical period model in studying the effect of CM on later health outcomes.(49) They studied 553 children age 7-13 attending a summer camp with nearly half the sample having experienced CM and the other half not. They asked the research question: does cortisol regulation mediate the relationship between CM and depressive and internalizing symptomatology? In essence, they are testing the diatheses-stress hypothesis which is based on the premise that those who are genetically predisposed to affective disorders and who experience adversity are more likely to develop disease as a result. Because there are times in development where stress may impact the developing brain more so than other times, Cicchetti et al. studied the effect of maltreatment on internalizing and depressive symptoms as a function of the time the abuse occurred. They found that those who experience early abuse scored higher on depressive and internalizing behaviors than those that experienced maltreatment after the age of 5 or those that did not experience maltreatment. The relationship between timing of CM and internalizing and depressive was moderated by diurnal cortisol levels with early CM children having less of a decline in cortisol over the course of the day, indicating dysregulation of the HPA (Hypothalamic–pituitary–adrenal) axis, for early maltreated children and not for late or non-maltreated children.

These results indicate that timing of the abuse in relationship to developmental stage is an important factor to consider and that abuse experiences during sensitive periods when neurological structures are developing may set a child on a trajectory of poor health. The strengths of this study come by way of design and measurement. The addition of the control group rules out many types of bias. Additionally, they used the validated maltreatment classification system (MCS) which utilizes Department of Human Service records to derive categories and severity of maltreatment. Most contributory to life course research on CM is their emphasis on timing of abuse. By including this variable in their analyses, it made it possible to test the interacting effects of life stage and adversity. Their work also raises a host of biological and genetic based questions concerning experience of CM in conjunction with age-related processes of human development. These contributions outweigh the limitations of this study. Limitations include 1) the study was conducted with a low SES group of children so the results may not be generalizable to other groups and 2) that other adversities were not controlled for and thus could account for the differences in HPA dysregulation.

Even when controlling for other adversities and adult health behaviors (such as smoking alcohol use, BMI, and physical activity) Fuller-Thomson and Brennenstuhl found an independent relationship between CM and cancer.(14) Those with CM had 49% higher odds of having a diagnosis of cancer than those who did not. This was a large population based cross-sectional study conducted in Canada with 13,092 adolescents and adults age 12 and older. Limitations were many in this study and include self-report of cancer and cross-sectional data which limited their ability to establish the direction of associations. This study provides evidence against the behavioral pathways often suggested for linking abuse to cancer as they controlled for many of the known behavioral risk factor for cancer and still the relationship between CM and cancer

remained. They suggest that HPA axis dysregulation and resulting immunosuppression may explain the CM cancer relationship.

2.8.2 Accumulation model

The Accumulation model proposes that those who have experienced multiple types of CM over longer periods of time, through multiple life stages will fare worst then those who experience one type of CM to a lesser degree and only during adolescence. In this model, it is not only adversities of the same type that can accumulate (e.g., physical abuse over a 5 year period) but all adversities, including loss of a loved one, parental divorce, and growing up in a low SES environment. Both risk and protection can accumulate in synergistic or additive ways to either increase or decrease the risk of disease. Kun describes different ways that risk accumulates to cause health outcomes.(47) The *accumulation model with risk clustering* is an accumulation model that describes a social, environmental, or biological root factor that increases the risk to multiple other risk factors, each which independently increases the risk of disease. Socioeconomic status is an example of a root factor that increases or decreases risk to a host of other factors. Another model that Kun describes is one where each risk is unrelated to the others and independently increases the risk of disease. For example, experiencing a natural disaster and CM are unrelated adversities, but both risks accumulate to increase overall risk, independently.

Jonson-Reid, Kohl, and Drake studied the effect of CM chronicity to determine how repeated exposure to maltreatment affects health in adulthood.(57) They tested the "dosage effects" of CM and found that as the number of CM reports increased, the percentage change of experiencing one or more negative outcomes increased in a linear fashion. Data were derived from a dataset that comprised of a total of 5,994 children, 59% who were reported CM and 41%

who did not (comparison group). Children were followed longitudinally for approximately 16 years. The dose relationship was attenuated or altogether non-significant when controlling for other advertises (childhood head trauma, STDs, and/or suicide attempts) indicating that the accumulation of adversity is likely responsible for the observed increase of disease.

Weirsma et al. (58) asked the question - is childhood maltreatment and other childhood adversities related to chronicity of depression in adults? Using Cross-sectional data from a depression anxiety study in the Netherlands (n=1,230) they found that CM, and not simply adversity, was associated with increased risk of depression chronicity. Those who reported abuse and neglect that occurred often or very often had a three-fold increased risk for chronic depression. Lesser abuse and neglect was associated with increased risk but non-significant. These findings, too, supports the notation that more than exposure alone, dose and duration are important in understanding CA related disease risk.

2.8.3 Pathways model

Under the pathways model, health outcomes later in life are a result of "chains of risk" whereby exposure to one risk factor early in life increases the probability of exposure to other risks in adolescence and then still others in adulthood. This model differs from the accumulation model in that each risk is related in such a way that one increases the probability of the other forming a "chain of risks." Through this probabilistic chain of risks, disease may not be the result until the final link of the chain is connected. Kuh refers to this type of phenomenon as the "trigger effect."(47) For example, CM increases the risk of anti-social peers, which increases the risk of alcohol and drug use which increases the likelihood of risky sexual behaviors which can be considered the "trigger" for HIV and other STIs. By determining what "chains of risks" form as

a result of CM, we can develop effective interventions targeting the links in the chains that increase significantly the occurrence of disease. The other "chain of risks" model Kuh discusses is similar to the accumulation model in that each risk independently increases the risk of disease, but here the risk are also related in a probabilistic manner.(47) For example, being exposed to child abuse and neglect increases the likelihood of poor school performance, which in turn increases the likelihood of high-school dropout and poor job opportunities, which increases the likelihood of lower income, lack of resources, and poorer living conditions. These factors are related in a probabilistic way, yet they also independently increase the probability of morbidity and mortality over the lifespan. The pathways model is different from the critical period model in that it is not a deterministic model but rather a probabilistic one. That is each exposure does not guarantee the occurrence of another exposure or disease state rather each exposure only increases the probability of another exposure or disease state. Child abuse increases the probability of poor school performance, it does not, however, determine school performance.

2.9 MODERATORS, MEDIATORS, AND MECHANISMS

2.9.1 Environmental factors

What environmental factors moderate the relationship between CA and adult health outcomes? Both physical and psychosocial environments in which children live, play, grow, and learn present resources and additional health threats that can alter the trajectory of recovery, resilience, and health and wellbeing across the lifespan. For example, children who are maltreated and who attend schools that have supportive teachers, quality after school programs, and quality

education, may fare much better in terms of academic performance, overall health, and post high school employment opportunities than maltreated children who attend schools that lack order, have few extracurricular activities, and do not provide quality education. Maltreated children are more likely to develop cognitive, conduct, and behavioral disorders as a result of abuse and neglect than non-abused children. In the absence of a strong school environment, these cognitive and behavioral disorders can be exacerbated and lead to additional problems in other areas of life such as in developing friendships or strong social ties. These examples most closely resemble the accumulation model or the pathways models of the LCP discussed above, in that CM in addition to related and unrelated adversities such as poverty (which decreases access to quality education) additively produce worse outcomes in both the short and long term.

Using data from the longitudinal studies of child abuse and neglect (LONSCAN) Yonas et al. found that neighborhood collective efficacy (operationalized as the caregivers' perceived level of participation of neighbors to create a close, responsible, and accountable neighborhood) moderated the effect of child neglect on externalizing behavior problems at age 12.(59) That is, children who were neglected and who lived in neighborhoods perceived to be high in collective efficacy, endorsed similar levels of externalizing or aggressive behavior than those children who were not neglected, and significantly better than neglected children in neighborhoods with low collective efficacy. One possibility that may explain these findings is that in a neighborhood with low collective efficacy, there is less parental supervision and communication between parents about their children's activities and behaviors resulting in increased externalizing behavior. Maltreated children who grow up in neighborhoods with low collective efficacy may find it easier to align themselves with antisocial peers due to shared attitudes towards violence and the mere proximity of their dwellings. Herrenkohl et al. investigated the mediating pathways

from child abuse to adolescent violence and found that attitudes toward violence and preference toward antisocial peers mediated the relationship between CM and violent behavior.(60) The case can be made, then, that maltreated children may be protected to some extent from delinquency and externalizing and violent behaviors if the neighborhoods in which they live are high in perceived collective efficacy.

The family environment, and particularly, the relationships among family members, is also important with regards to the developmental and health trajectories of maltreated children. Bifulco et al. conducted a study to examine the extent to which attachment styles mediated the relationship between CM and adult depression and anxiety.(61) Attachment styles are typically formed early in life and are dependent on the type of relationships children have with their caregivers. Bifulco et al. found that in their study of 154 community-dwelling high risk women, the relationship between CM and adult depression and anxiety was partially mediated by highly fearful and highly angry-dismissive attachment styles in adulthood. These findings stress the important of the family environment and parental nurturing in the CM-adult health pathway.

Fletcher, using data from the ADD Health (National Longitudinal Study of Adolescent Health) dataset, asked the question – is the relationship between CM and adult depression moderated by or confounded by community, school, and family level factors?(62) Fletcher, found that school, family and community all effect the CM-adult depression. relationship; however family level factors such as married parents, family income, maternal education appeared to moderate the effect more so than school or neighborhood factors. Also the effects of CM on depression increased with age providing evidence for the accumulation of adversities and "chains of risk" models. This study is limited in that it focuses on one specific outcome and relied heavily on self-report, yet overall this study provides evidence that accumulation of stress

and adversity impact health well into adulthood and that family level and community level factors play a significance role in the CM adult depression relationship.

2.9.2 Behavioral and psychosocial factors

Rohde et al. tested behavioral pathways by which CM may affect obesity and depression in adulthood.(52) Specifically, using cross sectional data from a population based survey of 4,641 middle-aged women, they asked whether or not CM status was associated with obesity and depression in adulthood. They also tested whether or not CM explained, in part, the relationship between obesity and depression and if body dissatisfaction and binge eating explained the relationship between CM and obesity. Results indicated that child abuse was associated with both depression and obesity in adulthood, although the mediated pathways were not supported. The relationships between CM and binge eating and body dissatisfaction were attenuated but not significantly. Results supported, to the extent possible, the independent effect that CM has on obesity and depression.

Other behavioral pathways examined in the CM adult health relationship include health care utilization. Chartier, Walker, and Naimark studied the relationship between child physical sexual abuse on physical health problems and health care utilization.(63) They found significant moderate positive associations between CM and multiple physical health indicators such as pain, disability, and self-reported health and frequency of general practitioner, emergency room, and professional use. Chartier et al., in a 2009 study, examined CM in relationship to health risk behaviors and adult disease and wanted to determine if health risk behaviors such as smoking and alcohol use mediated the relationship between CM and adult heath and health care utilization.(51) Findings supported the long term effects of CM on health and health utilization.

In addition, CM was related to health behaviors which were also found to mediate the relationship between CM and adult health when they were coupled with mental health problems. Alone, however, the health risk behaviors only attenuated the relationship. These results emphasize the need to look at how clustering of negative health behaviors with mental and physical disorders coexist in vulnerable populations to increase the burden of disease.

2.9.3 Socio-demographic factors

The effect that CM has on adult health varies depending on the type and timing of CM, other adversities experienced, and numerous mediating factors, some of which were discussed above. The effect may also be modified by age, gender, and socioeconomic status across the lifespan. Even though some of these factors are not modifiable (e.g., age and gender) identifying which characteristics are most associated with negative adult health outcomes becomes important for determining who is at most risk, identifying what the resilience factors are, and what outcomes are most likely given particular socio-demographics. This information greatly increases our ability to target appropriate prevention and interventions strategies.

Batten, Maciejewski, and Mazure used data from the U.S. National comorbidity Study (Part 2) to ask whether or not gender differences in depression and CVD could be explained, in part, by histories of CM.(11) Using Multiple Linear Regression and controlling for age, ethnicity, marital status and income, they found strong relationships between CM and depression for both genders and between CM and CVD for women only. The CM-depression relationship was significantly stronger for men than for women. Also for men, a relationship between CM and CVD was noted but in the opposite direction than expected and non-significant. Compared to women who did not report CM, women who reported CM were 8.8 times more likely to have

CVD. In addition to the descriptive analyses, they tested whether or not a history of depression "mediated" the relationship between CM and CVD and found no evidence for this. Although formal mediational analysis did not take place, the results suggest that independent of depression, CM impacts CVD risk. Limitations of this study include: a one-item measure of CM, self-report of both physical and mental health conditions, and a cross-sectional study design. However the gender differences are striking, with the gender difference typically associated with CVD (i.e., women have lower incidence men) being wiped away in the presence of CM. Further analyses are needed to determine the different gender pathways to disease; however the gender differences in this study emphasize the need to conduct stratified analyses to parse differential effects based on gender.

Rangel et al. studied the racial and SES differences in infant mortality after non-accidental trauma at nine pediatric trauma centers across the U.S.(64) Five years of data were extracted from trauma registries based on abuse status. Insurance status, injury severity, and coma scales were also extracted along with demographic variables. Results indicated that insurance status and being from a low income area, not race, predicted mortality when controlling for the other factors. This study suggests that factors in the social and physical environments of the infants may be contributing to a weaker baseline level of health resulting in an increase in mortality. It could be that social or cultural biases are at play such that discrimination may result in decreased attention or care for those with low SES status. Access to care for low SES groups and longer response times for emergency services in rural areas may account for some of the increased mortality. The findings of this study highlight the need to parse the effects of SES, race, and CM on health outcomes.

Shaw et al. used data from MIDAS study to examine the life-time effects of poor parental support on adult alcohol abuse.(65) The aims of the study were to determine what effect parental support had on alcohol abuse across age groups and whether or not the functional form of the relationship was linear or curvilinear. In a linear functional form even small deficiencies in parental support would be associated with increases in alcohol abuse. Shaw et al. also investigated whether or not the effects of poor parental support persisted into old age or whether or not age and parental support interacted in a way that with increasing age the effects of poor parental support on alcohol abuse would diminish. They found a relationship between maternal support and alcohol abuse. The functional form of this relationship was found to be linear with even small deficiencies in maternal support conferring an increase in the odds ratio for substance abuse. The interaction between age and maternal support was non-significant and therefore the inference can be drawn that regardless of age, the effects of poor parental support confer an increased risk for alcohol abuse. These findings are consistent with the critical period model in so far as a lack of parental support during early childhood, "a critical period" for the development of a sense of security which facilitates interpersonal closeness through-out the life course, affects health outcomes independent of age. Conversely, the pathway models may better explain these findings in that exposure to unsupportive parenting environments may foster withdrawal and avoidance pattern of socialization in early and later childhood; the latter leading to an increased risk of alcohol abuse. Determining which model is best is not as important as using the models to understand the complex pathways and mechanisms that may be occurring.

2.10 SUMMARY

Childhood adversity (CA) is a significant public health issue affecting millions of children and adults each year. Both the immediate and long-term costs, in terms of health and dollars, present society with challenges to: a) better monitor CA prevalence and incidence in populations; b) develop and evaluate sound interventions that prove effective in primary and secondary prevention of CA, and c) target those in our society that are most at risk for CA and the accompanying negative health consequence. The LCP provides a framework for investigating the CA pathways that lead to adult disease and for understanding how disparities as a result of CA emerge and are sustained in vulnerable populations within and across generations. Although much is known about the prevalence of CA in the population and the correlations CA has to adult mental and physical health outcomes; less is known about the pathways by which CA impacts adult health and if the effects of CA on adult health vary as a function of age, gender, race, SES, and neighborhood characteristics. Social, behavioral, and biological mechanisms have all have been proposed as intervening factors in the CA adult health relationship, yet more research is needed to understand what factors (e.g., lifestyle factors, education, employment, social cohesion) may be leveraged to mitigate and prevent CA.

3.0 RESEARCH AIMS

The overarching aim of this study is to describe the prevalence and health effects of childhood adversity (CA) in a local population-based sample of adults and to evaluate possible intervening factors that affect the CA adult health relationship. Data collected from the 2009-2010 Allegheny County Health Survey (ACHS) are used to address the specific aims below and test the subsequent hypotheses.

Specific Aim 1: To estimate and describe the prevalence of CA in terms of type and frequency and by social, demographic, and geographic characteristics.

- Hypothesis 1.1: Compared to non-Hispanic Whites, non-Hispanic Blacks have a higher lifetime prevalence of CA.
- Hypothesis 1.2: Compared to males, females have a higher lifetime prevalence of CA.
- Hypothesis 1.3: Compared to high SES individuals (measured in terms of household income, employment status, and education), low SES individuals have a higher lifetime prevalence of CA.
- Hypothesis 1.4: Compared to employed individuals, those unemployed or unable to work have a higher lifetime prevalence of CA.
- Hypothesis 1.5: Compared to those without disability, disabled individuals have a higher lifetime prevalence of CA.
- Hypothesis 1.6: Compared to geographic areas with low unemployment, areas with high unemployment have a higher lifetime prevalence of CA.

- Hypothesis 1.7: Compared to geographic areas with high proportion of non-Hispanic Whites, areas with a high proportion non-Hispanic Blacks have a higher lifetime prevalence of CA.
- Hypothesis 1.8: Compared to geographic areas with a low proportion of individuals living below the poverty line, areas with a high proportion of individuals living below the poverty line have a higher lifetime prevalence of CA.

Specific Aim 2: To describe the adult health risks associated with CA and quantify the proportion of disease in the adult population attributable to CA. The analyses here are descriptive and exploratory in nature and thus there are no *a priori* hypotheses. These analyses will quantify the odds of disease given exposure to adverse childhood events (ACEs) and estimate the excess morbidity in the population associated with CA.

Specific Aim 3: To evaluate the roles of demographic, social, behavioral and geographic factors in the relationship between CA and the pathogenesis and development of adult disease. All hypotheses below are subsumed in the model depicted in Figure 1.

- Hypothesis 3.1: Adult SES (a composite measure of achieved level of education and household income) mediates the relationship between CA and adult health outcomes.
- Hypothesis 3.2: Perceived social support mediates the relationship between CA and adult health outcomes.

- Hypothesis 3.3: Health behaviors (i.e., fruits and vegetables consumption, alcohol and tobacco use, obesity, physical activity) mediate the relationship between CA and adult health.
- Hypothesis 3.4: Age moderates the relationship between CA and adult health risk behaviors (e.g., smoking, alcohol use, consumption of fruits and vegetables) such that younger age groups will have increased odds of behavioral risks and morbidity in adulthood.
- Hypothesis 3.5: Gender moderates the relationship between CA and adult health risk behaviors such that females who have experienced CA have increased odds of behavioral risks and morbidity in adulthood.
- Hypothesis 3.6: Childhood SES (measured as parental education) moderates the relationship between CA and adult health outcomes such that those with lower childhood SES who also have experienced CA have an increased odds of behavioral risks and morbidity in adulthood.
- Hypothesis 3.7: Race moderates the relationship between CA and adult health such that African Americans who have experienced CA have an increased odds of behavioral risks and morbidity in adulthood.
- Hypothesis 3.8: Current neighborhood quality (measured as neighborhood walkability environment, availability of healthy foods, and social cohesion) moderates the relationship between CA and adult health such that neighborhood quality interacts with mediators described above to alter the relationship between CA and adult health.

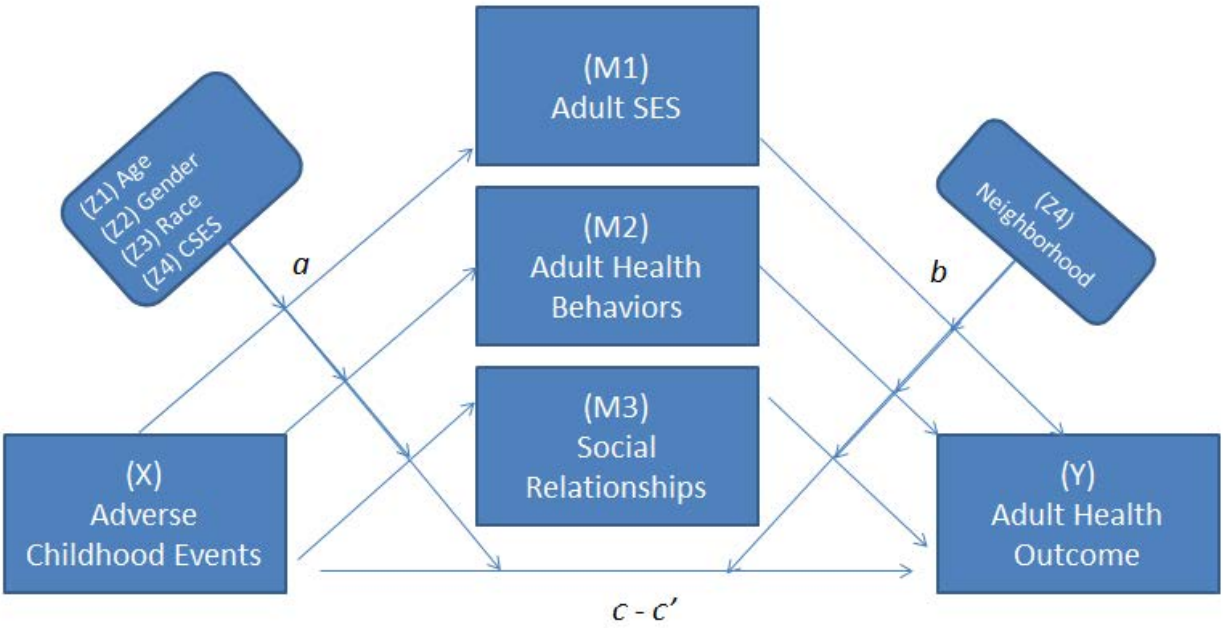

Figure 3-1 Analytic Model of How Childhood Adversity Affects Adult Health

4.0 RESEARCH DESIGN AND METHODS

4.1 SOURCE DATA

The primary data for this study comes from the 2009-2010 Allegheny County Health Survey (ACHS).(66) Modeled after the Center for Disease Control's Behavioral Risk Factor Surveillance System (BRFSS), the ACHS was designed to collect generalizable health and behavioral risk factor data from adult residents of Allegheny County, Pennsylvania. Although the Pennsylvania Department of Health conducts the BRFSS study annually across the state; the number of interviews conducted in Allegheny County is insufficient to conduct subpopulation analyses within the county. In response to this limitation and with a goal of collecting community specific health data for the purposes of developing and guiding local health policy, interventions, and prevention efforts, the Allegheny County Health Department (ACHD) and the Graduate School of Public Health (GSPH) at the University of Pittsburgh partnered with local organizations to conduct the 2009-2010 ACHS. Under contract with the ACHD, the survey was conducted by the Evaluation Institute's Office of Health Survey Research, which is housed in the Department of Behavioral and Community Health Sciences, GSPH. What follows is a brief description of the target population and sampling, data collection methods, relevant survey measures, and data imputation and weighting methods. Ethical issues associated with data collection are also briefly discussed.

4.1.1 Target population

The target population for the ACHS was the adult resident population, age 18 and older, of Allegheny County, Pennsylvania. According to the 2010 Census (67) there were a total of 1,223,348 people living in Allegheny County of which 981,685 were adults, age 18 or older. The population in 2010 was predominantly White (82%) with a greater percentage of females (52%). Approximately 19,070 Latinos or Hispanics lived in Allegheny County making up less than 2% of the population. The median age was 41.3 years old.

4.1.2 Sampling methods

A disproportionate stratified sampling strategy was employed to increase efficiency and to ensure adequate representation of African Americans and those with lower household incomes. Using data from the 2007 Claritas survey for Allegheny County, census tracts were stratified into those with greater or equal to 50% African American composition and those with less than a 50% African American composition. Census tracts were then mapped to telephone exchanges, and those census tracts that had greater than a 50% African American composition were oversampled. The same procedure was conducted to oversample telephone numbers from census tracts that had greater than 50% of households with incomes below the county's median household income of $40,000. Further stratification of the sampling frame occurred to increase efficiencies and the likelihood of contact with households. The ACHS researchers sampled blocks of 100-series telephone banks known to have at least 1 listed residential number (1+ listed banks) at a higher rate than unlisted banks. The 1+ listed banks are known to have a higher incidence rate (i.e., more likely to reach a household rather than a non-working number) and thus

increase efficiency in contacting households.(68) Sampling also occurred at the household level when households had more than one eligible adult resident. After the interviewer enumerated the adult residents by gender, a randomly selected respondent was chosen to be interviewed (e.g., the second oldest adult male). Only the identified person could be interviewed; no proxy interviewing was permitted.

Eligibility criteria for the ACHS existed at both the household and the individual level. A household was eligible to participate if it was located in Allegheny County and if it was a primary residence. Vacation homes (i.e., homes that are occupied for less than 20 days per year), group homes (e.g., shelters, and sororities), and institutions (e.g., college dormitories and nursing homes) were excluded from the sample and if contacted were considered ineligible. An individual was eligible to participate if he/she was at least 18 years of age and resided in Allegheny County at the time of the interview.

4.1.3 Data collection methods

The ACHS employed of random-digit-dialing (RDD) sampling strategy to generate a representative sample of adults residing in Allegheny County, PA. The telephone survey, conducted between August 2009 and September 2010, was administered by trained interviewers using a computer assisted telephone interviewing (CATI) system. With skip patterns, the survey consisted of approximately 215 questions and took respondents, on average, approximately 35 minutes to complete. Topics included in the survey consisted of both BRFSS core and optional modules (e.g., health care coverage and access, health status, diabetes, heart disease, hypertension, smoking, alcohol consumption, immunization, and adverse childhood events). In addition, community partners who supported the ACHS added questions to the survey particular

to their areas of interest and service. Some of the questions added by partners included social support items, parental education as a measure of childhood socioeconomic status, and neighborhood level measures, such as social cohesion, walkability, and availability of fresh foods.

To reduce non-response and to ensure that selected households were given a sufficient opportunity to participate, a maximum of 15 call attempts were made to each telephone number in the sample. Calls that resulted in a "soft refusal" [e.g., when the respondent simply hung-up without responding or only they only responded that they were "not interested"] were called until a second refusal was received or the 15 call maximum was reached. If a selected household respondent refused to participate following informed consent or if an irate response was received by the interviewer, additional phone calls to that household were suspended. In addition to multiple call attempts, the contact rate was increased by varying the day and time of call attempts. Following the CDC BRFSS calling rules, at least 2 call attempts were made during each weekday day (9am-5pm) and evening (5pm-9pm) time periods and at least 2 calls were made during the weekend.

4.1.4 Data imputation and weighting

Post data collection, design weights were calculated to adjust for the disproportionate stratified sampling design. For example, because communities that were predominantly African American were oversampled to increase the overall number of African Americans in the sample, weights were applied to participants from those oversampled area to adjust for the increased probability of selection. Probability of selection weights were also added to account for households that had more than one landline phone number and for households that had more than 1 eligible adult.

Post-stratification weights were included to adjust for differential nonresponse and non-coverage via iterative-proportional-fitting. This process ensures that the weighted distributions of the sample match those of the target population. Prior to post-stratification weighting, missing data for several demographic variables (i.e., age, race, education, household income) were imputed using a "hot deck" imputation method. The "hot deck" method uses data from known sample elements to impute values for missing data. Imputation was done for only those variables that were needed to calculate post-stratification weights, namely age, race, and education. Additionally imputation for household income was performed because a large percentage (15%) of the income data was incomplete. The result of these two procedures ensured that every record in the data set had a final weight and that those weights could be applied to the sample in order to make statistical inferences about the target population.

Age, Race, and Education were imputed using the "hot deck" method although the proportion of missing cases was negligible, 0.2%, 2.1%, and 0.3%, respectively.

4.1.5 Final Sample

The overall response rate of the ACHS was 29% and the cooperation rate was 66.1%. Response rates were calculated using the formulas used for calculating CDC BRFSS response rates.(69) Overall, females, older adults, and African Americans were overrepresented in the sample.

Table 4-1 Final Sample Allegheny County Health Survey

Distribution of the 2009–2010 ACHS Sample and Adult Allegheny County Population Data for Selected Characteristics

		2009–2010 ACHS Sample		2010 Allegheny County	
		No.	%	No.	%
	All Adults	5,442	100.00	981,685*	100.00
	Male	1,790	32.89	462,137*	47.08
	Female	3,652	67.11	519,548*	52.92
	Age:				
	18–29	399	7.33	208,582*	21.25
	30–44	1,010	18.56	218,474*	22.26
	45–64	2,241	41.18	349,570*	35.61
	65+	1,792	32.93	205,059*	20.89
	Race:				
	White	4,259	78.26	863,532**	86.29
	Black	1,058	19.44	107,399**	10.73
	All Other	125	2.29	29,827**	2.98
	Latino Origin:				
	Yes	78	1.43	8,244**	0.82
	Non-Hispanic	5,364	98.57	992,514**	99.18

*Indicates Census 2010 data.
**Indicates Census 2000 data.
NOTE: Race data includes Latinos.

4.1.6 Ethical considerations

The ACHS was approved by the University of Pittsburgh's Institutional Review Board. Informed verbal consent was received from all respondents. Prior to data collection respondents were told that information collected would be anonymous and that their participation in the research was voluntary. To maintain anonymity, personal identifiers (e.g., names and phone numbers) were not linked to collected survey data. Participants were not remunerated for their participation and were free to terminate the survey at any time during the interview

4.1.7 Survey measures

All of the variables relevant to the analyses proposed herein were first examined in terms of their raw and weighted frequencies and percentages. Based on descriptive statistics, many variables were recoded to: 1) facilitate statistical computation by collapsing categories when there are too few observations in any one category; 2) create composite scores for ACE scores, SES measures, and neighborhood measures; or 3) simplify interpretation and reporting (e.g., age was recoded from a ratio to an ordinal scale to facilitate reporting). Descriptive statistics were also produced to verify that values were within expected range and that skip patterns were executed properly. Consistencies checks were performed to ensure variables were logically consistent within subject (e.g., checks were made to verify that males were not reporting cervical cancer screenings).

CA is the **main independent variable** in the analyses discussed below. CA was measured in the ACHS using a modified version of the ACE scale which was originally developed by Felitti et al. and used in both the ACE and CDC BRFSS surveys. The original ACE scale was developed using items from several reliable and valid scales including the Conflict Tactics Scale (70) which is used to measure physical and emotional abuse, the Childhood Trauma Questionnaire (70) which is used to measure neglect and household dysfunction, items from Wyatt (71) which measure exposure to sexual abuse, and items from Schoenborn (72) which measure parental substance abuse. The ACE scale used in the ACHS excluded several questions from the original ACE scale, and several questions were modified (See Table 4-2). The ACHS ACE scale did not include questions about physical and emotional neglect, parental divorce, or parental incarceration. Another major difference is that and in the original ACE scale the question about sexual abuse asked about victimization from “any adult or person 5 years older than you” whereas the ACHS question asked about victimization from “a

parent or adult living in your home." The ACHS variable for sexual abuse is unique in that it provides a measure of "in home" sexual abuse, however, most surveys that have used the ACE scale ask the broader question. This makes the ACHS ACE estimates difficult to comparable to other national and state estimates as the ACHS estimates excluded a great number of sexual abuse events that occurred outside of the home.

Table 4-2 Comparison between Original ACE Questions and ACHS ACE Questions

ORIGINAL ACE SCALE	ACHS ACE SCALE
While you were growing up, during your first 18 years of life:	**When you were growing up…**
1. Was a household member depressed or mentally ill, or did a household member attempt suicide?	1. Was anyone living in your home depressed, mentally ill, or suicidal?
2. Did you live with anyone who was a problem drinker or alcoholic or who used street drugs?	2. Did you live with anyone who was a problem drinker, alcoholic or drug user?
3. Did a parent or other adult in the household often or very often… Push, grab, slap, or throw something at you? or Ever hit you so hard that you had marks or were injured?	3. How often did a parent or adult living in your home hit, beat, kick, or physically hurt you?
4. Did a parent or other adult in the household often or very often… Swear at you, insult you, put you down, or humiliate you? Or Act in a way that made you afraid that you might be physically hurt?	4. How often did a parent or adult living in your home swear at you, insult you, or put you down?
5. Was your mother or stepmother: Often or very often pushed, grabbed, slapped, or had something thrown at her? or Sometimes, often, or very often kicked, bitten, hit with a fist, or hit with something hard? or Ever repeatedly hit at least a few minutes or threatened with a gun or knife?	5. How often did a parent or adult living in your home push, grab, slap, or throw something at your mother?
6. Did an adult or person at least 5 years older than you ever… Touch or fondle you or have you touch their body in a sexual way? or Attempt or actually have oral, anal, or vaginal intercourse with you?	6. How often did a parent or adult living in your home touch you sexually or try to make you touch them sexually?
7. Did you often or very often feel that … No one in your family loved you or thought you were important or special? or Your family didn't look out for each other, feel close to each other, or support each other?	
8. Did you often or very often feel that … You didn't have enough to eat, had to wear dirty clothes, and had no one to protect you? or Your parents were too drunk or high to take care of you or take you to the doctor if you needed it?	
9. Were your parents ever separated or divorced?	
10. Did a household member go to prison?	

The **main dependent variables** for the analyses included adult physical, mental, social, and behavioral health indicators (See Table 4-3). Physical health indicators were derived primarily from the CDC's BRFSS survey and included measures of General Health, Physical

Health Related Quality of Life (PHQoL), Cancer, Diabetes, Hypertension, High Cholesterol, Cardiovascular Disease, Asthma, Overweight and Obesity, Cancer Screening, and Venereal Disease. General Health was measured by asking respondents to rate their health as excellent, very good, good, fair, or poor. PHQoL was measured by asking respondents about the number of days in the past 30 days their health was not good. Greater or equal to 15 poor health days in the past month was used to recode the variable into high and low PHQoL. Cancer, Diabetes, Hypertension, High Cholesterol, Cardiovascular Disease, and Asthma, were all measured by asking the respondent if they were ever told by a doctor or health care professional that they had the condition. Overweight and Obesity was not asked directly but were calculated based on questions about current height and weight. A BMI score of 25 or more was classified as overweight or obese.(73) Venereal Disease was measured by asking respondents if they have been treated for a sexually transmitted or venereal disease in the past five years.

Table 4-3 Dependent Variables

Physical Health Measures	Mental and Social Health Measures	Behavioral Measures
General Health	MHQoL	Binge Drinking
PHQoL	Life Satisfaction	Multiple Sex Partners
Cancer	Serious Mental Illness	Smoking
Diabetes	Perceived Social Support	Physical Activity
Hypertension	Access to Health Care	Fruits and Vegetables
High Cholesterol	Census Measures	Mammogram compliance
Cardiovascular Disease	Percent Unemployment	Pap test compliance
Asthma	Percent Poverty	
Overweight/Obesity	Percent Black Population	
Venereal Disease		

Mental health indicators included Mental Health Related Quality of Life (MHQoL) (measured and coded the same way as PHQoL), Life Satisfaction, and the Kessler 6 (K6) scale which is a scale that measures nonspecific psychological distress. Life satisfaction was included here as a mental health indicator as it has been shown to predict psychiatric morbidity.(74) It is measured with a single item that ask respondents to rate how satisfied they are with their life on a four point scale ranging from very satisfied to very dissatisfied. The K6 is a six item scale used to screen populations for serious mental illness (SMI) and has been shown to be a reliable and valid measure.(75) The K6 measures feelings of nervousness, hopelessness, restlessness or agitation, depression, fatigue, and worthlessness in the past 30 days. Each of the six items was measured on a 5-point Likert response scale ranging from “none of the time” to “all of the time.” The scale was summed across the 6 items to produce a cumulative score which can range from 0-

24, with higher scores indicating greater psychological distress. A cut-point of greater than or equal to 13 was used to signify the potential for SMI.(76)

Social variables included Perceived Social Support, Access to Health Care, and three census measures, namely Percent Unemployment, Percent Poverty, and Percent Black population. Perceived Social Support was measures using a validated four item scale that asks respondents how often certain types of support would be available it they needed it (77). The response scale consisted of a 5-item scale ranging from "none of the time" to "all of the time". Scores were calculated by summing across the items with a possible score ranging from 0-16. Higher scores indicated more perceived social support. Access to health care included 4 items. The first item asked respondents if they had any kind of health care coverage, including health insurance, prepaid plans, or government plans. The second item asked if they had a person that they think of as their personal doctor or health care provider. The third item asked if they needed to see a doctor in the past 12 months, but could not due to cost. All three of these items were coded as yes or no. The final item asked when they last visited a doctor for a routine check-up. Responses to this question were recoded and dichotomized into yes or no depending on whether or not they had a routine check-up within the past year.

Several 2010 Census measures were added to the ACHS data set to represent community level factors that may be related to prevalence of ACEs and thus could be used to identify possible places for intervention. For all respondents who had complete geographical information (i.e., x and y coordinates as ascertained by asking respondents to identify the streets that make up the nearest corner to their house) census information was added to the corresponding record including percent unemployment, percent living at or below the poverty line, and percent Black population. These variables were then recoded into dichotomous variables. Using the 2006-

2010 American Community Survey 5-Year Estimates for Allegheny County, unemployment was coded high if unemployment for a given tract was above the county rate of 6.8. The sample median of 5.2% was used to recode tracts into poverty high and poverty low tracts, and the 75% quartile was used to recode tracts into Black high population and Black low population.

Behavioral measures used in the analyses below included Binge Drinking, Multiple Sex Partners, Smoking, Physical Activity, Fruits and Vegetables consumption, and for women Mammogram and Pap Test compliance, all of which come from the CDC BRFSS core measures. Binge drinking was assessed by asking respondents the number of times in the past 30 days that they had 4 (for female) or 5 (for male) drinks on an occasion. This information was then recoded into a dichotomous variable to indicate potential for alcohol abuse or problem drinking. Five or more binge drinking episodes in the past 30 days was considered as possible alcohol problems. Multiple Sexual Partners was assessed by asking respondents how many people they had sexual intercourse with in the past 12 months. Those that reported 2 or more were coded as having multiple sexual partners while those who did not have sex or sex with only 1 person were coded as not having multiple sexual partners. Smoking was treated as an ordinal variable. Respondents were asked whether or not they ever smoked 100 cigarettes in their lifetime. If they did, then they were asked if they currently smoke every day, some days, or not at all. Those that responded "no" to smoking at least 100 cigarettes in their lifetime were coded as "0." Those that were former smokers were coded as "1" and those that were currently smoking but not every day were coded as "2." Current everyday smokers were coded as "3." The physical activity measure asked respondents to indicate how many days in a typical week they engage in moderate or vigorous activities for at least 10 minutes at a time. They were then asked to estimate, on average, the amount of time they spend engaged in that activity. Using the CDC's

recommendations for weekly physical activity (2.5 hours per week of moderate or vigorous activity; or 1.25 hours of vigorous activity) the activity variables were combined and recoded into compliance versus non-compliance with recommendations. The CDC's recommendations were also used to recode fruit and vegetable consumption. Those that reported, on average, eating at least 2 fruits and 3 vegetable servings per day were considered to be in compliance with recommendation and those that did not meet that threshold were not compliant. Pap Test and Mammogram Compliance were determined based on screening guidelines in effect at the start of data collection (78): a mammogram every two years for women 40 years and older; a Pap test in the past year for women 21-29 years old, and a Pap test in the past three years for women 30 to 65 years of age. [Note: In mediation analyses, when behavioral variables were entered into models they were not included as dichotomous variables but as ratio or interval level measures.]

The **main intervening variables** (mediating and moderating variables) included demographic, neighborhood, and behavioral and social variables (discussed above). Demographic variables included age, race, gender, household income, education level, and parental education. (See Appendix A, Table 1). Environmental measures included several neighborhood level indicators taken from a scale developed by Mujahid et al. (79) with subscales pertaining to neighborhood walking environment, social cohesion, and two items regarding the availability of healthy and fast-foods in one's neighborhood. Each of these items was measured on a five-point Likert scale, ranging from "strongly agree" to "strongly disagree." A measure of childhood socio-economic status (i.e., highest education level achieved by respondent's parent or guardian) was collected and used as a proxy for exposure to low socioeconomic status in childhood which has been shown to be related to exposure to adversity.

4.1.8 Analyses of missing data

Pertinent variables were analyzed in terms of item non-response (i.e., missing, "refused", and "don't know" responses). Variables that had a large percentage (greater than 5%) of item non-response were considered for potential non-response bias.(80)

Prior to data analysis, several procedures were conducted in order to determine the potential for bias due to item nonresponse. Of the 5442 participants who participated in the survey, 539 or 9.9% did not receive or complete the ACE module. In order to determine if those completing the ACE module differed significantly from those who either terminated before the ACE module was administered or who failed to answer all or part of the ACE module, a comparison between the missing and non-missing ACE groups was conducted by evaluating differences in age, gender, race, education, and household income. The results of the comparisons (t- test for age and Rao-Scott chi-square for proportions (81)) are presented in Table 4-4. Overall, those missing all or part of the ACE module were more likely to be non-Hispanic Black, have less than college education, and have household incomes below $25,000 per year. No significant differences were noted on the basis of age and gender. Results from this analysis indicate that the bias, if any, is likely skewed towards an underreporting of ACEs given that minorities and those with lower SES are more likely to have experienced hardships and adversity which as a result of incomplete data is not captured fully in this survey.

Table 4-4 Demographic Comparison between those with Complete and Incomplete ACE Scores

	Complete ACE Module (n=4903)	***Missing/Incomplete ACE Module (n=539)***	***p***
Age (Mean)	51.1 (95%CI: 49.4, 52.8)	51.8 (95%CI: 48.2, 55.3)	0.72
Gender (% female)	52.7 (95%CI: 50.9, 54,6)	55.7 (95%CI: 50.3, 61.2)	0.31
Race (% Non-Hispanic White)	88.7 (95%CI: 87.6, 89.2)	81.9 (95%CI: 78.7, 85.2)	<0.0001
Education (% college)	59.9 (95%CI: 58.1, 61.7)	51.4 (95%CI: 46.1, 56.7)	<0.01
Household Income (% < $25,000)	29.2, (95%CI: 58.1, 61.7)	40.1 (95%CI: 34.9, 45.4)	<0.0001

Household income was also assessed in terms of missing data. Of the 5442 participants who participated in the survey, 832 or 15.3% did not provide information on household income. Data were imputed for these missing data points (see Data Weighing and Imputation, section 4.1.4). Because such a large percentage of income data was missing, imputed data was evaluated in terms of how imputation may have altered the distribution of ACE scores by household income. Table 4-5 presents comparisons of the percentage reporting 1 or more ACEs (1+ACEs) by household income for those with and without (i.e., imputed) income. Inspection of the confidence intervals reveals that across levels of income, the percentages of those reporting 1+ACEs was not significantly different between the two groups. Rao-Scott chi-square tests were performed separately for those with and without imputed income data to determine if within groups there were significant difference in ACE reporting across income groups. Only for the group with reported income data was there a significant association (p=.04) between household income and reporting 1+ACEs. That is, those in the highest income category reported significantly less adversity than those in the lowest income category. In the sample with imputed income no significant differences were observed. Thus, including the imputed income data in subsequent analyses should not inflate the effect of income on childhood adversity prevalence. If anything, SES differences that are observed should be considered conservative given that nearly 15% of the income data was imputed and for that 15% no significant relationship between

SES and ACEs was observed. The imputed income data was retained in the analyses below to increase statistical power.

Table 4-5 Prevalence of ACEs by Income Level: Comparison of Actual and Imputed Data

Income level	Weighted percentages of 1+ACEs for those with reported household income (n=4239)		Weighted percentages of 1+ACE for those with imputed household income (n=664)	
	% 1+ACE	(95%CI)	% 1+ACE	(95%CI)
< $15,000	67.6	(62.2, 73.0)	51.6	(36.1, 67.0)
$15,000 – $24,999	62.2	(57.8, 66.6)	51.5	(39.5, 63.6)
$25,000 – $49,999	59.7	(56.5, 63.0)	59.8	(51.0, 68.5)
$50,000 – $74,999	60.0	(55.5, 64.5)	61.2	(47.2, 75.3)
$75,000 +	57.3	(53.4, 61.3)	51.3	(40.7, 62.0)

4.1.9 Scale Reliability and Construction

Adverse Childhood Adversity Scale: Given that the ACE scale used in the ACHS was modified from the original, reliability coefficients (Cronbach's Alpha) were calculated for the overall ACE scale (6 items) as well as separately for the two subscales, child-maltreatment (3 items) and household dysfunction (3 items). The purpose of this analysis was to determine whether or not items on the full scale were internally consistent and collectively measuring CA. This was also done to determine whether the two subscales could be used separately and alone to measure the dimensions of CA captured in the ACHS. Results from these analyses are presented below in Table 4-6 and indicate that the full 6-item ACE scale appeared to have good internal consistency (standardized alpha =.73) while the two subscales had mediocre to poor internal consistency: household dysfunction (standardized alpha = .56) and child maltreatment (standardized alpha = .65). A reliability coefficient of 0.7 is often used to indicate an acceptable

reliability coefficient for scales.(82) Therefore in subsequent analyses only the full ACE scale was used as a measure of CA.

Table 4-6 Reliability Coefficients for ACE Scale and Subscales

	Cronbach's Alpha	
Scale	Raw	Standardized
Full ACE Scale	0.72	0.73
Child Maltreatment Scale	0.50	0.56
Household Dysfunction Scale	0.65	0.65

To further evaluate the ACE scale to determine if it needed to be collapsed to ensure robust cell sizes, summary scores were computed using the coding scheme presented in Appendix Table 2. Results indicated that ACE scores were heavily skewed to the left with less than 10% of the sample having ACE scores between 6 and 18, while 60% of the sample had scores that fell between 0 and 2 (See Table 4-7). To ensure that small cell sizes would not become problematic the 19 point scale was reduced to a 5 point scale. ACE scores 0, 1, and 2 remained unchanged and reflect the actual number of ACEs reported. ACE scores 3, 4, 5 were combined into a single category, as were ACE score 6 through 18. Tables 4-7 and 4-8 present the distributions of the raw and recoded ACE scores.

Table 4-7 Distribution of Adverse Childhood Experiences Scores

Raw ACE Score	Frequency	Percent	Cumulative Frequency	Cumulative Percent
0	2127	43.38	2127	43.38
1	832	16.97	2959	60.35
2	578	11.79	3537	72.14
3	347	7.08	3884	79.22
4	284	5.79	4168	85.01
5	161	3.28	4329	88.29
6	147	3.00	4476	91.29
7	110	2.24	4586	93.53
8	83	1.69	4669	95.23
9	65	1.33	4734	96.55
10	60	1.22	4794	97.78
11	36	0.73	4830	98.51
12	17	0.35	4847	98.86
13	22	0.45	4869	99.31
14	11	0.22	4880	99.53
15	7	0.14	4887	99.67
16	7	0.14	4894	99.82
17	4	0.08	4898	99.90
18	5	0.10	4903	100.00

Table 4-8 Distribution of Recoded Adverse Childhood Experiences Scores

Recoded ACE score	Frequency	Percent	Cumulative Frequency	Cumulative Percent
0	2127	43.38	2127	43.38
1	832	16.97	2959	60.35
2	578	11.79	3537	72.14
3	792	16.15	4329	88.29
4	574	11.71	4903	100.00

The K6 and the 4-item Social Support (derived from the MOS-36) scale have been shown to be reliable in population-based samples and thus internal consistency of the scales was not calculated. However, the ACE scale and the neighborhood quality scale used in the ACHS contain only a subset of items from their original scales. To my knowledge the scales included in the ACHS have not been tested for reliability. An alpha coefficient for each of the subscales was calculated using PROC CORR with ALPHA option in SAS and an alpha coefficient

between 0.7 and .09 was considered acceptable.(83) Neighborhood quality was measured using Mujahid scale (79) which included neighborhood social cohesion (4-items), neighborhood walkability (8 items), access to fresh foods (1 item), and availability to fast foods (1 item). Reliability coefficients (Cronbach Alpha) for the overall walkability and social cohesion scales were calculated. Results from these analyses indicated that both scales had good internal consistency, standardized alpha = 0.75 and standardized alpha =0.79, respectively.

4.1.10 Composite SES Measures

Three socioeconomic status (SES) measures, namely childhood SES (CSES), adult SES (ASES), and lifetime SES (LSES), were used in the analyses below. CSES was measured using the highest reported level of education received by either parent and trichotomized into low, middle, and high CSES. Low CSES was defined as highest parental education less than high school. Middle CSES was defined as highest parental education equal to high school graduate or equivalent. High CSES was defined as parental education greater than high school. ASES was comprised of two variables, reported education level and annual household income level. These two variables were combined to create a single measure by calculating Z scores using a mean of 0 and standard deviation of 1. The z scores were then trichotomized into low, middle, and high ASES groups using the 33^{rd} and 67^{th} percentiles. Similarly, LSES was calculated and categorized but in addition to ASES, LSES also included CSES. All three SES variables and their frequency distribution and weighted percentages are presented in Table 4-9 below.

Table 4-9 Recoded and Composite SES Measures

SES Measure	Frequency	Weighted percentage	95% CI
Childhood SES (highest parental education)			
< Grade 12 or GED	849	14.8	(13.7, 15.9)
Grade 12 or GED	1964	43.9	(42.0, 45.7)
> Grade 12 or GED	1675	41.3	(39.4, 43.3)
Adult SES			
Low ASES	1320	24.8	(23.2, 26.3)
Middle ASES	2684	49.9	(48.2, 51.6)
High ASES	1439	25.4	(23.9, 26.8)
Lifetime SES			
Low LSES	1512	32.2	(30.5, 33.9)
Middle LSES	1497	35.8	(33.9, 37.6)
High LSES	1479	32.0	(30.3, 33.8)

4.2 DATA ANALYSIS

4.2.1 Software

SAS software, Version 9.2 of the SAS system for Windows was used to analyze the ACHS data as it supports several statistical procedures that account for the design effects and post-stratification weighting of complex surveys (PROC SURVEYFREQ, PROC SURVEYMEANS, PROC SURVEYREGRESSION, and PROC SURVEYLOGISTIC).

4.2.2 Specific Aim 1: To estimate and describe the prevalence of CA in terms of type and frequency and by social, demographic, and geographic characteristics.

Descriptive analyses for ACEs were conducted and entailed calculating both crude and age-adjusted estimates for the total population and across sub-populations. To account for any differences in the age distributions across sub-populations, all estimates were weighted to Census 2000 age estimates (See Table 4-10). Age adjustment was done because ACEs are known to be significantly and negatively correlated with age.(84)

Table 4-10 Age Adjusted Weights

Age	Population in Thousands	Adjustment Weight
18-29	43980	0.2157458
30-39	41691	0.204517
40-49	42285	0.2074309
50-59	30531	0.1497712
60-69	20064	0.0984248
70+	25300	0.1241103
Total	203851	1.0

Age-adjusted weighted percentages and 95%CI were calculated using SAS 9.2 PROC SURVEYREGRESSION. The ACE estimates, separately and as a summary score, were compared across gender, race, employment status, disability status, SES strata, and census tract

characteristics. In order to attach census tract information to the ACHS data set, all records with complete geographic data (n=3910) were imported into ARC GIS and mapped to 2010 census tracts. Aggregated census tract information (e.g., Percent Unemployment) from the 2006-2010 American Community Survey was then merged with ACHS data set.

To evaluate the hypotheses concerning differences in ACE prevalence across social, demographic, and geographic characteristics, comparisons of the 95% CI were made. If there was no overlap between the confidence intervals (CI) being compared, then a statistically significant difference between groups was concluded at an alpha level of 0.05.

4.2.3 Specific Aim 2: To describe the adult health risks associated with CA and quantify the proportion of disease in the adult population attributable to CA.

Bivariate and multivariate regression models were used to determine the association between CA and the adult physical, mental, social outcomes as well as adult behavioral risk factors in adulthood. In multivariate models, age, sex, race, and lifetime SES (i.e., a composite of parental education, adult education, and adult household income) were entered as covariates. Both odds ratios (OR) and adjusted odds ratios (AOR) and their respective CI were calculated. An alpha level of .05 was used to determine statistical significance. Analyses were conducted using SAS PROC SURVEYLOGISTIC so that the weighting and stratification of the survey data could be accounted for in variance estimations.

For those health outcomes and behavioral risk factors that were found to be significantly associated with exposure to ACEs, attributable risk (AR) and population attributable risk (PAR) fractions were calculated. AR is the proportion of disease that can be attributed to a specific exposure and is calculated by subtracting the disease risk of the unexposed from the disease risk

of the exposed.(85) PAR estimates the proportion of the disease or outcome in the population that could be eliminated if the exposure was eliminated and accounts for the level of exposure in the population. From a public health perspective these measures indicate the amount of preventable morbidity that could be avoided if CA was eliminated from the population. The formulas used for calculating the AR and PAR are as follows. (85, 86) (87)

$$AR = (AOR–1)/1+(AOR–1)$$

$$PAR= P(AOR–1)/1+P(AOR–1)$$

Where:

AOR = Adjusted odds ratio for ACE and adult health outcome for exposed

P = Percentage in the population exposed to ACE

Significance of the PAR estimates was assessed by constructing a 95% CI. The upper and lower limits, PAR_L and $PARu$, were calculated as follows.

$$PAR_L = 1 – \exp(\ln(1\text{-}PAR) + 2.24s)$$

$$PAR_U = 1 + \exp(\ln(1\text{-}PAR) - 2.24s)$$

Where:

s = s.e. (ln(1-PAR))

s.e. = standard error of PAR estimate

If the interval included zero, an absence of statistical significance was concluded. PAR is a function of both prevalence and odds ratio and so a Bonferonni correction was used to calculate confidence limits around PAR to maintain a combined alpha level of .05.(88)

4.2.4 Specific Aim 3: To evaluate the roles of demographic, social, behavioral and geographic factors in the relationship between CA and the pathogenesis and development of adult disease.

Given the large number of health indicator variables in the ACHS dataset, the focus for these analyses was constrained to SMI and CVD. These two health outcomes were most associated with ACEs when considering mental and physical health indicators separately, and they represent the largest PARs for mental health physical health outcomes, respectively, as determined by the analyses conducted under Specific Aim 2.

Mediation of the ACE adult health relationship (hypotheses 3.1-3.3) was tested separately for each of the mediators (M1-M3) depicted in Figure 1. Using both logistic and linear regression models, paths *a b* were estimated separately. Path *a* represents the correlation between the ACE score and the mediator, and path *b* represents the correlation between the mediator and the health outcome while controlling for ACE score. The use of either logistic or linear regression depended on whether or not the dependent variable was nominal or ordinal. The mediated or indirect effect was then computed by taking the product of the *a b* parameters (89). Given that the distribution of *ab* is often non-normal, asymmetrical confidence limits were constructed using bootstrap sampling.(89) Bootstrapping the *ab* parameter consisted of taking 1000 samples (with replacement) from the original sample of the same size as the original sample, and then calculating the *ab* parameter for each of those samples. The *ab* parameters were then ordered in ascending order to identify the value in the ordinal position at the 2.5 and 97.5 percentile of the distribution. These two values represent the lower and upper CLs, respectively. The mediated effect was considered significant at an alpha level of .05 if the resulting bootstrapped CI was did not include zero.

Full mediation occurred when the direct effect of ACEs on the health outcome of interest was no longer significant when the mediator was included in the model. Partial mediation occurred when the indirect effect was significant but the direct effect also remained significant. For example ACEs may affect CVD through smoking, but also ACEs may affect CVD directly or independently of smoking. To estimate the effect size of the indirect effect, the proportion of the indirect effect (P_m) was calculated. [Note: P_m = the indirect effect (*ab)/* the total effect*(c)*. The closer P_m is to one, the more the effect of x on y operates through the mediator.] This effect size measure was used to describe and compare across mediators in terms of how much of the ACE adult health relationship is accounted for by the meditators.

Hypotheses 3.4-3.7 address questions about whether or not the direct or indirect effects of ACEs on adult health outcomes vary as a function of age, gender, race, CSES, or neighborhood characteristics. Answers to these questions provide information as to for whom and under what circumstances ACEs affect adult health. To test moderation of the direct effects, interaction terms were included into the statistical models used to test mediation above. If the interaction term *xz* was significant at alpha level 0.05, then the indirect effect of ACEs on adult health was said to be moderated by the interaction variable z. Similarly moderated mediation seeks to determine if the *indirect* effects of ACEs on adult health through a mediator, such as alcohol use, differ by an intervening variable such as age or race. To test for moderation of the indirect effects, again the interaction term was entered into the model, but rather than *xz*, the term *zm* was included. If the interaction term was significant at alpha level 0.05, then the indirect effect of ACEs on adult health through M was said to be moderated by the interaction variable.

For models where interaction was significant, interaction effects were probed to determine at what levels of the interaction variable the relationship between ACEs and adult

health was significant. To assist with the large number of models to be estimated, a SAS macro developed by Andrew Hayes called PROCESS was used. (90)

5.0 RESULTS

5.1 PREVALENCE OF CHILDHOOD ADVERSITY

To estimate, describe, and compare the prevalence of CA in terms of type and frequency and by social, demographic, and geographic characteristics. Overall, 60.7% (95%CI: 58.8, 62.7) of the adult population in Allegheny County, Pennsylvania experienced at least 1 ACE (See Figure 4-1). The two most prevalent types of ACEs reported were childhood emotional abuse (26.3%) and growing up while living with someone who was a problem drinker, alcoholic, or drug user (26.0%). Sexual abuse was the least endorsed at 4.5%.

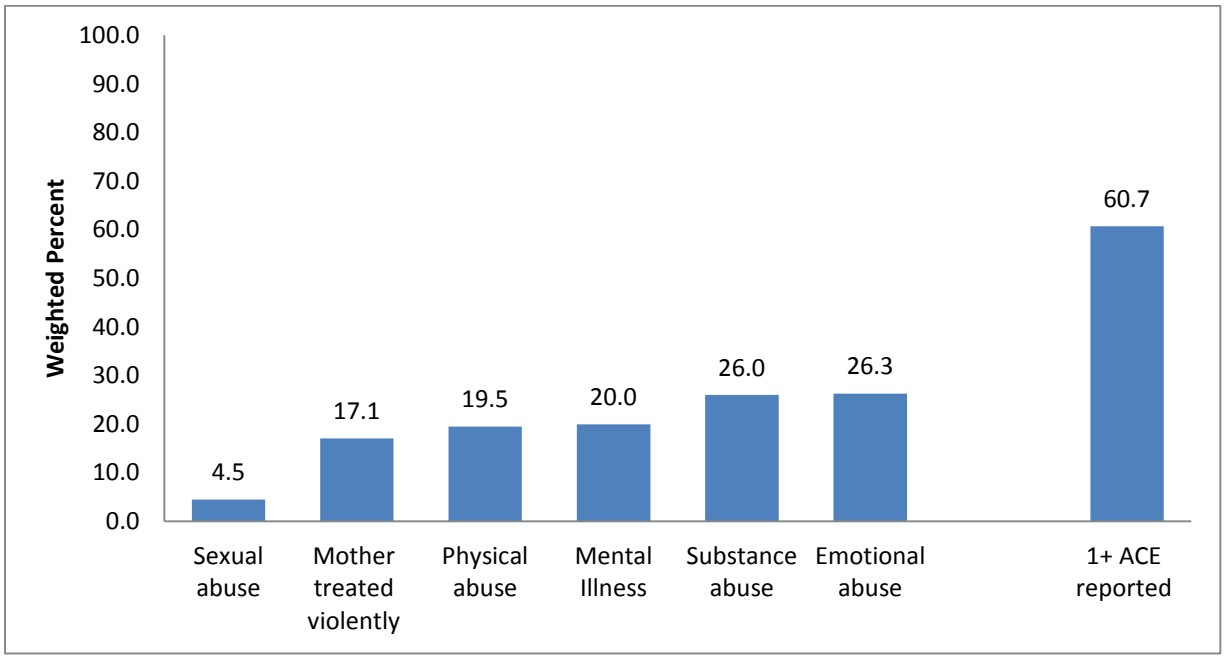

Figure 5-1 Age-Adjusted Weighted Prevalence of Childhood Adversity by Type

Crude and age-adjusted prevalence estimates of ACEs are presented in Table 5-1. Age and ACE score were significantly and negatively correlated ($r = -0.16$, $p < .0001$) such that with

increasing age there was a significant decrease in reported ACEs. The age-adjustment resulted in overall increases in prevalence by ACE type ranging from 0.3 % to 1.4%. These increases are likely a result of the negative correlation between ACE score and age and the fact that the ACHS sample was over representative of older adults. In order to account for the possible varying age structures amongst sub-populations, all ACE comparisons between social, demographic, and geographic sub-populations were done so using age-adjusted estimates.

Table 5-1 Crude and Age Adjusted Prevalence (Un-weighted Counts and Weighted Percentages) of Adverse Childhood Experiences by Event Type and Frequency for Adult Residents of Allegheny County, Pennsylvania 2009-2010

Adverse childhood experiences	n	Crude % (95%CI)	Age-Adjusted % (95%CI)
Childhood Maltreatment			
(When you were growing up, how often did a parent or adult living in your house. . .)			
(Emotional) sometimes, often or very often swear at you, insult you, or put you down?	1134	25.3 (23.6, 26.9)	26.3 (24.3, 28.2)
(Physical) sometimes, often or very often hit, beat, kick, or physically hurt you?	905	19.2 (17.7, 20.6)	19.5 (17.9, 21.2)
(Sexual) ever touch you sexually or try to make you touch them sexually?	212	4.1 (3.2, 5.0)	4.5 (3.4, 5.5)
Household Dysfunction			
(When you were growing up, how often did a parent or adult living in your house. . .)			
(Mother treated violently) ever grab, slap, or throw something at your mother?	730	16.3 (14.9, 17.7)	17.1 (15.5, 18.7)
(When you were growing up as a child . . .)			
(Substance abuse) did you live with anyone who was a problem drinker, alcoholic, or drug user?	1167	25.0 (23.4, 26.6)	26.0 (24.1, 27.9)
(Mental illness) was anyone living in your home depressed, mentally ill, or suicidal?	811	18.6 (17.1, 20.1)	20.0 (18.2, 21.7)
# of Adverse childhood experiences (ACE score)			
0	2127	40.2 (38.5, 42.0)	39.3 (37.3, 41.2)
1	832	16.7 (15.4, 18.0)	16.6 (15.1, 18.0)
2	578	12.1 (11.0, 13.2)	12.0 (10.7, 13.3)
3	792	18.0 (16.5, 19.5)	18.7 (16.9, 20.4)
4	574	12.9 (11.6, 14.2)	13.5 (12.0, 15.0)

Hypothesis 1.1: Compared to males, females have a higher lifetime prevalence of CA. Results indicated that aside from physical abuse, females reported experiencing greater CA across all types, however only the differences in sexual abuse and growing-up with someone

who was mentally ill were statistically significant. The percentage of females reporting sexual abuse was 4.6 times that of males and 1.3 times that of males for growing-up with someone who was mentally ill. Counter to my hypothesis, females more than males reported having experienced no ACEs, 41.3% versus 36.7% respectively, however this difference is based on point-estimates and was not statistically significant. A greater percentage of females did report an ACE score of 4, indicating that when women experienced CA they tended to experienced multiple adversities more so than males. Again, this difference was not statistically significant.

Table 5-2 Age-Adjusted Prevalence of Adverse Childhood Experiences by Gender

Adverse childhood experience	Females, % (95%CI)	Males, % (95% CI)
Childhood Maltreatment		
(When you were growing up, how often did a parent or adult living in your house. . .)		
(Emotional) sometimes, often or very often swear at you, insult you, or put you down?	26.7 (24.2, 29.1)	26.0 (23.1, 29.0)
(Physical) sometimes, often or very often hit, beat, kick, or physically hurt you?	19.5 (17.2, 21.8)	20.0 (17.6, 22.4)
(Sexual) ever touch you sexually or try to make you touch them sexually?	7.3 (5.3, 9.2)	1.6 (0.7, 2.4)*
Household Dysfunction		
(When you were growing up, how often did a parent or adult living in your house. . .)		
(Mother treated violently) ever grab, slap, or throw something at your mother?	18.5 (16.5, 20.5)	15.9 (13.3, 18.4)
(When you were growing up as a child . . .)		
(Substance abuse) did you live with anyone who was a problem drinker, alcoholic, or drug user?	27.9 (25.4, 30.4)	24.0 (21.2, 26.8)
(Mental illness) was anyone living in your home depressed, mentally ill, or suicidal?	22.7 (20.3, 25.1)	17.3 (14.7, 19.8)*
# of Adverse childhood experiences (ACE score)		
0	41.3 (38.9, 43.7)	36.7 (33.6, 39.8)
1	16.0 (14.1, 17.9)	17.3 (15.1, 19.6)
2	10.0 (8.6, 11.4)	14.2 (12.0, 16.4)*
3	17.4 (15.3, 19.4)	20.0 (17.2, 22.8)
4	15.4 (13.1, 17.6)	11.7 (9.8, 13.7)

* indicates a significant difference, $p < .05$

Hypothesis 1.2: Compared to Non-Hispanic Whites, Non-Hispanic Blacks have higher lifetime prevalence of CA. Overall and by type, Non-Hispanic Blacks reported greater CA. Non-Hispanic Blacks reported significantly more sexual and physical abuse, 2.5 times and 1.7 times that of Non-Hispanic Whites, respectively. Non-Hispanic Blacks also reported experiencing 4 or more adversities significantly more often than Non-Hispanic Whites. Non-Hispanic Whites did report experiencing living with someone who was mentally ill or suicidal more so than Non-Hispanic Blacks, but this was non-significant. No significant racial differences were observed in those reporting no ACEs.

Table 5-3 Age-Adjusted Prevalence of Adverse Childhood Experiences by Race

Adverse childhood experience	Non-Hispanic White, % (95%CI)	Non-Hispanic Black, % (95%CI)
Childhood Maltreatment		
(When you were growing up, how often did a parent or adult living in your house. . .)		
(Emotional) sometimes, often or very often swear at you, insult you, or put you down?	25.5 (23.4, 27.6)	25.4 (22.2, 28.7)
(Physical) sometimes, often or very often hit, beat, kick, or physically hurt you?	17.8 (16.0, 19.6)	29.4 (26.0, 32.8)*
(Sexual) ever touch you sexually or try to make you touch them sexually?	3.3 (2.4, 4.1)	8.2 (6.2, 10.2)*
Household Dysfunction		
(When you were growing up, how often did a parent or adult living in your house. . .)		
(Mother treated violently) ever grab, slap, or throw something at your mother?	16.6 (14.8, 18.4)	18.5 (15.6, 21.4)
(When you were growing up as a child . . .)		
(Substance abuse) did you live with anyone who was a problem drinker, alcoholic, or drug user?	25.2 (23.1, 27.2)	30.1 (26.8, 33.5)
(Mental illness) was anyone living in your home depressed, mentally ill, or suicidal?	20.4 (18.5, 22.4)	17.0 (14.1, 19.8)
# of Adverse childhood experiences (ACE score)		
0	39.8 (37.5, 42.0)	38.1 (34.5, 41.6)
1	17.1 (15.4, 18.7)	12.6 (10.1, 15.0)
2	11.6 (10.1, 13.1)	15.4 (12.7, 18.1)
3	19.2 (17.2, 21.1)	17.1 (14.2, 20.0)
4	12.4 (10.9, 13.9)	16.8 (14.0, 19.7)*

* indicates a significant difference, p <.05

Hypothesis 1.3: Compared to high SES individuals (measured in terms of household income, education level, and parental education), low SES individuals have a higher lifetime prevalence of CA. Childhood SES, Adulthood SES, and Lifetime SES were measured and compared on the basis of ACE prevalence. Results from these analyses are presented in Tables 14-16. Across all three measures of SES a clear pattern was observed such that with increasing SES there was a decrease in the prevalence of reported CA. Those with high CSES had significantly less emotional abuse when compared to those with middle CSES and

significantly less physical abuse when compared to those with low CSES. Although there were great differences in the point estimates of sexual abuse between those with middle and high CSES and those with low CSES, these differences were non-significant. Overall, classification of CSES into low, middle, and high did not reveal significant differences in the prevalence of having experienced at least one ACE; however, there were statistically significant differences on the ACE score such that 21.7% of those with low CSES had an ACE score of 4 compared to only 9.8% of those with high CSES. Results suggest that certain types of ACEs occurred more when CSES was low and that when CSES was low a greater number or severity of adversities was reported when compared to those with higher CSES.

Table 5-4 Age-Adjusted Prevalence of Adverse Childhood Experiences by Childhood SES

Adverse childhood experience	Low CSES, % (95%CI)	Middle CSES, % (95%CI)	High CSES, % (95%CI)
Childhood Maltreatment			
(When you were growing up, how often did a parent or adult living in your house. . .)			
(Emotional) sometimes, often or very often swear at you, insult you, or put you down?	36.2 (24.5, 48.0)	29.2 (26.0, 32.3)	22.7 (20.0, 25.4)*
(Physical) sometimes, often or very often hit, beat, kick, or physically hurt you?	33.9 (21.9, 45.8)	20.9 (18.1, 23.7)	16.1 (13.8, 18.4)*
(Sexual) ever touch you sexually or try to make you touch them sexually?	15.9 (4.9, 26.9)	3.9 (2.7, 5.0)	4.2 (2.5, 5.9)
Household Dysfunction			
(When you were growing up, how often did a parent or adult living in your house. . .)			
(Mother treated violently) ever grab, slap, or throw something at your mother?	28.2 (16.7, 39.6)	19.4 (16.6, 22.2)	13.8 (11.6, 16.0)*
(When you were growing up as a child . . .)			
(Substance abuse) did you live with anyone who was a problem drinker, alcoholic, or drug user?	37.8 (25.9, 49.6)	29.2 (26.0, 32.3)*	22.2 (19.6, 24.8)*
(Mental illness) was anyone living in your home depressed, mentally ill, or suicidal?	26.5 (15.2, 37.7)	20.5 (17.6, 23.4)	19.7 (17.3, 22.2)
# of Adverse childhood experiences (ACE score)			
0	32.2 (21.1, 43.4)	37.5 (34.4, 40.6)	40.8 (37.8, 49.7)
1	15.3 (10.3, 20.4)	13.8 (11.9, 15.6)	19.3 (16.9, 21.7)
2	11.2 (5.3, 17.0)	12.1 (10.0, 14.3)	11.9 (10.1, 13.8)
3	14.2 (9.8, 18.6)	21.1 (18.1, 24.0)	18.2 (15.7, 20.6)
4	27.1 (15.4, 38.7)	15.6 (13.1, 18.1)	9.8 (7.8, 11.8)*

* indicates a significant difference compared to low CSES group, $p < .05$

A similar, but more dramatic pattern was observed when comparing ACEs across ASES and LSES strata. In many comparisons the prevalence of ACES was 2 times greater in the Low SES group compared to the High SES group, and as much as 5 times greater when comparing sexual abuse. For every ACE type, there was a significant difference in prevalence between the Low and High SES groups for both the ASES and LSES measures. Moreover, there were significant differences between SES groups for reporting at least one ACE as well as for reporting 4 or more ACEs.

Table 5-5 Age-Adjusted Prevalence of Adverse Childhood Experiences by Adult SES

Adverse childhood experience	Low ASES, % (95%CI)	Middle ASES, % (95%CI)	High ASES, % (95%CI)
Childhood Maltreatment			
(When you were growing up, how often did a parent or adult living in your house. . .)			
(Emotional) sometimes, often or very often swear at you, insult you, or put you down?	38.1 (31.4, 40.8)	28.8 (25.6, 31.9)	19.2 (16.7, 21.7)*
(Physical) sometimes, often or very often hit, beat, kick, or physically hurt you?	32.7 (28.1, 37.4)	20.6 (18.1, 23.2)*	12.3 (10.3, 14.3)*
(Sexual) ever touch you sexually or try to make you touch them sexually?	10.0 (6.4, 13.6)	3.8 (2.5, 4.9)*	2.1 (1.4, 2.8)*
Household Dysfunction			
(When you were growing up, how often did a parent or adult living in your house. . .)			
(Mother treated violently) ever grab, slap, or throw something at your mother?	23.7 (19.7, 27.6)	19.7 (16.8, 22.6)	11.2 (9.5, 13.0)*
(When you were growing up as a child . . .)			
(Substance abuse) did you live with anyone who was a problem drinker, alcoholic, or drug user?	41.6 (36.9, 46.4)	26.4 (23.6, 29.3)*	17.2 (14.8, 19.6)*
(Mental illness) was anyone living in your home depressed, mentally ill, or suicidal?	28.4 (23.9, 32.9)	18.0 (15.4, 20.5)*	17.1 (14.8, 19.6)*
# of Adverse childhood experiences (ACE score)			
0	31.4 (27.4, 35.5)	37.0 (33.8, 40.2)	44.9 (41.8, 48.1)*
1	12.1 (9.0, 15.2)	16.2 (13.9, 18.4)	19.5 (17.0, 22.1)*
2	10.8 (8.1, 13.6)	12.1 (10.1, 14.2)	12.9 (10.5, 15.2)
3	18.6 (15.0, 22.2)	21.5 (18.5, 24.6)	15.5 (13.2, 17.8)
4	27.1 (22.4, 31.7)	13.2 (11.1, 15.2)*	7.1 (5.8, 8.5)*

* indicates a significant difference compared to low ASES group, $p < .05$

Table 5-6 Age-Adjusted Prevalence of Adverse Childhood Experiences by Lifetime SES

Adverse childhood experience	Low LSES, % (95%CI)	Middle LSES, % (95%CI)	High LSES, % (95%CI)
Childhood Maltreatment			
(When you were growing up, how often did a parent or adult living in your house. . .)			
(Emotional) sometimes, often or very often swear at you, insult you, or put you down?	34.8 (30.3, 39.3)	28.0 (24.7, 31.2)	19.8 (16.8, 22.8)*
(Physical) sometimes, often or very often hit, beat, kick, or physically hurt you?	27.8 (23.3, 32.2)	21.2 (18.3, 24.0)	11.6 (9.7, 13.6)*
(Sexual) ever touch you sexually or try to make you touch them sexually?	8.3 (4.7, 11.8)	4.0 (1.3, 5.4)	2.0 (1.3, 2.7)*
Household Dysfunction			
(When you were growing up, how often did a parent or adult living in your house. . .)			
(Mother treated violently) ever grab, slap, or throw something at your mother?	21.6 (18.0, 25.3)	18.9 (16.0, 21.8)	12.1 (9.5, 14.8)*
(When you were growing up as a child . . .)			
(Substance abuse) did you live with anyone who was a problem drinker, alcoholic, or drug user?	39.6 (35.2, 44.0)	25.8 (23.6, 30.0)*	16.2 (13.9, 18.6)*
(Mental illness) was anyone living in your home depressed, mentally ill, or suicidal?	27.3 (23.1, 31.5)	19.6 (16.7, 22.5)*	16.5 (14.1, 18.9)*
# of Adverse childhood experiences (ACE score)			
0	33.0 (29.0, 37.0)	36.4 (32.9, 39.9)	44.4 (41.0, 47.8)*
1	12.5 (10.0, 14.9)	16.9 (14.2, 19.6)	19.8 (17.1, 22.4)*
2	12.0 (9.3, 14.6)	11.0 (9.0, 13.1)	13.1 (10.7, 13.1)
3	19.1 (15.5, 22.6)	22.9 (19.7, 26.1)	15.9 (13.1, 18.7)
4	23.5 (19.1, 27.9)	12.8 (10.6, 15.0)*	6.8 (5.3, 8.2)*

* indicates a significant difference compared to low LSES group, $p < .05$

Hypothesis 1.4: Compared to employed individuals, those unemployed or unable to work have a higher lifetime prevalence of CA. Results clearly indicate that those who were employed report much less CA than those who reported being "unable to work." Results were significant across all six types of adversity measured. For CM adversities the differences in prevalence between employed versus "unable to work" ranged from 4.2 times greater prevalence for sexual abuse to 1.6 times greater prevalence for emotional abuse. Physical abuse was in the middle with a 2.1 times greater prevalence for those "unable to work" compared to those employed. Likewise, there was nearly a 2 fold increase in prevalence for all household

dysfunction adversities when comparing those employed with those "unable to work." The ACE score revealed that those unable to work reported nearly a 3 fold increase in experiencing severe CA, with 34.6% having an ACE score of 4 compared to only 12.5% for those reporting being currently employed. Although those that were unemployed reported more adversity for each ACE compared to those employed, differences were not significant at alpha level 0.05.

Table 5-7 Age-Adjusted Prevalence of Adverse Childhood Experiences by Employment Status

Adverse childhood experience	Employed, % (95%CI)	Unemployed, % (95%CI)	Unable to Work, % (95%CI)
Childhood Maltreatment			
(When you were growing up, how often did a parent or adult living in your house. . .)			
(Emotional) sometimes, often or very often swear at you, insult you, or put you down?	25.7 (23.2, 28.2)	30.0 (23.6, 36.3)	40.8 (30.8, 50.9)*
(Physical) sometimes, often or very often hit, beat, kick, or physically hurt you?	17.6 (15.4, 19.9)	22.5 (17.2, 27.8)	36.2 (27.9, 44.5)*
(Sexual) ever touch you sexually or try to make you touch them sexually?	3.6 (2.1, 5.2)	4.9 (2.4, 7.2)	15.0 (8.2, 21.9)*
Household Dysfunction			
(When you were growing up, how often did a parent or adult living in your house. . .)			
(Mother treated violently) ever grab, slap, or throw something at your mother?	15.9 (13.9, 17.9)	16.5 (10.5, 22.5)	34.3 (24.8, 43.9)*
(When you were growing up as a child . . .)			
(Substance abuse) did you live with anyone who was a problem drinker, alcoholic, or drug user?	24.7 (22.2, 27.2)	30.8 (24.7, 36.9)	44.5 (34.3, 54.9)*
(Mental illness) was anyone living in your home depressed, mentally ill, or suicidal?	19.2 (16.9, 21.5)	23.9 (16.8, 31.1)	40.9 (30.6, 51.1)*
# of Adverse childhood experiences (ACE score)			
0	40.7 (37.9, 43.4)	26.0 (19.4, 32.6)*	30.4 (20.5, 40.3)
1	16.8 (14.9, 18.8)	20.6 (13.7, 27.4)	10.8 (5.7, 16.0)
2	11.9 (10.1, 13.6)	13.1 (8.2, 17.9)	4.5 (4.0, 11.0)
3	18.1 (16.0, 20.1)	26.3 (19.0, 33.6)	16.7 (7.8, 26.0)
4	12.5 (10.5, 14.6)	14.1 (10.1, 14.6)	34.6 (25.0, 44.1)*

* indicates a significant difference compared to employed group, $p < .05$

Hypothesis 1.5: Compared to those without disability, disabled individuals have a higher lifetime prevalence of CA. Across every CA there was a significant difference in

prevalence estimates ranging from a 2.7 fold difference in sexual abuse to a 1.5 fold difference for violence against mother. More revealing of the disparity in CA by disability status were the results comparing ACE scores. Those that reported disability also reported a 2.3 fold increase in experiencing severe adversity (i.e., ACE score of 4) and a 1.5 fold increase in experiencing moderate adversity (i.e., ACE score of 3) when compared to those without disability.

Table 5-8 Age-Adjusted Prevalence of Adverse Childhood Experiences by Disability Status

Adverse childhood experience	No Disability, % (95%CI)	Disability, % (95%CI)
Childhood Maltreatment		
(When you were growing up, how often did a parent or adult living in your house. . .)		
(Emotional) sometimes, often or very often swear at you, insult you, or put you down?	22.3 (20.3, 24.4)	40.7 (35.6, 45.7)
(Physical) sometimes, often or very often hit, beat, kick, or physically hurt you?	16.5 (14.7, 18.2)	31.6 (26.7, 36.4)
(Sexual) ever touch you sexually or try to make you touch them sexually?	3.2 (2.1, 4.4)	8.5 (6.1, 10.8)
Household Dysfunction		
(When you were growing up, how often did a parent or adult living in your house. . .)		
(Mother treated violently) ever grab, slap, or throw something at your mother?	15.4 (13.7, 17.1)	22.4 (18.7, 26.1)
(When you were growing up as a child . . .)		
(Substance abuse) did you live with anyone who was a problem drinker, alcoholic, or drug user?	23.4 (21.4, 25.4)	35.3 (30.4, 40.2)
(Mental illness) was anyone living in your home depressed, mentally ill, or suicidal?	16.8 (15.5, 18.7)	32.1 (27.2, 37.0)
# of Adverse childhood experiences (ACE score)		
0	42.9 (40.6, 45.0)	27.0 (22.8, 31.2)
1	17.2 (15.5, 18.9)	13.2 (10.7, 16.0)
2	12.2 (10.7, 13.6)	9.9 (7.6, 12.1)
3	17.2 (15.4, 19.1)	25.9 (20.9, 30.9)
4	10.6 (9.0, 12.1)	24.1 (20.1, 28.1)

Hypothesis 1.6: Compared to geographic areas with low unemployment (less than the county estimate of 6.8% unemployment), areas with high unemployment (greater or equal to 6.8%) have a higher lifetime prevalence of CA. A significant difference was found

between areas that had high versus low unemployment such that areas with high unemployment reported growing up with someone who was depressed or suicidal more so than areas with low unemployment. No other significant differences in prevalence were observed, although across all ACE types the point estimates were higher for areas with high unemployment.

Table 5-9 Age-Adjusted Prevalence of ACEs by Census Tract Percent Unemployment

Adverse childhood experience	Low Unemployment, % (95%CI)	High Unemployment, % (95%CI)
Childhood Maltreatment		
(When you were growing up, how often did a parent or adult living in your house. . .)		
(Emotional) sometimes, often or very often swear at you, insult you, or put you down?	17.7 (15.3, 20.1)	20.7 (19.7, 23.4)
(Physical) sometimes, often or very often hit, beat, kick, or physically hurt you?	23.6 (21.0, 26.1)	29.0 (25.8, 32.2)
(Sexual) ever touch you sexually or try to make you touch them sexually?	3.5 (2.1, 4.9)	4.6 (3.3, 5.9)
Household Dysfunction		
(When you were growing up, how often did a parent or adult living in your house. . .)		
(Mother treated violently) ever grab, slap, or throw something at your mother?	17.6 (14.8, 20.3)	16.5 (13.7, 19.2)
(When you were growing up as a child . . .)		
(Substance abuse) did you live with anyone who was a problem drinker, alcoholic, or drug user?	23.7 (21.0, 26.5)	27.6 (24.7, 30.5)
(Mental illness) was anyone living in your home depressed, mentally ill, or suicidal?	17.2 (14.9, 19.6)	22.9 (20.1, 25.7)
# of Adverse childhood experiences (ACE score)		
0	41.3 (38.2, 44.4)	36.2 (33.3, 39.1)
1	16.9 (14.3, 19.5)	17.6 (15.2, 20.0)
2	11.9 (10.1, 13.9)	11.7 (9.6, 13.9)
3	17.9 (15.6, 20.2)	20.1 (17.1, 23.1)
4	12.0 (9.9, 14.0)	14.3 (12.1, 16.6)

Hypothesis 1.7: Compared to geographic areas with a low proportion of individuals living below the poverty line, areas with a high proportion of individuals living below the poverty line (greater than 5.2%) will have a higher lifetime prevalence of CA. No

significant differences in prevalence were observed (Table 5-10), although across all ACE types the point estimates were higher for areas with a greater percentage of residents living at or below the poverty line.

Table 5-10 Age-Adjusted Prevalence of ACEs by Census Tract Percent Poverty

Adverse childhood experience	Low Poverty, % (95%CI)	High Poverty, % (95%CI)
Childhood Maltreatment		
(When you were growing up, how often did a parent or adult living in your house. . .)		
(Emotional) sometimes, often or very often swear at you, insult you, or put you down?	17.3 (14.8, 19.7)	20.9 (18.3, 23.5)
(Physical) sometimes, often or very often hit, beat, kick, or physically hurt you?	24.6 (21.9, 27.3)	27.7 (24.7, 30.8)
(Sexual) ever touch you sexually or try to make you touch them sexually?	3.7 (2.2, 5.2)	.4 (3.2, 5.6)
Household Dysfunction		
(When you were growing up, how often did a parent or adult living in your house. . .)		
(Mother treated violently) ever grab, slap, or throw something at your mother?	17.0 (14.3, 19.7)	17.2 (14.4, 19.9)
(When you were growing up as a child . . .)		
(Substance abuse) did you live with anyone who was a problem drinker, alcoholic, or drug user?	23.4 (20.6, 26.2)	27.7 (24.8, 30.5)
(Mental illness) was anyone living in your home depressed, mentally ill, or suicidal?	18.6 (16.1, 21.2)	21.3 (18.6, 23.9)
# of Adverse childhood experiences (ACE score)		
0	40.3 (37.2, 43.4)	37.5 (34.6, 40.4)
1	18.7 (15.9, 21.5)	15.9 (13.9, 17.9)
2	12.0 (10.0, 14.0)	11.7 (9.6, 13.7)
3	17.0 (14.7, 19.4)	20.8 (18.0, 23.7)
4	12.0 (9.9, 14.1)	14.2 (12.0, 16.3)

Hypothesis 1.8: Compared to geographic areas with high proportion of non-Hispanic Whites, areas with a high proportion non-Hispanic Blacks (defined as areas with greater than a 32% non-Hispanic Black population) have a higher lifetime prevalence of CA. Again, no significant differences in prevalence were observed (Table 5-11), although across

all ACE types the point estimates were higher for areas with a greater percentage of Black residents.

Table 5-11 Age-Adjusted Prevalence of ACEs by Census Tract Percent Black

Adverse childhood experience	Low Black Pop., % (95%CI)	High Black Pop., % (95%CI)
Childhood Maltreatment		
(When you were growing up, how often did a parent or adult living in your house. . .)		
(Emotional) sometimes, often or very often swear at you, insult you, or put you down?	18.4 (16.3, 20.6)	21.9 (18.1, 25.7)
(Physical) sometimes, often or very often hit, beat, kick, or physically hurt you?	25.3 (22.9, 27.7)	29.3 (24.8, 33.7)
(Sexual) ever touch you sexually or try to make you touch them sexually?	4.0 (2.8, 5.2)	4.7 (2.7, 6.6)
Household Dysfunction		
(When you were growing up, how often did a parent or adult living in your house. . .)		
(Mother treated violently) ever grab, slap, or throw something at your mother?	16.8 (14.6, 19.1)	18.0 (14.1, 21.9)
(When you were growing up as a child . . .)		
(Substance abuse) did you live with anyone who was a problem drinker, alcoholic, or drug user?	25.5 (23.1, 28.0)	27.6 (23.7, 31.4)
(Mental illness) was anyone living in your home depressed, mentally ill, or suicidal?	19.6 (17.4, 21.9)	22.4 (18.8, 26.0)
# of Adverse childhood experiences (ACE score)		
0	40.0 (37.4, 42.6)	34.8 (31.0, 38.6)
1	17.6 (15.4, 19.9)	16.2 (13.3, 19.2)
2	11.6 (9.9, 13.3)	12.2 (9.2, 15.1)
3	18.4 (16.2, 20.5)	20.8 (16.8, 24.8)
4	12.4 (10.6, 14.2)	16.0 (12.8, 19.3)

5.2 ATTRIBUTABLE AND POPULATION ATTRIBUTABLE RISKS

Table 5-12 provides the results from the bivariate and multivariate analyses which provide measures of association between having an ACE score 1 and the odds of reporting negative health indicator. In bivariate analyses, 20 of the 25 health indicators (80%) were significantly

correlated with ACE scores in the expected direction (i.e., the odds of poor health increased with increasing CA). In multivariate analyses, while controlling for age, gender, race, and lifetime SES, 18 of the 25 (72%) indicators remained significant. Of those that remained significant, mental and social health indicators were most correlated with reported ACEs; specifically, Serious Mental Illness (AOR = 1.78), Not Good Mental Health Days in the past 30 days (AOR = 1.59) Life Dissatisfaction (AOR = 1.51) and low Perceived Social Support (AOR 1.33). Physical health indicators most associated with ACE score included CVD (AOR = 1.28) and Not Good Physical Health Days >=14 in the past 30 days (AOR =1.38). Least associated was Diabetes (AOR = 1.08) and Cancer (AOR = 1.08). The odds ratios for health behaviors such as Cancer Screening, Smoking and Alcohol Use largely fell in between mental/social indicators and physical health outcomes with AORs ranging from 1.09 for Mammogram Non-compliance to 1.25 for Smoking. Cholesterol, Physical Activity, No Health Care Provider, Fruits and Vegetable Consumption, Hypertension, Asthma, and having No Insurance were *not* significantly correlated with ACEs in multivariate models.

Table 5-12 Odds and Adjusted Odds Ratios of Reporting a Negative Health Indicator: Comparing those with an ACE Score of 1versus those with No Reported ACEs

Health Indicator	Bivariate Logistic			Multivariate Logistic*		
	OR	LCL	UCL	AOR	LCL	UCL
Diabetes	1.00	0.94	1.07	1.08	1.00	1.17
Cancer	0.94	0.88	1.00	1.08	1.01	1.16
No check-up past year	1.12	1.06	1.19	1.09	1.03	1.16
Mammogram non-compliance	1.08	1.02	1.15	1.09	1.02	1.17
Overweight/Obese	1.08	1.02	1.15	1.12	1.06	1.19
Pap Test non-compliance	1.14	1.05	1.25	1.12	1.02	1.24
Alcohol Abuse (>4 binge drinking past month)	1.21	1.09	1.36	1.17	1.03	1.32
Multiple sex partners 2+	1.29	1.14	1.45	1.22	1.07	1.40
Smoking (ever 100 lifetime)	1.25	1.19	1.32	1.28	1.21	1.35
Venereal disease	1.43	1.15	1.78	1.28	1.08	1.52
Cardiovascular Disease	1.06	0.99	1.13	1.28	1.19	1.39
Fair/Poor Health	1.20	1.13	1.28	1.30	1.22	1.39
Not good Physical health days >=14	1.27	1.19	1.36	1.31	1.21	1.41
No Access due to cost	1.50	1.37	1.65	1.33	1.21	1.45
Low perceived social support	1.33	1.26	1.41	1.33	1.25	1.41
Life dissatisfaction	1.48	1.39	1.58	1.51	1.41	1.61
Not good Mental health days >=14	1.64	1.52	1.76	1.59	1.46	1.73
Serious Mental Illness (K6)	1.93	1.72	2.17	1.78	1.57	2.01
Cholesterol	0.89	0.83	0.96	0.97	0.89	1.05
Physical Activity	1.00	0.95	1.05	0.97	0.92	1.03
No health care provider	1.11	1.02	1.20	1.02	0.93	1.12
Fruits and vegetable consumption	1.07	0.96	1.19	1.03	0.92	1.15
Hypertension	0.94	0.89	0.99	1.05	0.99	1.12
Asthma	1.11	1.03	1.20	1.07	0.98	1.17
No insurance	1.18	1.08	1.30	1.09	0.98	1.22

* adjusted for age, race, gender, and lifetime SES

Table 5-13 presents the adjusted odds ratios by ACE score for all health indicators that were significantly correlated with ACE score in multivariate models. Compared to those reporting no ACEs, those with an ACE score of 4 had nearly a 10 fold increase in the odds of reporting serious mental illness, a 6.4 fold increase in the odds of reporting greater than or equal

to 14 days of Not Good Mental Health Days in the past 30 days, and a 3.4 fold increase in the odds of Life Dissatisfaction.

Table 5-13 Adjusted Odds Ratios for Health Indicators by ACE Score

Health Indicator	AOR ACE1	AOR ACE2	AOR ACE3	AOR ACE4
Diabetes	1.08	1.18	1.27	1.38
Cancer	1.08	1.18	1.27	1.38
No check-up past year	1.09	1.19	1.30	1.42
Mammogram non-compliance	1.09	1.20	1.31	1.43
Overweight/Obese	1.12	1.25	1.40	1.57
Pap Test non-compliance	1.13	1.27	1.43	1.61
Alcohol Abuse (>4 binge drinking past month)	1.17	1.36	1.59	1.85
Multiple sex partners 2+	1.22	1.49	1.82	2.23
Smoking (ever 100 lifetime)	1.28	1.63	2.09	2.67
Venereal disease	1.28	1.64	2.09	2.68
Cardiovascular Disease	1.28	1.64	2.11	2.70
Fair/Poor Health	1.30	1.69	2.20	2.86
Not good Physical health days >=14	1.31	1.70	2.22	2.90
No Access due to cost	1.33	1.76	2.33	3.09
Low perceived social support	1.33	1.77	2.35	3.12
Life dissatisfaction	1.51	2.27	3.42	5.14
Not good Mental health days >=14	1.59	2.53	4.02	6.39
Serious Mental Illness (K6)	1.78	3.16	5.61	9.97

Attributable Risk (AR) is the proportion of disease risk that can be attributed to a specific exposure. Table 5-14 presents the AR percentages by ACE score for each of the health indicators that were significant in multivariable models above. With an ACE score of 4, the percentage of excess cases attributable to exposure to ACEs ranged from 28% to 90%, meaning that if everyone in the population had an ACE score of 4, removing that exposure would decrease the prevalence by 28% to 90% depending on the health indicator of focus. For several of the mental

health indicators more than 50% of the excess cases were attributable to exposure to only two ACEs.

Table 5-14 Attributable Risks for Health Indicators Associated with Exposure to ACEs

Health Indicator	AR ACE1	AR ACE2	AR ACE3	AR ACE4
Diabetes	0.08	0.15	0.21	0.28
Cancer	0.08	0.15	0.21	0.28
No check-up past year	0.08	0.16	0.23	0.29
Mammogram non-compliance	0.09	0.16	0.24	0.30
Overweight/Obese	0.11	0.20	0.29	0.36
Pap Test non-compliance	0.11	0.21	0.30	0.38
Alcohol Abuse (>4 binge drinking past month)	0.14	0.27	0.37	0.46
Multiple sex partners 2+	0.18	0.33	0.45	0.55
Smoking (ever 100 lifetime)	0.22	0.39	0.52	0.63
Venereal disease	0.22	0.39	0.52	0.63
Cardiovascular Disease	0.22	0.39	0.53	0.63
Fair/Poor Health	0.23	0.41	0.55	0.65
Not good Physical health days >=14	0.23	0.41	0.55	0.66
No Access due to cost	0.25	0.43	0.57	0.68
Low perceived social support	0.25	0.43	0.57	0.68
Life dissatisfaction	0.34	0.56	0.71	0.81
Not good Mental health days >=14	0.37	0.60	0.75	0.84
Serious Mental Illness (K6)	0.44	0.68	0.82	0.90

Table 5-15 provides the PAR fractions which take into account the level of ACE exposure in the population and provide information about how much of a reduction in morbidity could be expected in the population if ACEs were prevented. The PAR fractions ranged from 10.6% for diabetes and cancer to 41.3% for SMI. PAR fractions were significant in all health indicators examined except for Diabetes and Cancer where the 95%CL included zero. The results indicate that if all ACEs were eliminated, a reduction of 41.3% in SMI and a 26.1% reduction in CVD could be expected in this population.

Table 5-15 Population Attributable Risks for Health Indicators Associated with Exposure to ACEs

Health Indicator	PAR	PAR lower	PAR upper
Diabetes	10.6%	-1.5%	19.9%
Cancer	10.6%	-0.3%	19.1%
No check-up past year	11.3%	2.0%	18.6%
Mammogram non-compliance	11.6%	1.2%	19.7%
Overweight/Obese	14.2%	5.8%	20.6%
Pap Test non-compliance	14.7%	1.4%	24.4%
Alcohol Abuse (>4 binge drinking past month)	18.3%	1.8%	29.2%
Multiple sex partners 2+	22.4%	5.7%	32.7%
Smoking (ever 100 lifetime)	25.8%	19.6%	30.0%
Venereal disease	25.9%	6.0%	36.9%
Cardiovascular Disease	26.1%	17.4%	31.8%
Fair/Poor Health	27.1%	19.8%	31.9%
Not good Physical health days >=14	27.3%	19.1%	32.7%
No Access due to cost	28.4%	19.1%	34.4%
Low perceived social support	28.5%	22.2%	32.6%
Life dissatisfaction	35.4%	30.2%	38.5%
Not good Mental health days >=14	37.7%	32.2%	40.8%
Serious Mental Illness (K6)	41.3%	35.3%	44.5%

5.3 MEDIATORS AND MODERATORS

Focus for this objective was restricted to the mental and physical health outcomes most associated with ACEs as determined above. In this population Serious Mental Illness (SMI) and Cardiovascular Disease (CVD) were attributable to ACEs more so than other mental and physical health indicators evaluated; thus these two were evaluated to determine the potential pathways by which ACEs impact adult health. When mediation was found to be present, moderated mediation analyses were conducted to evaluate for whom and in what contexts ACEs were more or less impactful on adult health.

ACEs and CVD

Is the relationship between ACEs and CVD mediated by ASES? The relationship between ACE Score and CVD was fully mediated by ASES such that the direct effect (*c'*) of ACEs on CVD was no longer significant when ASES was included in the model (Table 5-16). The indirect effect accounted for 46% of the total effect ACEs have on CVD and the ratio of the indirect to direct effect was 78%.

Table 5-16 Model Coefficients for Mediation of the ACE-CVD Relationship by ASES

	Dependent Variable							
	M (ASES)				Y (CVD)			
Independent Variable		**Coeff.**	**SE**	**p**		**Coeff.**	**SE**	**p**
X (ACE Score)	*a*	-0.077	0.0165	<.0001	*c'*	0.0344	0.0296	0.2443
M (ASES)		__	__	__	*b*	-0.3466	0.0277	<.0001
	i_1	0.1563	0.0165	<.0001	i_2	-2.0991	0.0634	<.0001
***ab* = 0.0267 Bootstrap CL (0.0158,0.0387)**								

Is the direct or indirect effect of ACEs on CVD through ASES the same across demographic and neighborhood characteristics? No significant interactions of the direct or indirect effects by gender or race were observed. The indirect effect of ACEs on CVD through ASES was not moderated by perceived neighborhood level characteristics, either. However, the indirect effect that ACEs had on CVD through ASES was a function of age. A significant age interaction of the indirect effect was observed such that those that experienced ACEs and were younger in age were more negatively affected in terms of ASES then those who were older in age (a_3 in Table 5-17). Probing the interaction revealed that the indirect effect of ACEs on CVD

through ASES was age conditional. Using plus and minus one standard deviation age to probe the interaction, it became apparent that the indirect effect was only significant at lower ages i.e., at or below age 56 (see Table 5-18).

Table 5-17 Model Coefficients for Moderated Mediation of the ACE-ASES-CVD Relationship by AGE

	Dependent Variable							
	M (ASES)				Y (CVD)			
Independent Variable		**Coeff.**	**SE**	**p**		**Coeff.**	**SE**	**p**
X (ACE Score)	a_1	-0.514	0.109	<.0001	c_1'	0.3642	0.1414	0.01
M (ASES)		__	__	__	b	-0.2187	0.0299	<.0001
Z (AGE)	a_2	-0.0345	0.0018	<.0001	c_2'	0.0592	0.0048	<.0001
X*Z	a_3	0.0071	0.001	<.0001	c_3'	-0.0026	0.0021	0.2326
Constant	i_1	2.175	0.109	<.0001	i_2	-5.9827	0.3397	<.0001

Table 5-18 Conditional indirect effects of ACEs on CVD through ASES at values of AGE

	Age	**Effect**	**Boot SE**	**BootLLCI**	**BootULCI**
ASES	38.9941	0.0516	0.0092	0.0358	0.0718
ASES	56.1025	0.0250	0.0052	0.0161	0.0376
ASES	73.2109	-0.0017	0.0053	-0.0124	0.0088

A significant CSES interaction of the indirect effect of ACEs on CVD through ASES was also observed such that when holding ACEs constant, as CSES increased there was a significant negative associate between CSES and ASES (a_3 in Table 5-19). Meaning that for those with low CSES, ACEs did not have a significant effect on CVD through ASES, whereas for those with middle to high CSES, the effect ACEs have on CVD through ASES was significant. That is to say, for those with higher CSES, a higher ACE score resulted in a significant decrease in ASES which in turn increased the association between ACES and CVD. For those with lower CSES the effect of ACEs on CVD was more direct (see Table 5-20).

Table 5-19 Model Coefficients for Moderated Mediation of the ACE-ASES-CVD Relationship by CSES

	Dependent Variable							
	M (ASES)				Y (CVD)			
Independent Variable		Coeff.	SE	p		Coeff.	SE	p
X (ACE Score)	a_1	0.0490	0.0502	0.3287	c_1'	0.0009	0.094	0.0092
M (ASES)		__	__	__	b	-0.251	0.0299	<.0001
Z (CSES)	a_2	0.8888	0.0437	<.0001	c_2'	-0.5186	0.1004	<.0001
X*Z	a_3	-0.0685	0.022	0.0018	c_3'	0.0345	0.0457	0.4507
Constant	i_1	-1.5745	0.1006	<.0001	i_2	-1.1211	0.2056	<.0001

Table 5-20 Conditional indirect effects of ACEs on CVD through ASES at values of CSES

Mediator	CSES	Effect	Boot SE	BootLLCI	BootULCI
ASES	Low	0.0049	0.0079	-0.0099	0.0225
ASES	Middle	0.0221	0.0053	0.0135	0.0339
ASES	High	0.0393	0.0082	0.0243	0.0555

Is the relationship between ACEs and CVD mediated by adult health behaviors?

The relationship between ACEs and CVD was not mediated by Fruit and Vegetable consumption or by Physical Activity or alcohol Binge Drinking.

The relationship between ACE Score and CVD was fully mediated by BMI, such that the direct effect of ACEs on CVD was no longer significant when BMI was included in the model (*c'* in Table 5-21). The indirect effect accounted for 12% of the total effect ACEs have on CVD and the ratio of the indirect to direct effect was 13%. In moderated mediation analyses neither the direct or indirect effect of ACEs on CVD through BMI was moderated by gender, race, age, CSES or by perceived neighborhood level characteristics.

Table 5-21 Model Coefficients for Mediation of the ACE-CVD Relationship by BMI

	Dependent Variable							
	M (BMI)				Y (CVD)			
Independent Variable		Coeff.	SE	p		Coeff.	SE	p
X (ACE Score)	*a*	0.3588	0.0597	<.0001	*c'*	0.057	0.0298	0.0559
M (BMI)		__	__	__	*b*	0.0211	0.0071	0.003
Constant	i_1	0.4774	0.0534	<.0001	i_2	-2.6113	0.2085	<.0001
***ab* = 0.0076 (0.0026, 0.0136)**								

The relationship between **ACE Score and CVD was fully mediated by smoking status**, such that the direct effect of ACES on CVD was no longer significant when Smoking was included in the model (*c'* in Table 5-22). The indirect effect accounted for 31% of the total effect ACEs have on CVD and the ratio of the indirect to direct effect was 13%.

Table 5-22 Model Coefficients for Mediation of the ACE-CVD Relationship by ASES Smoking

	Dependent Variable							
	M (Smoking)				Y (CVD)			
Independent Variable		Coeff.	SE	p		Coeff.	SE	p
X (ACE Score)	*a*	0.1389	0.0102	<.0001	*c'*	0.0382	0.0299	0.2018
M (Smoking)		__	__	__	*b*	0.128	0.0399	0.0013
Constant	i_1	0.6923	0.0204	<.0001	i_2	-2.1183	0.0683	<.0001
***ab* = 0.0178 (0.0078, 0.0291)**								

The indirect effect of ACEs on CVD through Smoking was moderated by age (see Tables 5-23 and 5-24). As age increased, the indirect effect of ACES on CVD through Smoking decreased significantly. At age 73 the mediated effect (.0275) of Smoking on CVD was approximately half of the effect at age 39 (.0421). No significant interactions were observed by gender, race, CSES, or by perceived neighborhood level characteristics.

Table 5-23 Model Coefficients for Moderated Mediation of the ACE-Smoking-CVD Relationship by AGE

	Dependent Variable							
	M (Smoking)				Y (CVD)			
Independent Variable		Coeff.	SE	p		Coeff.	SE	p
X (ACE Score)	*a1*	0.2184	0.0354	<.0001	*c1'*	0.4364	0.1446	0.0025
M (Smoking)		__	__	__	*b*	0.2697	0.0458	<.0001
Z (AGE)	*a2*	-0.0012	0.0012	0.2827	*c2'*	0.0714	0.0048	<.0001
X*Z	*a3*	-0.0016	0.0006	0.0112	*c3'*	-0.0039	0.0022	0.0769
Constant	*i1*	0.7697	0.0706	<.0001	*i2*	-6.9316	0.3537	<.0001

Table 5-24 Conditional indirect effects of ACEs on CVD through Smoking at values of AGE

Mediator	**AGE**	**Effect**	**Boot SE**	**BootLLCI**	**BootULCI**
SMOKE	38.9674	0.0421	0.0082	0.0268	0.0594
SMOKE	56.0746	0.0348	0.0063	0.0229	0.0479
SMOKE	73.1818	0.0275	0.0058	0.0174	0.0399

Is the relationship between ACEs and CVD mediated by Perceived Social Support?

The relationship between ACE Score and CVD was fully mediated by Social Support, such that the direct effect of ACEs on CVD was no longer significant when Social Support was included in the model (see Table 5 25). The indirect effect accounted for 63% of the total effect ACEs have on CVD. The ratio of the indirect effect to direct effect was 1.83 which means that the indirect effect was 83% larger than the direct effect of ACEs on CVD. A significant age interaction for the direct effect (see c3' in Table 5 26) of ACEs on CVD while controlling for Social Support was observed, indicating simply that as age increased the effects of ACEs on CVD decreased. No significant interactions by race, gender, CSES, or perceived neighborhood level characteristics were observed.

Table 5-25 Model Coefficients for Mediation of the ACE-CVD Relationship by Social Support

	Dependent Variable							
	M (Social Support)				Y (CVD)			
Independent Variable		Coeff.	SE	p		Coeff.	SE	P
X (ACE Score)	*a*	-0.5572	0.0346	<.0001	*c'*	0.0193	0.0305	0.5276
M (Social Support)		__	__	__	*b*	-0.0634	0.0117	<.0001
Constant	*i1*	13.4042	0.0692	<.0001	*i2*	-1.1989	0.1625	<.0001
***ab* = 0.0353 (0.0212, 0.0484)**								

Table 5-26 Model Coefficients for Moderated Mediation of the ACE-Social Support-CVD Relationship by AGE

	Dependent Variable							
	M (Social Support)				Y (CVD)			
Independent Variable		Coeff.	SE	p		Coeff.	SE	p
X (ACE Score)	*a1*	-0.5591	0.2372	<.0001	*c1'*	0.4796	0.1452	0.001
M (Social Support)		__	__	__	*b*	-0.0307	0.0124	0.0132
Z (AGE)	*a2*	-0.0278	0.0039	<.0001	*c2'*	0.0688	0.0048	<.0001
X*Z	*a3*	-0.001	0.0021	0.6192	*c3'*	-0.0044	0.0022	0.043
Constant	*i1*	15.0334	0.0706	<.0001	*i2*	-6.1434	0.3919	<.0001

ACEs and SMI

Is the relationship between ACEs and SMI mediated by ASES? The relationship between ACE Score and SMI was *partially* mediated by ASES such that the direct effect of ACEs on SMI was attenuated but remained significant when ASES was included in the model (see *c'* in Table 5-27). The indirect effect accounted for 5% of the total effect and the ratio of the indirect to direct effect was also 5%. No significant interactions by gender or race were observed.

Table 5-27 Model Coefficients for Mediation of the ACE-SMI Relationship by ASES

	Dependent Variable							
	M (ASES)				Y (SMI)			
Independent Variable		Coeff.	SE	p		Coeff.	SE	p
X (ACE Score)	*a*	-0.0773	0.0164	<.0001	*c'*	0.6451	0.0547	<.0001
M (ASES)		__	__	__	*b*	-0.4324	0.0503	<.0001
Constant	*i1*	0.1486	0.0326	<.0001	*i2*	-4.7368	0.1723	<.0001
***ab* = 0.0334 (0.0183, 0.0516)**								

There were significant AGE and CSES interactions for the indirect effect of ACEs on SMI through ASES (see Table 5-28). AGE moderated the effect of ACEs on ASES such that the effect of ACES on SMI through ASES decreased with increasing AGE. At age 73, the indirect effect was no longer significant (see Table 5-29).

Table 5-28 Model Coefficients for Moderated Mediation of the ACE-ASES-SMI Relationship by AGE

	Dependent Variable							
	M (ASES)				Y (SMI)			
Independent Variable		Coeff.	SE	p		Coeff.	SE	p
X (ACE Score)	*a1*	-0.515	0.0543	<.0001	*c1'*	0.4282	0.1686	0.0111
M (ASES)		__	__	__	*b*	-0.466	0.051	<.0001
Z (AGE)	*a2*	-0.0348	0.0018	<.0001	*c2'*	-0.0267	0.0048	<.0001
X*Z (ACE Score* AGE)	*a3*	0.0071	0.001	<.0001	*c3'*	-0.0044	0.0088	0.0025
Constant	*i1*	2.1893	0.1085	<.0001	*i2*	0.0031	0.0031	0.3149

Table 5-29 Conditional indirect effects of ACEs on SMI through ASES at values of AGE

Conditional indirect effects of ACEs on SMI at values of AGE					
	AGE	**Effect**	**Boot SE**	**BootLLCI**	**BootULCI**
ASES	39.0727	0.1105	0.0165	0.0814	0.1447
ASES	56.2099	0.0538	0.0094	0.0357	0.0733
ASES	73.3471	-0.0030	0.0113	-0.0262	0.0180

For those with low CSES the indirect effect was non-significant whereas for those with Middle to High CSES the indirect effect was significant (see Table 5-30 and Table 5-31). Neither the direct effect of ACES on SMI nor the indirect effect of ACEs on SMI through ASES was moderated by perceived neighborhood level characteristics.

Table 5-30 Model Coefficients for Moderated Mediation of the ACE-ASES-SMI Relationship by CSES

	Dependent Variable							
	M (ASES)				Y (SMI)			
Independent Variable		Coeff.	SE	p		Coeff.	SE	p
X (ACE Score)	*a1*	0.0447	0.0499	0.3702	*c1'*	0.7489	0.1823	<.0001
M (ASES)		__	__	__	*b*	-0.4679	0.0563	<.0001
Z (CSES)	*a2*	0.8861	0.8861	<.0001	*c2'*	0.2376	0.2509	0.3435
X*Z	*a3*	-0.0669	0.0219	0.0022	*c3'*	-0.0628	0.0829	0.4489
Constant	*i1*	-1.5711	0.1001	<.0001	*i2*	-5.1409	0.5631	<.0001

Table 5-31 Conditional indirect effects of ACEs on SMI through ASES at values of CSES

Conditional indirect effects of ACEs on SMI at values of CSES					
	CSES	**Effect**	**Boot SE**	**BootLLCI**	**BootULCI**
ASES	Low	0.0104	0.0144	-0.0177	0.0396
ASES	Middle	0.0417	0.0094	0.0254	0.0628
ASES	High	0.0730	0.0152	0.0480	0.1063

Is the relationship between ACEs and SMI mediated by adult health behaviors? The relationship between ACEs and SMI was not mediated by Fruit and Vegetable consumption or Physical Activity, Alcohol, or BMI. Only Smoking *partially* mediated the effect of ACEs on SMI (see *c'* in Table 5-32). The indirect effect accounted for 9% of the total effect and the ratio of the indirect to direct effect was 1%. No significant interactions of the direct or indirect effect were observed.

Table 5-32 Model Coefficients for Mediation of the ACE-SMI Relationship by Smoking

	Dependent Variable							
	M (Smoking)				Y (SMI)			
Independent Variable		Coeff.	SE	p		Coeff.	SE	p
X (ACE Score)	*a*	0.138	0.0102	<.0001	*c'*	0.6127	0.0558	<.0001
M (Smoking)		__	__	__	*b*	0.4476	0.0628	<.0001
Constant	i_1	-4.9915	0.0203	<.0001	i_2	-4.7368	0.1833	<.0001
***ab* = 0.0618 (0.0418, 0.0821)**								

Is the relationship between ACEs and SMI mediated by Social Support? The relationship between ACE Score and SMI was partially mediated by social support, such that the direct effect of ACEs on SMI was significantly attenuated when social support was included in the model (see Table 5-33). The indirect effect accounted for 16% of the total effect and the ratio of the indirect to direct effect was 20%.

Table 5-33 Model Coefficients for Mediation of the ACE-SMI Relationship by Social Support

	Dependent Variable							
	M (Social Support)				Y (SMI)			
Independent Variable		Coeff.	SE	p		Coeff.	SE	p
X (ACE Score)	*a*	-0.5564	0.0344	<.0001	*c'*	0.5815	0.0573	<.0001
M (Social Support)		__	__	__	*b*	-0.2042	0.0185	<.0001
Constant	i_1	13.3956	0.0688	<.0001	i_2	-2.1613	0.2572	<.0001
***ab* = 0.1136 (0.0909, 0.1419)**								

Table 5-34 below summarizes the results for the mediation analyses in terms of effect size. The effect size in this table is the ratio of the indirect effect to the total effect. Social Support had the largest effect size for both CVD and SMI. The indirect effect of ACEs on CVD through ASES accounted for nearly half the total effect whereas the indirect effect of ACEs on SMI through ASES accounted for only 5%. Smoking was a significant mediator for both as well

and BMI was significant for only CVD. Alcohol, Fruits and Vegetable conception, and Physical Activity had no mediating effect.

Table 5-34 Summary of Effect Sizes for Mediated Pathways

	Dependent Variables	
	CVD	**SMI**
Mediators	**Effect Size**	**Effect Size**
Social Support	63%	16%
ASES	46%	5%
Smoking	31%	9%
BMI	12%	n.s.
Alcohol	n.s.	n.s.
Fruits and Vegetables	n.s.	n.s.
Physical Activity	n.s.	n.s.

6.0 DISCUSSION

Consistent with national and state estimates Adverse Childhood Experiences (ACEs) as measured in the ACHS were prevalent in the adult population of Allegheny County, Pennsylvania with 59.8% reporting at least one ACE. As hypothesized, ACEs were not equally distributed across social and demographic characteristics. Females, minorities, those with disabilities, and those with lower SES reported greater prevalence of ACEs.

These childhood adversity (CA) disparities may partially account for current disparities in adult health such as gender, racial, and SES health disparities in cardiovascular disease (CVD) and serious mental illness (SMI). Results clearly showed an association between reported ACEs and many of the adult health indicators measured, including smoking, alcohol binge drinking, mental illness, quality of life, and CVD. Moreover the percentage of morbidity in the adult population that can be attributable to ACEs was quite high with 41.3% of SMI and 26.1% of CVD being attributable to ACEs.

The pathways that lead from ACEs to increased CVD and SMI prevalence were examined and perceived social support, adult SES and smoking appeared to be significant factors mediating both relationships. BMI mediated the relationship between ACEs and CVD only. Outside of age and childhood SES, these mediated pathways were not significantly different when comparing across demographic or neighborhood characteristics.

Direct effects of ACEs on mental health support a critical or sensitive period model. The effect of ACEs on CVD was not fully mediated by adult behavior, social support, or adult SES. Physical health outcomes appeared to be influenced more so by adult SES (ASES) social support and behavior and thus indicate more plasticity in support of accumulation models. Study aims

are discussed below in light of current research, the life course perspective, and with regard to the public health significance of the findings.

Prevalence of ACEs

Overall, the estimates of childhood adversity (CA) in the Allegheny County population are similar to other national and state estimates. Kessler found that 75% of the national adult population had experienced at least 1 childhood adversity.(4) In the ACHS study only 59.8% reported at least 1 adversity, but this is likely a result of the adversity measures used in each of studies and not an actual difference. In Kessler's study the adversity measure included many more types of adversities such as divorce, natural disasters, parental death, and witness to violence, whereas the focus of the ACHS adversity measure focused solely on child maltreatment (CM) and household dysfunction. When compared to the original ACE study (3) and the BRFSS ACE surveys (28) the ACHS results are quite comparable. Felitti et al. found that 52% of the population experienced at least 1 ACE and in the CDC BRFSS study of 5 states 59% reported at least one ACE. One major difference to note is the prevalence of sexual abuse. In the original ACE Study 22% reported sexual abuse and in the CDC BRFSS, 12% reported sexual abuse. These are much higher than the 4.1% reported in the ACHS. These differences in estimates are likely due to the way in which questions were asked rather than real differences in prevalence of sexual abuse (i.e., ACHS restricted sexual abuse to "in home" abuse whereas other studies asked about any sexual abuse that may have occurred).

In 2010 the Pennsylvania state BRFSS included CA measures for the first time (See Table 1). A direct comparison is difficult given the textual differences and the scope of the questions. For example in the PA BRFSS the questions about alcohol and drug use were asked

separately and in the ACHS study they were asked together in a single question. One of the only questions that was nearly identical and thus comparable was "lived with someone who was depressed, mentally ill, or suicidal." On this measure the ACHS point estimate was higher 20.0% (95% CI: 18, 21) versus the state estimate of 16% (95% CI: 15, 18) but this difference was not statistically significant. The comparability of the ACHS results with national and state estimates gives credence to the methods and reliability of the ACHS survey.

ACEs type and severity by demographic, social, and geographic characteristics.

Significant gender differences in CA were restricted to sexual abuse, with females reporting a 4.6 fold greater prevalence than males. This is consistent with recent and past NIS finding.(91, 92) Given the strong relationship between sexual abuse and psychopathologies, the increased female exposure to childhood sexual abuse may account for gender based health disparities observed in adulthood. Rohde et al. (52) found that childhood sexual abuse was related to a doubling of the odds of both depression and obesity in a population-based sample of 4641 middle-aged women. From a life course perspective, sexual abuse in childhood could lead to a number of social and behavioral problems that in turn affect education, social relationships, employment, and marital status all of which are related to adult health in some meaningful way. Gender disparities in adult depression may be, in part, a result of childhood sexual abuse. Likewise racial disparities in obesity and diabetes may be explained by events that occurred years or decades ago. Using case control or longitudinal study designs that compare genders or racial groups across exposure type and overtime could help to elucidate health disparities and specific pathways that may be a result of differential exposure to CA.

The ACHS results indicate a clear racial difference in physical and sexual abuse with non-Hispanic Blacks reporting 2 and 3 times the prevalence of physical and sexual abuse, respectively, compared to their non-Hispanic-White counterparts. These results are similar to estimates in the most recent NIS IV which measured incidence of CM and found African Americans had an incidence rate double that of Whites.(91) No statistical differences by race were observed for emotional abuse or any of the other household dysfunction adversities. Non-Hispanic Blacks did, however, report experiencing more severe CA with 16.8% reporting an ACE score of 4 compared to only 12.4% for non-Hispanic Whites. There is some debate about whether or not observed CM disparities exist by race or socioeconomic status.(32) One might suspect that if CA disparities were due to SES then one would see significant increases across all adversities for those in the lower SES stratum compared to those in the middle and high SES stratum, as low SES environments typically expose individuals and families to a host of environmental and psychosocial stressors. Given that this was not the case in the ACHS data (See Table 5-5), racial disparities in CA may be better explained by additional adversities and social factors not measured in this survey but that are related to occurrence of CA, including racial discrimination, parental incarceration, and family structure. More globally then, as Barclay and Braveman purport, social stratification by race and class likely leads to differential exposure of CA, differential vulnerabilities, and differential health consequences, all of which in turn may lead to further social stratification and an increased likelihood of CA exposure.(40)

Analyses of the ACEs by childhood socioeconomic status (CSES) indicated differences across CSES strata with ACEs increasing as CSES decreased. Significant differences were observed when comparing the highest CSES group with the low and middle CSES groups. No significant differences were seen between middle and low CSES groups. This may indicate a

threshold effect whereby at some level of CSES there are too few resources available or too many environmental and psychosocial threats which as a result lead to an increase in CA. CSES was measured by using the highest level of parental education and therefore the difference in CA by CSES may be a reflection of parental education, parenting skills, and other factors such as coping skills and social capital which are related to education level. Evidence based programs, such as the Triple P program or the nurse home-visiting program, both of which teach parents skills and provide resources, may help low SES families cope with stress and turmoil and, as a result, reduce SES disparities in CA.

In addition to CSES disparities, more dramatic SES disparities were seen when comparing ACEs across adult SES (ASES) and lifetime SES (LSES). Although tests of trend were not conducted, a visual inspection of the point estimates and CI reveals a monotonic relationship such that with each increase in SES level there was a significant decrease in ACEs reported. Whereas with CSES there might be a threshold effect based on environment and resources, here the difference in reported CA is likely related to the direct effects CA has on adult SES. That is, SES stratification of CA is not an explanation of the observed CA disparities (i.e., lower SES groups experience more adversity, more abuse, more drug and alcohol issues than higher SES groups) but rather SES stratification of CA is a result of CA experienced in childhood. Those that had low CSES and experienced greater CA likely did not increase their SES standing, and those in the middle and upper class as children who also experienced CA likely did not fare as well in maintaining or increasing their SES when compared to their counterparts who did not experience CA. To some extent the effect ACEs can have on social class mobility could be used to explain health disparities that persist across generations. ACEs are more likely to occur in low SES environments and also decrease opportunities to move up the

SES ladder, thus causing further stratification in adulthood and increasing the likelihood of CA for the next and future generations. Adversities happen over the lifespan and are not particular to childhood; however because childhood adversities occur during critical and sensitive periods of human development, socialization, and education they have the potential to cause greater damage and set less than optimal trajectories of health, education, employment and overall wellbeing.

This argument becomes more poignant when we look at how ACEs were distributed amongst those who are unemployed or "unable to work" compared to those who were employed, and also amongst those who reported disability compared to those who did not. Stark contrasts were observed such that CA was much more prevalent in those "unable to work" and those with disability. From this we might hypothesize that CA causes unemployment and disability in adulthood and thus lowers ASES and also increases the likelihood of CA for the next generation. If CA causes lower ASES then this might explain the pattern observed in the Lifetime SES data where there was greater SES stratification of CA. Caution must be given here as the cross-sectional nature of the data cannot be used to establish temporal ordering of CA occurrences and measures of SES. Also, because the ACHS did not include measures of child health, it is difficult to rule out the explanation that child health is confounding the relationship between CA and adult SES. From the most recent NIS we know that children with disability are more likely to experience child maltreatment.(91) We can also surmise that children with disabilities are more likely to grow up to be adults with disabilities and therefore more likely to be unemployed or "unable to work." Whether it is children's health or CA that is causing further ASES stratification is a moot point from a prevention standpoint in that CA and child health are intertwined such that improving one will improve the other, regardless of which is responsible for causing adult SES disparities in CA.

Comparisons of ACE by geographic characteristics, on the whole, revealed no statistically significant differences. In all three analyses the point estimates of CA were higher for those areas with higher poverty, higher unemployment, and higher percentage of Black residents. However, in all but one comparison the differences failed to be statistically significant. Rather than an attempt to explain geographic patterns of CA, the purposes of these analyses were more practical in nature. That is, these analyses focused on where and how we should intervene and prioritize local efforts to prevent, reduce, and mitigate the negative health effects of CA. One reason for the lack of statistical significance seen here is that the data of neighborhood characteristics was contemporaneously aligned with the time of data collection and not with the time the childhood adversity occurred. However, if CA does in fact constrain one's ability to climb the SES ladder (as suggested above) then we might expect that those reporting CA would be more likely as adults to remain in low SES environments. From these results it makes practical sense to direct prevention efforts to those areas with high poverty, high unemployment, and a greater percentage of minorities because point estimates were higher across all types of CA and higher for level of severity of CA. It is highly likely that these areas, as defined, overlap substantially. Additional analyses are required to determine which geographical indicators are most correlated with prevalence of adult reported CA.

Relationship between CA and adult health

Results indicated that large percentages (72-80%) of the adult health indicators measured were related to CA in both bivariate and multivariate models. Mental Health indicators were most strongly and independently related to ACE score with an increased odds of 5.14 for life dissatisfaction, 6.39 for reporting greater than 14 poor mental health days in the past 30 days,

and 9.97 for SMI when comparing those with an ACE score of 4 to those with an ACE score of zero. Even after controlling for age, gender, race, and lifetime SES, adult mental health indicators remained significant and were only slightly attenuated. Again these results are consistent with previous estimates. Kessler found a 2-fold increase in major depression when comparing those with and without CA and Felitti found a 12 fold increase in odds in attempted suicide when comparing those with four or more ACEs with those with zero reported ACEs.(20) Life dissatisfaction is highly correlated with suicide. The strong relationship between ACE and adult mental health were likely a reflection of the direct impact of ACEs have on psychosocial factors such as mood, social status and integration, stress, and coping. Experiencing great adversity in childhood when one is learning how to cope with stress, make friends and socialize, and learn about the world likely has serious ramifications to personality and behavior that spill over into adulthood. The direct and indirect effects of ACEs on adult mental health are further discussed below.

Physical health outcomes such as diabetes and cancer were much less associated with CA than mental health indicators in the ACHS. Fuller-Thomson and Brennenstuhl found a 49% increase in odds of cancer for those who experienced childhood physical abuse compared to those who did not.(14) In the ACHS odds of cancer increased with increasing ACE score, and those with an ACE score of 4 had a 38% increase in odds of cancer compared to those reporting no ACES. Differences may be due to the measures used (e.g., Fuller-Thomson and Brennenstuhl used a measure asking whether or not they were *ever* abused by someone close to them while growing up and before moving out, whereas in the ACHS physical abuse was measured based on frequency of abuse and coded as abuse only if there were multiple incidents) or difference could be explained

by cultural or political differences, as the Fuller-Thomson and Brennenstuhl study was conducted in Canada.

Health behaviors were also significantly correlated with CA such that with increasing CA the odd of smoking, alcohol binge drinking, multiple sexual partners, and cancer compliance increased in the direction of health adverse behaviors. Results support the view that stress may be a mechanism that links CA to adult health.(93) Smoking, binge drinking, and BMI (a proxy for diet) were all related to CA more so than positive health behaviors such as cancer screening compliance, physical activity, and consumption of fruits and vegetables. Because smoking, binge drinking, and poor dieting may be considered as negative behavioral responses to stress, it is likely that those who experience CA are using these behaviors to cope with stressful events and circumstances in their lives. Greenfield et al found that obesity in adulthood was associated to reported CA in childhood and that using food as a response to stress mediated that relationship.(23) Changes in the HPA axis as a result of CA may be responsible for a heightened response to stressors which in turn may produce a propensity to engage in stress reducing behaviors and increase the risk of disease. The results herein supporting smoking as a mediator of the CA adult health relationship also provide evidence for this viewpoint. However, to truly test stress as the mechanism linking CA to adult health through health behaviors, one would need longitudinal data following both those exposed and not exposed to CA over time with measures of stress (e.g., self-report, cortisol, catecholamine) behavior, and health outcomes.

Social support was also strongly associated with CA. Those with an ACE score of 4 had three times the odds of reporting low perceived social support compared to those with no reported ACEs. Low social support and insolation are themselves risk factors for CM. (36) Those who experienced ACEs are more likely as adults to have less social support than those

who have not experienced ACEs and this in turn may increase the risk for disease and perpetuates the likelihood that the next generation will also experience CM or other ACEs. Given that low social support is a mediator of the ACE adult health relationship and also a risk factor for parents to commit CM, social support is a factor that should be targeted, bolstered, and monitored in at-risk populations. By doing so, the intergenerational effects of and disparities in health associated with CA may be ameliorated.

Population Attributable Risk of ACEs

Few studies have calculated the population attributable risk fractions to estimate the proportion of morbidity in the population that is associated with ACEs.(84, 94) Afifi et al. using data from the National Comorbidity Survey Replication, found that the ACE related PARs for any psychiatric disorder (including mood, anxiety and substance abuse) were 36% and 27.3% of psychiatric disorders in the population for females and males, respectively.(95) These results are slightly lower, but congruent with ACHS data where 41.3% (95%CI: 35.3%, 44.5%) of serious mental illness (SMI) was attributable to ACEs. Kessler et al. recently conducted a study in 21 countries using data from the World Mental Health Surveys and found that 29.8% of twenty psychiatric disorders examined were attributable to ACEs.(96) Moreover, in high income countries the relationship between ACEs and mental health expressed as PARs decreased significantly as a function of CA age of exposure with those who were exposed at younger ages having much higher PARs (PAR = 57.1% for exposure at age 4-12) compared to those who were exposed to CA later in adolescence (PAR = 28.8 for exposure age at 13-19). From a life course perspective, the trajectory of one's mental health is a function of exposure to ACEs and age of exposure. In this regard, when considering the various timing models, it appears that a sensitive

period model could be used to explain Kessler et al. findings in that exposure to ACEs has a direct effect on adult mental health, and age of CA exposure greatly impacts the mental health trajectory. Results from this ACHS study and others that have looked at the PAR of psychiatric morbidity point to the great proportion of mental illness in populations that could be prevented by ACE prevention and mitigation.

In addition to mental health outcomes in the ACHS, PARs associated with physical health and behavioral outcomes were also calculated. Although PARs for diabetes and cancer failed to reach statistical significance, the upper confidence limits indicate that as much as 20% of cancer and diabetes in the population could be prevented if ACEs were eliminated. For venereal disease, CVD, and physical health related quality of life (PHQoL) all PARs were significant and accounted between 25% and 27% of morbidity in the population. Similar PAR results were observed in the 2009 Washington State BRFSS which found 25.4% of CVD was attributable to ACEs.(84) For sake of comparison in PARs, using data from the Framingham study, Kenchaiah et al. found that in men the PAR for heart failure due to obesity was 11% and for women and 14%. (97) In this regard, ACEs are and should be considered a significant risk factor for CVD. From a clinicians perspective the importance of assessing and measuring patients' ACE scores should be at least as important as measuring BMI when determining CVD risk. From a public health perspective, prevention and mitigation of ACEs could have a profound effect on reducing health care costs, improving quality of life, and reducing overall morbidity and mortality.

Mediation and moderation of ACE adult health relationship

The ACE- adult CVD relationship was fully mediated by Adult SES (ASES), Smoking, Social Support, and BMI indicating that the effects of ACEs on CVD are more indirect than direct and that there is plasticity to alter the cardiovascular health trajectories of those exposed to ACEs. For example increasing ASEs for those exposed to ACES can mitigate the effects ACEs have on CVD by 46% and by 68% by increasing social support. Smoking and BMI accounted for 31% and 12% of the total effect ACEs have on CVD, respectively. Several of these mediated effects were moderated by age so that with increasing age the indirect effects of ACEs on CVD decreased. Similarly, ACEs interacted with CSES such that only for those with middle and high CSES was the indirect effect of ACEs on CVD through ASES significant. This may be a result of floor effects whereby those with low CSES who have experienced ACEs have little room to move down the socioeconomic latter in adulthood and so no significant change in ASES is observed. Another possibility is that those with low CSES who experience ACEs rarely move up the SES latter and therefore again we see little change in ASES for this group. Early detection of ACEs and programs and policies aimed at increasing the social and economic status of those experiencing ACEs can significantly decrease the prevalence of CVD attributable to ACES, especially when intervention is applied early in the life course.

Although the relationship between ACEs and adult SMI was partially mediated by ASES, social support and smoking, the direct effect of ACEs on SMI remained significant. These results suggest that the relationship between ACEs is, by and large, independent of adult health behaviors, social support, and ASES meaning that primary prevention of ACEs is the best approach to reducing SMI in the adult population. Social support accounted for 16% of the ACE- adult SMI relationship and was the strongest of the mediator tested. Programs that

increase social support for adults and children who have experienced ACEs may significantly decrease the prevalence and occurrence of adult SMI in the population.

7.0 STRENGTHS AND WEAKNESSES

Strengths of this study are several. First, the ACHS is a relatively large population-based sample allowing statistical inferences to be made to the target population. Second, to my knowledge, this is the only county-level effort to collect measures of childhood adversity in a population-based survey which provides a unique opportunity to present the usefulness of this kind of surveillance and subsequent analyses at a local level. Third, having measures of CA coupled with the core and optional modules of the BRFSS as well as with partner added questions concerning childhood SES, social support, and neighborhood level variables, provided a wealth of information to examine the pathways involved in the CA adult health relationship. Fourth, the sampling strategy to oversample African Americans and those with low household incomes increase statistical power to investigate CA disparities and CA related disparities in adult health. Last, the calculation of PARs and the general focus on prevention make this research practical in the sense that policy and prevention strategies can be informed by the results herein.

Limitations of the proposed analytic plan involve design issues, coverage and nonresponse issues, and measurement issues. The ACHS is a cross-sectional descriptive survey and was not designed specifically to address the aims of this research agenda. Temporal ordering of CA exposures, health behaviors, and health outcomes cannot be firmly established leaving only inferences of association rather than causation. However, given that we know CSES and CA precede adult health behaviors, mediation analyses like the one conducted above have some credence. Coverage issues of the ACHS also affect the results and finding of this study. The ACHS did not include a cell-phone frame and only including landline telephones. Cellphone only households tend to consist of racial minorities and younger, lower SES

individuals and families. These groups are thus underrepresented in the ACHS sample. Moreover, the characteristics associated with cell-phone only household are likely related to CA and health, and so not including them in the sampling frame introduces bias. Although these groups were excluded from the sampling frame, the oversampling of African Americans and low income households coupled with post stratification weighing addressed this limitation. Finally, 2 of the measurement scales, namely the ACE scale and neighborhood quality scale, were modified and thus may threaten the reliability and validity of the measurements and may limit the comparability of the findings from this study to those of other studies that use the complete and unmodified versions. To address this limitation, reliability testing of the modified scales is proposed.

ACEs in this study were limited to child maltreatment and household dysfunction. Prevalence estimates are likely underreported due to the fact that acts of omission (e.g., medical neglect, physical and emotional neglect) were not included in the ACHS. Moreover, other adversities such as the loss of loved one, parental divorce, and bullying were not measured. Finkelhor et al. proposes adding peer rejection, peer victimization, community violence exposure, school performance, and socioeconomic status to the ACE measures to increase it predictive validity and association with adult mental health.(98)

8.0 CONCLUSIONS

Given the high prevalence of childhood adversity (CA), its strong association with short and long-term negative health outcomes, and its substantial economic costs, more resources are needed to secure healthy physical and social environments for children. This could be achieved, in part, through increased prevention activities and mitigation strategies aimed at reducing childhood adversities and childhood stress. Through primary prevention we can begin affecting the upstream determinants of adult mental and physical health that result in morbidity and premature mortality. Although primary prevention of CA should be our first objective and main thrust, given the current high prevalence of adults with a history of CA, secondary prevention efforts (e.g., The Triple P Program) can greatly help to mitigate the deleterious effects CA has on adult health and families and reduce costs by providing access and services to those at greatest risk.

Few studies have been conducted and published on the prevention cost-benefit ratio for programs designed to prevent CM or treat victims. Caldwell, studying the costs of CM in Michigan, estimates that the cost-benefit ratio to be 19 to 1, meaning that for every dollar spent on prevention of child maltreatment (e.g., Home Visitation programs), 19 dollars could be saved if the interventions were successful in preventing CM.(99) In a similar study conducted in Colorado, Gould and O'Brian estimated that if a home visitation program could reduce the costs of CM by only 6%, then the program costs would be offset.(100) Bruner, studying the effects that community centers would have if placed in high risk neighborhoods, found that with an investment of 18.5 million dollars to create or expand services in high risk neighborhoods, only a 5% reduction in preventable costs would be needed to offset the costs.(101) Although cost-

benefit analyses of this sort are difficult to conduct given the level of uncertainty in determining both the cost of maltreatment and the effectiveness of programs, it is clear that prevention efforts, even if only minimally successful, can save millions of dollars.

As Kemp's initial work "the battered child syndrome" resulted in major legislation to address child maltreatment and established mandates to monitor CM in the population, the initial work of Kessler, Felitti and all the work thus far studying ACEs and their impact on health across lifespan should prompt public health professionals and clinicians to monitor ACEs in both the general population as well as in vulnerable and at risk populations. The CDC BRFSS has taken steps to include ACEs in the annual survey; however to date only a few states have administered it and even fewer have administered it more than once. Expanding the ACE module to more states and more time periods will enable researchers, policy makers, and clinicians to better understand how ACEs are affecting the health of the local population they serve.

APPENDIX: SUPPLEMENTARY TABLES

Table S-1 Social Demographic Variables Coding

Social and demographic questions	Response Options	Proposed Recoding
1. What is your age?	1-100 year old 777 = Not Sure/Don't know 999 = Refused /Missing	Descriptive Coding: 1 = 18-29 2 = 30-39 3 = 40-49 4 = 50-59 5 = 60-69 6 = 70-79 7 = 80+ Analytic Coding: Continuous variable
2. What is your gender/sex?	1 = Male 2 = Female	1 = 0 2 = 1
3. What is your race?	1 = White 2 = Black/African American 3 = Asian 4 = Native Hawaiian or Other Pacific Islander 5 = American Indian or Alaska Native	Descriptive Coding: 1 = 0 2 = 1 3 = 2 (4,5) = 3 Analytic Coding: Dummy coding
4. (Employment status) Are you currently…?	1 =Employed for wages	Descriptive Coding:

	2 =Self-employed 3 =Out of work > 1 year 4 =Out of work < 1 year 5 =A Homemaker 6 =A Student 7 =Retired 9 =Refused	(1,2) = 0 (3,4) = 1 (5,6,7) = 2 <u>Analytic Coding:</u> Dummy coding
5. What is the highest grade or year of school you completed?	1 =Never attended school or only attended kindergarten 2 =Grades 1 through 8 (Elementary) 3 =Grades 9 through 11 (Some high school) 4 =Grade 12 or GED (High school graduate) 5 =College 1 year to 3 years (Some college or technical school) 6 =College 4 years or more (College graduate) 9 =Refused	<u>Descriptive Coding:</u> (1,2,3) = 0 4 = 1 5 = 2 6 = 3 <u>Analytic Coding:</u> Dummy coding
6. (Disability status) Are you limited in any way in any activities because of physical, mental, or emotional problems?	1 = Yes 2 = No 7 = Not Sure/Don't know 9 = Refused /Missing	1 = 1 2 = 0
7. (Disability status) Do you now have any health problem that requires you to use special equipment, such as a cane, a wheelchair, a special bed, or a special telephone?	1 = Yes 2 = No 7 = Not Sure/Don't know 9 = Refused /Missing	1 = 1 2 = 0
8. Is your annual household income from all sources…	1= <$10,000 2= $10,000 -$14,999	<u>Descriptive Coding:</u> 1= <$15,000-$19,999

	3= $15,000 -$19,999 4= $20,000 -$24,999 5= $25,000 -$34,999 6= $35,000 -$49,999 7= $50,000 -$74,999 8= $75,000+ 77= Not Sure/Don't know 99= Refused /Missing	2= $15,000-$24,999 3= $25,000-$49,999 5= $50,000-$74,999 6= $75,000+ Analytic Coding: Dummy coding
9. (Parental education (i.e., childhood SES)) What is the highest grade or year of school your father has completed?	1 =Never attended school or only attended kindergarten 2 =Grades 1 through 8 (Elementary) 3 =Grades 9 through 11 (Some high school) 4 =Grade 12 or GED (High school graduate) 5 =College 1 year to 3 years (Some college or technical school) 6 =College 4 years or more (College graduate) 9 =Refused	Descriptive Coding: (1,2,3) = 0 4 = 1 5 = 2 6 = 3 Analytic Coding: Dummy coding
10. (Parental education (i.e., childhood SES)) What is the highest grade or year of school your Mother has completed?	1 =Never attended school or only attended kindergarten 2 =Grades 1 through 8 (Elementary) 3 =Grades 9 through 11 (Some high school) 4 =Grade 12 or GED (High school graduate) 5 =College 1 year to 3 years (Some college or technical school) 6 =College 4 years or more (College graduate) 9 =Refused	Descriptive Coding: (1,2,3) = 0 4 = 1 5 = 2 6 = 3 Analytic Coding: Dummy coding

Table S-2 Main Independent Variable - Childhood Adversity Measure

ACHS Adverse Childhood Experiences questions	Response Options	Proposed Recoding
When you were growing up…		
1. was anyone living in your home depressed, mentally ill, or suicidal?	1 = Yes 2 = No 7 = Not Sure/Don't know 9 = Refused /Missing	1 = 1 2 = 0 (7,9) = missing
2. did you live with anyone who was a problem drinker, alcoholic or drug user?	1 = Yes 2 = No 7 = Not Sure/Don't know 9 = Refused /Missing	1 = 1 2 = 0 (7,9) = missing
3. how often did a parent or adult living in your home hit, beat, kick, or physically hurt you?	1 = Never 2 = Once/twice 3 = Sometimes 4 = Often 5 = Very often 7 = Not Sure/Don't know 9 = Refused /Missing	1 = 0 2 = 1 3 = 2 4 = 3 5 = 4 (7,9) = missing
4. how often did a parent or adult living in your home swear at you, insult you, or put you down?	1 = Never 2 = Once/twice 3 = Sometimes 4 = Often 5 = Very often 7 = Not Sure/Don't know 9 = Refused /Missing	1 = 0 2 = 1 3 = 2 4 = 3 5 = 4 (7,9) = missing

5. how often did a parent or adult living in your home push, grab, slap, or throw something at your mother?	1 = Never 2 = Once/twice 3 = Sometimes 4 = Often 5 = Very often 7 = Not Sure/Don't know 9 = Refused /Missing	1 = 0 2 = 1 3 = 2 4 = 3 5 = 4 (7,9) = missing
6. touch you sexually or try to make you touch them sexually?	1 = Never 2 = Once/twice 3 = Sometimes 4 = Often 5 = Very often 7 = Not Sure/Don't know 9 = Refused /Missing	1 = 0 2 = 1 3 = 2 4 = 3 5 = 4 (7,9) = missing
7. Cumulative ACE score		Sum of recoded questions 1-6

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