LIFE COURSE ORIGINS OF FRAILTY IN LATER LIFE

by

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For Rory—You are so loved.

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ABSTRACT

Frailty, generally characterized as a clinical state of increased vulnerability resulting from age-related decline in reserve and function across multiple physiologic systems, has been gaining attention in recent years due to its high correlates with a number of poor health outcomes including falls, hospitalization, and mortality. Although policy makers, health practitioners, and researchers have acknowledged that frailty is a major public health issue, few have investigated the life course predictors of this devastating and costly syndrome. The purpose of this dissertation is (1) to identify the early and later-life predictors of initial frailty and frailty growth over time among older US adults, (2) to examine if childhood exposures influence frailty directly and/or indirectly through adult risks and resources, and (3) to examine the role that social relationships play in frailty trajectories among older adults. Drawing from cumulative inequality theory, this dissertation uses longitudinal data from the Health and Retirement Study (HRS) to examine the effects of childhood exposures, adult risks/resources, and social relationships on frailty trajectories among adults 65 and older. The empirical investigation is presented in two main chapters.

The first study investigates the life course predictors of frailty prevalence and further examines how childhood exposures may be directly and indirectly associated with frailty through adult risks and resources. The second study builds on the findings of the first by examining the predictors of frailty growth over time and investigating how social relationships in later life may shape that growth. Findings reveal each childhood exposure domain influences frailty either directly or indirectly through adult factors and experiences. Specifically, analyses reveal that childhood chronic disease, impairments, and risky adolescent behaviors directly influence frailty in later life. Additionally, results reveal that poor childhood SES was one of the most consistent

predictors of adult frailty—but much of the effect was due to its influence on adult risks and resources. Few adult risk factors influence frailty trajectories over time. Among adult resources, socioeconomic status (particularly education) slows frailty growth over time. Finally, findings reveal that both social support and more social roles mediate the relationship between childhood exposures and frailty, and that the effect of more social roles continues over time.

This dissertation highlights a number of life course predictors of frailty and identifies areas for potential interventions—particularly those aimed at providing equal access to higher education and quality social relationships over the life course. Most importantly, this dissertation demonstrates that frailty prevention should not be a task delegated exclusively to older adults. Effective prevention of this often devastating and costly syndrome should begin early in life.

CHAPTER 1. INTRODUCTION

1.1. Statement of the Problem

1.1.1. A Public Health Crisis

The absolute and relative increase in the number of older adults has led to a rapid, notable shift in the distribution of the population in the United States and worldwide. This age group has grown faster in size than any other subpopulation in recent decades and is projected to do so for the foreseeable future; by 2070, those 65 and older are projected to represent over a quarter of the total US population. In many ways, an aging population signals a success story for humankind—a significant public health triumph over many diseases that had limited life expectancy for centuries and beyond. Yet longer lives do not necessarily mean healthier lives.

The results of a simple Google search for 'world's oldest marathon runners,' is evidence of the vast heterogeneity among older adults. Yet, the fact remains that, on average, about 40% of adults 65 and older report having a disability and over 80% suffer from at least one chronic condition that requires ongoing care and management (Buttorf et al., 2017). This rapid population transition, coupled with health systems that are increasingly tested to meet the needs of older adults, demands a comprehensive public health response. As such identifying and addressing the origins of the ailments and syndromes that most affect the health of older adults is imperative.

In this context, the idea of "frailty syndrome" or simply "frailty" is of notable research interest. Frailty is generally characterized as a clinical state of increased or extreme vulnerability to endogenous or exogenous stressors, resulting from age-related decline in reserve and function across multiple physiologic systems (Fried et al., 2001; Morley et al, 2013). This cumulative

decline drains homoeostatic reserves so that even minor stressors can trigger catastrophic changes in health status (Clegg et al., 2013). It has been described as a vicious cycle responsible for the onset of negative health-related outcomes (Fried et al., 2001)—a sort of senescence in hyperdrive— signaling a slippery slope between successful aging and disability (Clegg et al., 2013).

As noted by virtually all study introductions and/or abstracts on the topic, frailty is incredibly valuable as a predictor of a host of negative health-related events including falls (Fried et al., 2001; Kojima, 2015a; Lan et al., 2020), hospitalization (Chang et al., 2018), and mortality (Xue, 2011; Chang & Lin, 2015). For example, two review studies found that frail adults had 0.85 higher odds of a future fall (Kojima, 2015a) and two times higher risk of mortality (Chang & Lin, 2015) than their non-frail counterparts. Not surprisingly, the individual and system-wide implications of this condition are staggering. A recent study using Medicare data found that frailty *increases* the average estimated costs of medical care over a 9-month period by as much as $17,220 per patient ($10,690 when fully adjusting for chronic disease and medication use; Simpson et al., 2018). Extrapolated to one year, calculated using the lowest estimates of frailty prevalence among US adults 65 and older, the *minimum* total costs of frailty to Medicare alone is over 5.5 billion dollars annually.[1]

In addition to the economic strain associated with frailty, there is substantial concern being raised among researchers and physicians alike that the current system of care is ill-equipped to meet the needs of these older adults. At its 2016 assembly, the World Health Organization proposed the Framework for integrated people-centered health services (IPCHS), a

[1] Calculation: ($17,220*12)/9= $22,960 yearly cost per capita; 55,048,806 (US population 65+)*.04(frailty prevalence)= 2,201,952 (number of US frail adults); 2,201,952*22,960= $50,556,823,430.40 (annual cost)

call for a shift in the way health services are funded, managed, and delivered. They argued that patients, particularly those with chronic or complex conditions, experienced "fragmented, poorly integrated care from multiple providers, often with suboptimal outcomes and risk of harm due to failures of communication, inadequate sharing of clinical information, poor reconciliation of medicines, duplication of investigations and avoidable hospital admissions or readmissions (World Health Organization, 2018; p. 68)." Accordingly, one of the five proposed strategies to increase universal quality of care was to improve continuity and coordination of care. The current US healthcare system is indeed disjointed, designed mostly to address organ- and disease-specific problems, often independently from one another. For the most past, it does not have the processes in place to manage the complex and interconnected needs of frail older adults and to provide seamless, long-term care (Kojima, 2019).

1.1.2. The State of the Literature

Because of its predictive validity for poor health outcomes and related implications for the future of healthcare systems, frailty has been gaining traction with the academic community. In the past two decades, frailty has experienced exponential growth as the subject of empirical research. According to a PubMed search of *biomedical* literature alone, the term 'frailty' was used in only 74 articles in 2000 but is on pace for over 3,600 articles by the end of 2020 (see Figure 1.1; Tables and figures appear at the end of each chapter).

Although the concept is almost universally accepted as a topic ripe for investigation, its conceptual and operational definition remains controversial. In an attempt to review the vast conceptualization of frailty, Markle-Reid and Browne (2003) found that frailty is considered in the literature when there are one or more indicators of functional impairments and dependence on others that threaten independence, poor physical or mental health, disability, weakness, long-

term care needs, and/or advanced age. Similar reviews (Dent et al., 2016; Dolenc & Rotar-Pavlič, 2019; Faller et al., 2019) identify numerous conceptual models of frailty, each with vastly different underlying assumptions, dimensions, and causes. Furthermore, even when in agreement about conceptualization, researchers disagree on how to operationalize the concept. What results is a seemingly endless list of frailty measures and assessment tools (see Table 1.1 for examples).

The two most popular models are the *phenotypic* or *biologic syndrome model*, developed by Fried and colleagues (2001) and the *cumulative deficit model* developed by Rookwood and colleagues (2005). Fried and colleagues postulated frailty to be a "biologic syndrome of decreased reserve and resistance to stressors, resulting from cumulative declines across multiple physiologic systems" (Fried et al., 2001, p. M146). They defined a physical frailty phenotype in terms of five indicators (unintentional weight loss, exhaustion, weakness, slow walking speed, and low energy expenditure), in which the lowest quintile values were used to define the absence/presence of the indicators. In the model's original form proposed by Fried et al. (2001), presence of any three or more of the conditions indicate frailty, one or more indicates pre-frailty, and zero conditions indicate no frailty or "robustness." However, in the nearly twenty years since its conception, researchers have also used alternative forms of the measure, electing to dichotomize the measure (van der Linden, Cheval, et al., 2020; van der Linden, Sieber, et al., 2020) or analyze as a continuous count of indicators (Kennedy et al., 2019; Mijnarends et al, 2015). Alternatively, Rockwood and colleagues developed a frailty index (FI) as a measure of *deficit accumulation* measuring number of diseases, conditions, and symptoms. The higher the score in this index, the frailer the individual. Originally composed of 70 variables that range

from medical conditions and symptoms to functional decline, subsequent studies reveal that 30 variables can be used without loss of predictive validity.

Given the heterogeneity its measurement, obtaining an accurate estimate of its prevalence is challenging. In their systematic review and meta-analysis based on 21 empirical studies, Collard and colleagues (2012) found that the reported prevalence of frailty ranges from 4% to nearly 60%; however, categorizing by frailty definition drastically reduced this variability. They found that studies using the phenotypic model reported consistently lower prevalence than other models, noting that the diversity in criteria for the broader definition of frailty contributed significantly to the variability in prevalence. Guided by these findings, I elect to use the more popular phenotypic model of frailty for this dissertation. It is a narrower, yet more consistent definition which will aid in comparability of my findings with other studies. Additionally, previous studies have found a "minimal impact" on results when using the frailty phenotype score as a continuous variable as compared to the original, 3 ordered categories (Kennedy et al., 2019, p. 220). As such, this research analyzes frailty as a continuous score (0-5) to preserve variability across the indicators and heterogeneity within frailty "categories" rather than applying arbitrarily defined cutoffs.

1.1.3. The Origins of Frailty

Irrespective of how frailty is measured, its value in predicting health outcomes among older adults is consistent. As such, addressing the etiology of and subsequently identifying subpopulations at risk for frailty is a goal for many researchers and clinicians resolute to improve healthy life expectancies of older adults. Results from recent studies show that there are numerous risk factors for frailty across the life course including health, lifestyle, socioeconomic,

social and demographic factors—each of which can interact with one another to elevate frailty severity.

Frailty is more common with increasing age. As such, early investigation into the causes of frailty began with proximal risk factors. Studies have identified a number of factors in mid-later life associated with the development of frailty, including chronic disease and morbidity (Bandeen-Roche et al., 2015; Howrey et al., 2018), smoking (Hubbard et al., 2009; Kojima, Iliffe, & Walters, 2015), and sedentariness (Bandeen-Roche et al., 2015). Additionally, and as with many later life conditions, it is more prevalent among women and black and Hispanic adults (Cesari et al., 2016).

Though it is important to identify the proximal risk factors for any condition in later life, it has become increasingly evident among gerontologists that we must shift towards a life course approach to research in order to address health problems decades before they arise. Guided by the life course perspective and accumulation theories, early origins scholars seek to highlight the childhood origins of later life health. Though limited in quantity and scope, research on the early origins of frailty have identified the important role that early socioeconomic, health, and family conditions can play in the development of frailty (Alvarado et al., 2008; Gale et al., 2016; van der Linden, Cheval, et al., 2020; van der Linden, Sieber, et al., 2020). Yet, there is considerable empirical work to be done to understand the life course predictors of frailty so that we may begin to address the etiologies of this condition before they arise. As the adage goes, "an ounce of prevention is worth a pound of cure."

1.2. Specific Aims and Innovation

Using the Health and Retirement Study (HRS) and informed by cumulative inequality theory, this dissertation investigates the early origins of frailty by systematically examining the

early and later life predictors of frailty, along with mediation mechanisms. This research has three main aims:

Aim 1: To identify the early and later-life predictors of initial frailty and frailty growth over time among older US adults.

A vast literature has demonstrated the noxious effects of childhood exposures on later life health; yet, work remains to be done to fully understand its effects on frailty. Among published studies on the effects of childhood exposures on frailty, exposures most often explored are those related to maltreatment, poor socioeconomic status, and poor health. Furthermore, these studies often fail to consider the *cumulative* effects of experiencing misfortunes in more than one domain—i.e. SES and health. This study builds on the literature by considering the effects of six domains of childhood exposures on frailty among older adults in the United States.

Aim 2: To examine if childhood exposures influence frailty directly and/or indirectly through adult risks and resources.

Childhood exposures have been found to directly influence many mid- and later- life risk factors associated with frailty (i.e. smoking, physical activity, morbidity, etc.) and the resources most beneficial in avoiding becoming frail (i.e., education, wealth, etc.). As such, failing to account for those pathways, is likely to lead to biased estimates of the early effects on frailty. Although, I expect that childhood exposures will directly influence frailty in later life, this dissertation also will assess how these exposures may affect frailty through additional risks or mitigating resources in adulthood (see Figure 1.2).

Aim 3: To examine the role that social relationships play in frailty trajectories among older adults.

Extensive research has demonstrated that social relationships, both in terms of quality and number of social roles, is associated with physical and mental health outcomes among older adults. Specifically, studies have found that socially embedded older adults with positive social relationships are less frail than their isolated and socially strained counterparts (Lyu & Agrigoroaei, 2017). Yet, research also finds that childhood exposures are associated with the development and maintenance of social relationships throughout the life course (Bradley & Corwyn, 2002; Poulton & Capsi, 2005; Cohen et al., 2010). This dissertation builds upon these studies by considering the mediating effects of social support, social strain, and number of social roles on the relationship between childhood exposures and frailty trajectories (see Figure 1.3).

1.3. Data and Methods

This dissertation uses longitudinal data from the Health and Retirement Study (HRS) to assess the life course predictors of frailty in later life. The HRS is a longitudinal panel study collected by the University of Michigan that surveys a representative sample of over 30,000 adults aged 50 and older in the United States and is supported by the National Institute on Aging (NIA U01AG009740) and the Social Security Administration (Health and Retirement Study, 2020). Beginning nearly three decades ago in 1992, core data is collected every two years using a multistage, area stratified probability sample of Americans and oversamples Black and Hispanic Americans, as well as Florida residents. Initial participants were born from 1931-1941, who were then aged 51 to 61, and a second study was added a year later to capture those born prior to 1924—70 years and older at the time. In 1998, "children of the great depression" and "war babies" were recruited to bridge age gaps. The HRS now replenishes the sample every six years (2004, 2010, 2016) to maintain a representative sample of adults 50 and older in the US.

The HRS is the largest, nationally representative panel study of older adults in the

United States and boasts excellent response rates, with most waves exceeding 85%. The study collects comprehensive data on later life health outcomes and psychosocial resources throughout the life course. Additionally, the HRS collects quality data on childhood exposures, including information on socioeconomic status, chronic and infectious diseases, impairments, and risky behaviors during adolescence. It should be noted, however, that the survey also collects data on some risky parental behaviors (i.e. smoking, substance abuse, physical abuse), but data on more traumatic experiences (i.e. homelessness, sexual abuse) and household dysfunction (i.e. parent divorce) are lacking. Though this is a limitation of the data, I argue that the relationship between these exposures and later life health outcomes is more established, leaving other misfortunes more open to examination.

This dissertation draws primarily from 6 waves of data, spanning 10 years from 2006-2016. Due to survey design, I used two half samples to create a full sample with baseline frailty and covariates measured at 2006 or 2008 (See Figure 1.4). However, because several childhood measures were added to the survey in 2004 (with some SES measures dating back to survey initiation), I utilized data from those waves to fill in missing information about exposures during childhood. In addition to core data, this dissertation uses data from enhanced face-to-face interviews (EFTF) which included physical measures tests. These physical measures included handgrip strength and timed walking tests, which were used to create two of the five frailty indicators. By HRS survey design, those who were selected for the EFTF sample but lived in a nursing home, completed a telephone interview, or who were interviewed by proxy, were not asked to complete the physical measures (Crimmins et al., 2008). Additionally, adults under 65 years of age were not asked to complete walking speed tests; thus, this dissertation focuses on adults aged 65 and older. Finally, to reduce bias in retrospectively collected childhood exposures

data, a number of sample exclusions were employed; I describe these exclusions in depth in the methods sections of chapters 2 and 3.

All analyses for this dissertation were conducted using Stata SE, version 15.1. The first study uses a series of linear regression models to estimate frailty prevalence, followed by structural equation modeling (SEM) to assess the mediating effects of adult risks and resources. Similarly, the second study uses a series of growth curve models (GCM) to identify the predictors of frailty over time, followed by a mediation analysis to assess the potential mediating effects of social relationships on the association between childhood exposures and frailty trajectories. Both studies use post-estimation commands to calculate the total, direct, and indirect effects for each proposed path (Sobel 1987), along with standard errors using the delta method.

1.4. Description of Chapters

The remainder of the dissertation is divided into two empirical chapters and a concluding chapter. Chapter 2 presents the first empirical study and examines the life course predictors of frailty prevalence. This study also examines how childhood exposures may be directly and indirectly associated with frailty through adult risks and resources. Chapter 3 presents the second empirical study which builds on the findings of the first. I examine the predictors of frailty growth over time and investigate how social relationships in later life may shape that growth. Chapter 4 is the concluding chapter, in which I summarize the results of the previous two chapters, discuss the broader implications of those findings, and conclude with future research directions.

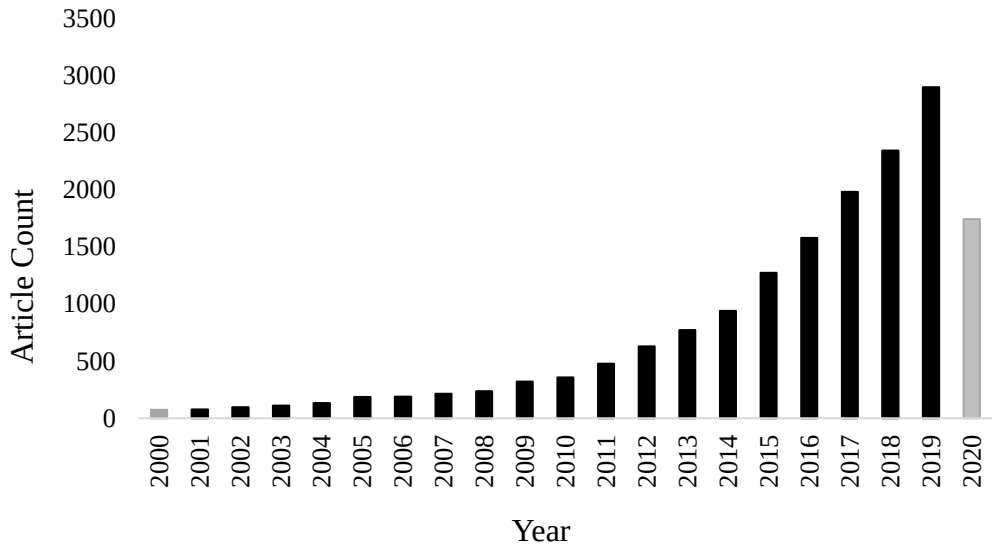


Figure 1.1. PubMed Search for 'Frailty' (2000-2020)

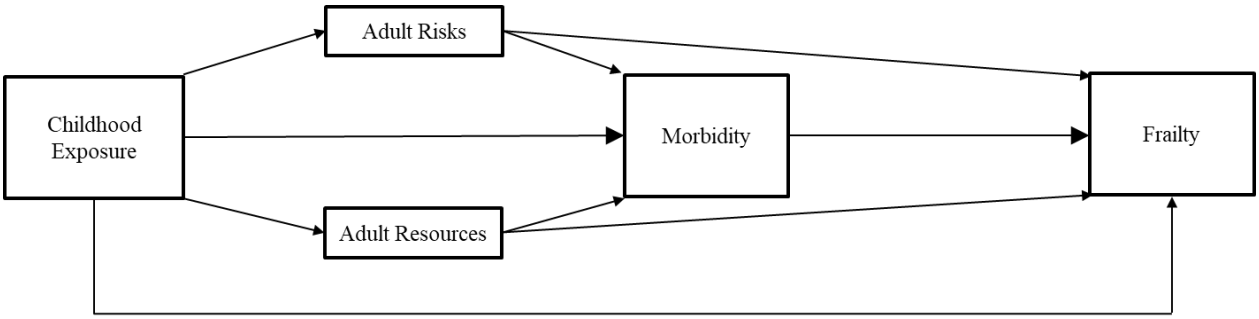


Figure 1.2. Conceptual Model of Proposed Relationships Among Childhood Exposures, Adult Risks, Adult Resources, and Frailty

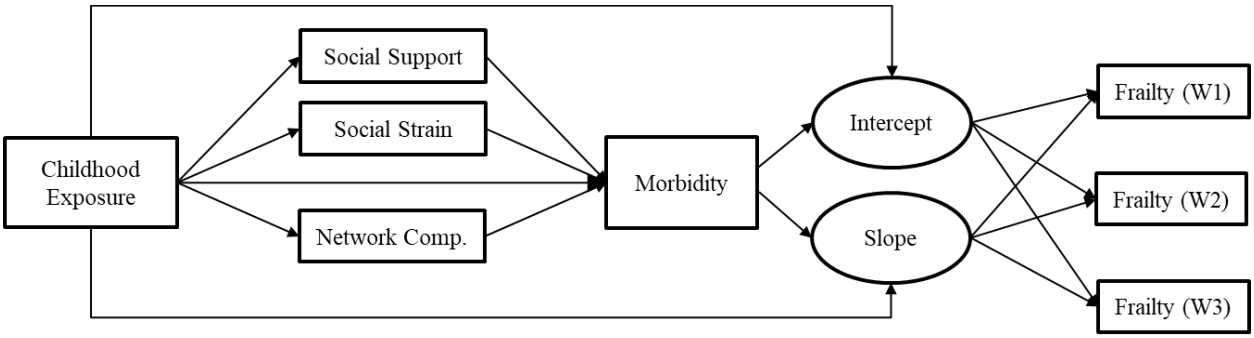


Figure 1.3. Conceptual Model of Proposed Relationships Among Childhood Exposures, Social Relationships and Frailty Growth over Time Note: Rectangles represent observed variables and ovals represent latent variables. Comp.= Composition.

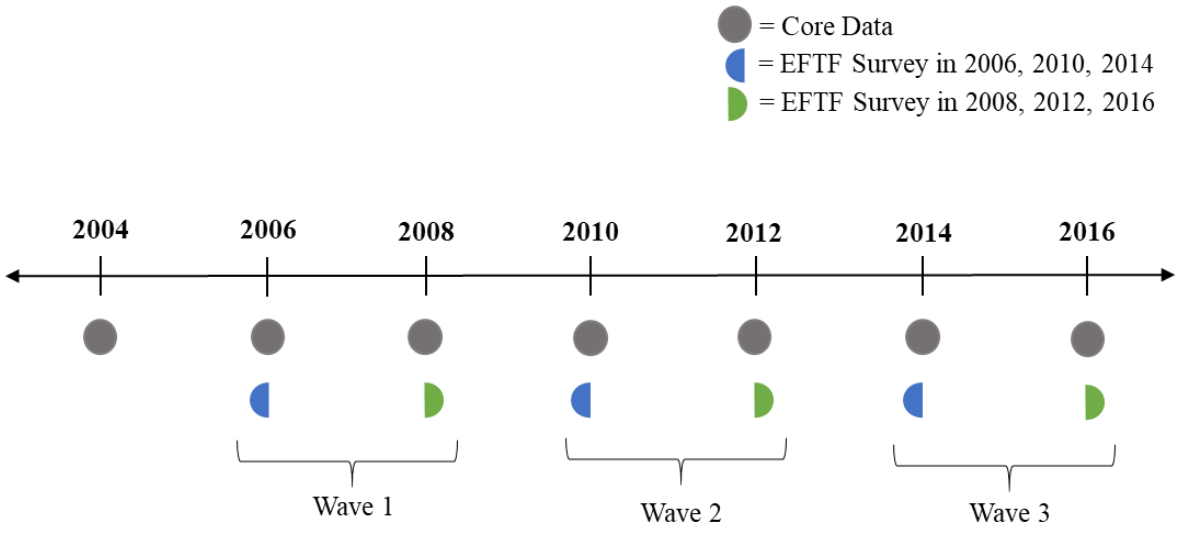


Figure 1.4. Data Source and Structure for the Analytical Sample. Note: Variables originating from core data, including demographics, adult risks, and adult resources, are collected every two years on the full sample. Social environment and frailty measures are collected from the leave-behind and enhanced face-to-face survey, respectively, and are collected every four years on alternating half samples.

Table 1.1. Selected Operationalizations of Frailty

Index	# of Items	Components
Cardiovascular Health Study Index (Fried Frailty Phenotype)	5	Weight loss, low physical activity, exhaustion, slowness, weakness
Frailty Index of Accumulated Deficits (FI-CD)	30+	Accumulated health deficits (0-1)
Strawbridge Frailty Questionnaire (SFQ)	4	Physical, nutritive, cognitive, and sensory functional domains
Frailty Index from Comprehensive Geriatric Assessment (FI-CGA)	30+	10 domains, 52 items- ADL, IADL, Comorbidity, Mood & Cognition
Kihon Checklist (KCL)	25	Subset of FI-CGA
Study of Osteoporotic Fracture (SOF) Index	3	Weight loss, exhaustion, unable to rise from chair
Edmonton Frailty Scale (EFS)	9	Cognition, health (2), hospitalization, social support, nutrition, mood, function, continence
Fatigue, Resistance, Ambulation, Illness and Loss of Weight (FRAIL) Index	5	Fatigue, resistance, ambulation, illness, weight loss
Clinical Frailty Scale (CFS)	1	Visual/written chart for frailty with 9 graded pictures (1=very fit; 9=terminally ill)
Multidimensional Prognostic Index (MPI)	8	Co-morbidity, nutrition, cognition, polypharmacy, ADL, IADL, pressure sore risk, living status
Tilburg Frailty Index (TFI)	15	Self-reported in 3 domains: physical, psychological and social
PRISMA-7	7	Self-reported: age (>85 years), gender (male), social support, ADLs
Groningen Frailty Indicator (GFI)	15	4 self-reported domains: physical, cognitive, social, psychological
Sherbrooke Postal Questionnaire (SPQ)	6	Self-reports of living alone, hearing polypharmacy, mobility, eyesight, memory
Gérontopôle Frailty Screening Tool (GFST)	6	(1) self-reports of living alone, weight loss, fatigue, mobility, memory, gait (2) clinical judgement

Note: Adapted from Dent et al., 2016

CHAPTER 2. LIFE COURSE PATHWAYS: CHILDHOOD EXPOSURES AND FRAILTY PREVALENCE

2.1. Introduction

Frailty, generally characterized as a clinical state of increased vulnerability resulting from age-related decline in reserve and function across multiple physiologic systems, has been gaining attention in recent years due to its high correlates with a number of poor health outcomes including falls, hospitalization, and mortality (Aguilar-Navarro et al., 2012; Fried et al., 2001; Lan et al., 2020; Lee et al., 2014). As the population ages, so has the prevalence of frailty—a recently published assessment of frailty among US non-institutionalized adults estimates that 15.3% of those aged 65 years and older are considered frail and 45.5% are in an intermediate, possibly "prefrail" clinic stage (Bandeen-Roche et al., 2015). Among those in nursing homes this figure jumps to 37.9-66.5% (Kojima, 2015), further illustrating its use as a prognostic tool in predicting overall health decline.

Many studies have identified a myriad of adult risk factors for developing frailty including, but not limited to sedentariness, nutrition, smoking, and alcohol use (Bandeen-Roche et al., 2015). Like other later-life health outcomes, research on the etiology of frailty has begun to move from proximal risk factors to those more distal in time, yet there is a need for more research on the childhood predictors of frailty in later life. The scientific literature on childhood exposures and frailty is limited in both quantity and scope, consisting of four studies examining the effects of only physical health and socioeconomic status.

To examine the early origins of frailty in older adults, I draw from cumulative inequality theory, illustrating the process by which childhood exposures can shape health trajectories through the accumulation of risk and disadvantage. To address the lacunae of knowledge

surrounding the process of childhood exposures and frailty in later life, I use data from the Health and Retirement study, a nationally representative panel study to address the following question: Do childhood exposures increase the likelihood of frailty in later life, both directly and indirectly through adult lifestyle factors?

2.2. Background

2.2.1. Frailty Across the Life Course

Chronic diseases have long been implicated in the development of frailty. Researchers report that older adults with a range of chronic diseases including diabetes, heart disease, lung disease, osteoporosis, and stroke are up to twice as likely to be classified as frail as compared to robust (Bandeen-Roche et al., 2015; Howrey et al., 2018; Walston et al., 2002). Similarly, those adults with multimorbidity, that is having two or more chronic diseases, are more likely than those with none or just one chronic condition to become frail (Klein et al., 2005). Mental health conditions such as depression also have been implicated in the development of frailty among older adults (Feng et al., 2017). However, due in part to the varying measurements of frailty and to the high co-occurrence of multimorbidity and frailty, research findings are inconclusive as to the causal direction of the two conditions. It is likely that the relationship is at least partially bidirectional (Vetrano et al. 2019).

Adult lifestyle factors have also been implicated in the frailty process, typically through the development and/or avoidance of health conditions. For example, those who smoke (Hubbard et al., 2009; Kojima et al., 2015), do not exercise regularly (Song et al., 2015), and those who have been exposed to poor nutrition at various points in the life course (Kuh et al., 2006; León-Muñoz et al. 2015) are also more likely to develop mobility limitations and become

frail in later life (Strawbridge et al., 1998). However, evidence is mixed on the effects of other "repeat offenders" of poor adult health outcomes, notably alcohol consumption and raised BMI. For example, while studies have found alcoholism to be an antecedent of frailty in later life, Shah, Paulson, and Nguyen (2015) found moderate, weekly alcohol consumption to be associated with reduced prevalent and incident frailty. Similarly, though high BMI has been linked to a host of related health problems in later life, some studies suggest there may be an "obesity paradox" associated with frailty wherein being overweight may be health protective, particularly for those with one or more chronic health conditions (Pamoukdjian et al., 2019).

Additionally, having (or not having) access to a number of resources throughout the life course have been implicated in the development of frailty, both directly and indirectly through some of the factors indicated above (Szanton et al., 2010). Socioeconomic resources, for example, are some of the most consistently researched topics. At the individual level, living in poverty or simply having relatively lower income or education (Hoogendijk et al., 2014; Szanton et al., 2010), and not holding a white-collar occupation (Woo et al., 2005) raise the risk of frailty. Failure to account for social stratification, therefore, may lead to an overreliance on individual health behaviors as the key causes of frailty.

2.2.2. Literature Gaps

A vast literature has demonstrated the deleterious effects of childhood exposures on later life health; yet, work remains to be done to fully understand its effects on frailty. In such studies, childhood exposures most often explored are those related to trauma, poor socioeconomic status, and poor health. Furthermore, most of these studies fail to consider the cumulative effects of experiencing misfortunes in more than one domain (i.e. SES *and* health). This phenomenon extends to the four studies identified which directly assess the effects of childhood exposures on

frailty (Alvarado et al., 2008; Gale et al., 2016; van der Linden, Cheval, et al., 2020; van der Linden, Sieber, et al., 2020)--each stressing the importance of early socioeconomic disadvantage in Latin America or Europe. Although Alvarado and colleagues consider both childhood health and SES, health is crudely measured by self-reports of excellent, good, or poor health. More recently, van der Linden, Sieber, and colleagues (2020) examined the effects of childhood trauma, health experiences, and SES throughout Europe, finding that each was associated with trajectories of frailty. The study is the most comprehensive to date; yet, it did not consider the effects of adolescence and did not distinguish between chronic health conditions, infectious diseases, and impairments. This present study builds on the literature by using data from the United States to consider a wider array of childhood exposures and the combined effects of those misfortunes.

In addition to considering additional childhood exposures, this study builds on the literature by not only considering the direct effects of early life, but also those more indirect. Because childhood exposures have been found to directly influence the aforementioned mid- and later- life risk factors linked to frailty and the resources most beneficial to avoid becoming frail, failing to account for those pathways could severely underestimate the true impact of childhood exposures on frailty. Although, I anticipate that childhood exposures will directly influence frailty in later life, this study also will assess how these exposures may affect frailty through additional risks or mitigating resources in adulthood.

2.3. Theory

Cumulative inequality is a middle-range theory that draws upon the ideas of stress proliferation and cumulative advantage/disadvantage theory to highlight the ways in which individuals stress and experiences throughout the life course occur within social systems and

structure to shape health trajectories, while also emphasizing the importance of personal agency and resource mobilization (Ferraro & Shippee, 2009).

The first axiom of cumulative inequality theory states that social systems generate inequality through developmental and demographic processes over the life course. For delineating the precursors of frailty, this means that assessing the early-life predictors is important for understanding the basis for an accumulation of risk throughout the life course. The second axiom focuses on the ways in which disadvantage and advantage shape exposures to risks and resources. CI maintains that inequality, shaped by social systems, may diffuse across life domains. In this way, a child who experiences poor socioeconomic conditions may be less likely to receive healthcare, which may make underlying health conditions worse—placing stress upon parents, which in turn may affect the mental health of their children. Conversely, as SES increases, so does the likelihood of family characteristics such as low conflict, positive relationships, and consistent parenting (Cohen et al., 2010). For this reason, it is important to consider a variety of childhood exposure domains, rather than a simple additive measure of disadvantage or adversity. As previously stated, the research on childhood exposures and frailty is limited to trauma, SES, and broadly defined health domains. However, prior studies of related health outcomes suggest that childhood impairments, chronic diseases, and infectious diseases influence the health of older adults differently with evidence suggesting that the latter may even be health protective (Smith et al., 2019; Williams et al., 2019). From this perspective, it is imperative to differentiate among these exposures and to consider other domains such as risky adolescent behavior, which often coincides with other adversities and is highly predictive of adult health behaviors (Herrenkohl, 2010).

Perhaps most pertinent to this research, the third axiom states the life course trajectories are shaped by the accumulation of risk, rewards, and agency. Experiencing a rough childhood can spawn poor health problems for older adults, but not everyone who experiences childhood misfortune, even major trauma, suffers from poor health in later life. Low socioeconomic status, risky families and the like can have lasting impacts on adult health by influencing continued exposure to stressors and behaviors unconducive to adult health. Yet, resilience through human agency and resource activation can help to protect against some of these health risks (Ferraro & Shippee, 2009). For example, smoking cessation has been associated with substantial reduction in risk of all-cause mortality among those with coronary heart disease (Critchley & Capewell, 2003).

2.4. Research Questions

Based on previous research findings and insights gleaned from cumulative inequality theory, the present study addresses the following research questions:

1. Do childhood exposures directly increase the likelihood of frailty in later life?

Research reveals that poor SES and health in childhood are related to physical frailty in later life (Alvarado et al., 2008; Gale et al., 2016). Similar research shows that a wider array of childhood exposures are associated with other frailty-related health outcomes such as handgrip strength (Smith et al., 2019) and being disease free (Schafer & Ferraro, 2012; Williams et al., 2019). As such, this study will assess the direct relationship between six domains of childhood exposures and frailty in later life.

2. Through which adult risks and resources do childhood exposures influence frailty in later life?

Older adults who experienced negative childhood exposures are more likely to engage in risky health behaviors and less likely to have access to resources as adults. These adult factors are, in turn, implicated in the development of multimorbidty and physical frailty. To examine the relationship between childhood exposures and frailty in later life, this study investigates the mediating effects of adult risks and resources, as illustrated in Figure 1.2.

2.5. Methods

2.5.1. Sample

Data for this project come from the Health and Retirement Study (HRS)—a multistage, probability study of adults aged 51+ years, with an oversampling of Black and Hispanic adults and Florida residents. Waves 7 to 9 (2004 to 2008) are used for this research, because a detailed battery of childhood misfortune indicators were first measured on the full sample in 2004.

For this study, eligible respondents include those aged 65 and older at baseline[2] who consented to physical measure tests during an enhanced face-to-face, in-home survey in 2006 or 2008. Respondents who were randomly assigned to 2006/2008 enhanced surveys were combined to create a full sample (N= 9,209). By HRS survey design, those who were selected for the enhanced face-to-face sample but were interviewed by proxy, lived in a nursing home, or who completed a telephone interview, were not asked to complete the physical measures (Crimmins et al., 2008). Those whose used proxy interviews for childhood experiences (N= 8,363) and whose cognition scores indicate the presence of dementia (total cognition score <6, N= 8,146) were excluded from the sample to preserve the validity of retrospective childhood

[2] Those under 65 years of age were not included in the study, because walking speed was only assessed for those individuals 65 and older by the HRS.

data. Finally, those with who were missing for baseline frailty also were excluded (N=6,805).

2.5.2. Measures

2.5.2.1. Frailty

Frailty is measured using Fried and colleagues' phenotypic model based on five components: unintentional weight loss, weakness, slowness, exhaustion, and low energy expenditure. Although unintentional weight loss cannot be directly assessed using the HRS, I follow the work of others (Cigolle et al. 2009) by defining *weight loss* as a calculated or self-reported weight loss of 10% or more since the previous wave (2 years) or a BMI of less than 18.5 kg/m^2. *Weakness* is identified by hand-grip strength using information from the physical health battery. Weakness is defined as having an assessed grip strength in the lowest 20% of the sample distribution, adjusting for sex and BMI. *Slowness* is identified by assessed walking speed over an 8-foot difference, also collected during the physical battery. Slowness is defined as having a walking speed in the lowest 20% of the sample distribution, adjusting for gender and height. Following the work of others (Cigolle et al., 2009), those participants who refused or did not complete either the handgrip or walking tests due to safety concerns or physical limitation were also classified as "weak" or "slow." *Exhaustion* is assessed by whether respondents responded "yes" to either of 2 CES-D items: "felt activities were efforts" and "could not get going." Finally, *low energy expenditure* is assessed using a gender stratified, weighted activity scale based on respondents' self-reports of frequency and intensity of physical activity. Those participants with activity scores in the lowest 20% of the sample distributions are classified as displaying low energy expenditure. These five indicators are summed to create a frailty scale from 0-5, where 0 indicates no frailty, or "robust." I present Table 2.1 to summarize the major

variables used for the analysis and their coding.

2.5.2.2. Childhood Exposures

Childhood exposures before the age of 18 are categorized into 6 domains based upon previous analysis: socioeconomic status (SES), risky parental behaviors, infectious diseases, chronic diseases, impairments, and risky adolescent behaviors, using 29 retrospective questions collected between 2004 and 2008 (See Figure 2.1). *Socioeconomic status (SES)* misfortune consists of 4 indicators: reporting poor or fair finances in relation to others, moving due to finances, having a father with an unskilled labor occupation, and father (or mother if not available) having less than an eighth-grade education. *Risky parental behavior* is assessed by having a parent who smoked, abused substances, and/or were physically abusive. *Infectious disease* includes measles, mumps, and/or chicken pox. *Chronic disease* includes asthma, diabetes, respiratory problems, seizures, migraines, stomach problems, allergies, heart disease, high blood pressure, and self-rated childhood health (poor or fair=1, good, very good, excellent=0). Presence of a childhood *impairment* includes head injury, disability for 6+ months, learning problems, visual impairment, and/or speech impairment. *Risky adolescent behavior* is assessed by having trouble with police, substance abuse, depressive symptoms, and/or other psychological issues.

Each indicator is coded dichotomously (1 if the condition is reported, 0 if not) and summed to create a count of misfortune within each domain. For most of the analysis, 0 indicates no experiences in that domain, and 1 indicates one or more experiences.

2.5.2.3. Covariates

Models adjust for demographic characteristics reported at baseline including age (in

years), gender (female=1), and race/ethnicity (non-Hispanic white=0, non-Hispanic black, and Hispanic). “Other” races are excluded from analysis as the number of cases is insufficient for meaningful comparison.

Adult resources include measures of socioeconomic factors and social context. Education is measured in years and top coded at 17+. Wealth is measured in tens of thousands of dollars and cube rooted to correct for skewness. A dichotomous indicator of health insurance status is created where 1= private insurance (private only and/or Medigap plans) and 0=otherwise (no insurance, Medicaid and/or Medicare only).

Adult risks include health and lifestyle factors. Morbidity, or number of chronic diseases, is assessed by asking respondents if a doctor had ever told them that they have arthritis, diabetes, lung disease, stroke, heart problems, hypertension, and cancer (excluding skin). A dichotomous indicator of smoking was created where 1=current smoker and 0=otherwise. Heavy drinking is coded dichotomously as having an average of 5+ drinks per day for men and 4+ drinks per day for women (Dawson, 2011). BMI is based on self-reports and ranges from 9.6-66.1 kg/m^2.

2.5.3. Analysis

The analysis was conducted in two parts using Stata/SE 15.1. To examine the life course predictors of frailty prevalence, a series of linear regression models were estimated. First, models were estimated without any adult risks or resources to establish a baseline relationship between the childhood exposure domains and later life frailty. Next, adult morbidity was added because it is so highly associated with frailty. Finally, adult risks and resources were simultaneously added into the model.

To further examine the potential mediating effect of adult risks and resources on the relationship between childhood exposures and frailty, mediation analysis was conducted using

Structural Equation Modeling (SEM) and the post-estimation command "estat teffects" to calculate the total, direct, and indirect effects for each path (Sobel 1987), along with standard errors using the delta method. To test the significance of *individual* paths from each CED to frailty (i.e. through each adult risk and resource), I used the "nlcom" command to calculate the products of the coefficients and their corresponding standard errors.

2.5.3.1. Supplemental Analysis

In addition to the analyses listed above, several supplemental analyses were also performed. First, several other coding schemes for the childhood exposure domains were considered including a trichotomous variable for each (0,1, 2+ indicators) and an alternative dichotomous scheme (2+ misfortunes versus otherwise). Second, a cumulative measure of all childhood exposure variables was created (0-27), without the infectious disease indicators (See Table S.2.1). Third, preliminary analyses using frailty as an ordered scale were also performed, where 3+ indicators indicated "frailty", 1-2 indicated "pre-frailty," and individuals with no indicators were considered "robust" (Fried, 2001; see Table A.2.1). Fourth, a squared term for BMI and an ordinal coding scheme for alcohol consumption were considered to test for a curvilinear relationship between these adult risks and frailty (results not shown)

2.6. Results

2.6.1. Sample Characteristics

The descriptive statistics for all variables at baseline (2006/2008) are presented in Table 2.1. On a scale of 0-5 frailty indicators, the average frailty score was 1.04 (SE=1.00), with approximately one third (33.93%) of the sample being free from any frailty. Among the

childhood experience domains, respondents were most likely to experience infectious diseases (92.65%) and socioeconomic misfortunes (76.63%). The majority of respondents experienced one or more risky parental behaviors (66.74%), about 30% had a chronic disease in childhood, but far fewer experienced impairments (16.52%) or risky adolescent behaviors (6.67%).

Over half of the sample was female (55.7%) and white (83.06%), with mean age of 74.49 years (ranging from 65-100). On average, respondents reported at least some accumulated wealth and had just over a high school education (12.57 years; SE=2.99). Most had private insurance in addition to Medicare (56.78%), whereas only about 5% also received Medicaid (Results not shown). Most respondents were currently non-smokers (90.65%) and refrained from heavy drinking (94.7%). Respondents were "overweight" on average (BMI=26.91; SE=4.86) and reported between two and three chronic diseases (2.28; SE=1.29).

2.6.2. Frailty Prevalence

Table 2.2 presents the linear regression models predicting frailty during 2006 or 2008. When adjusting for age, gender, and race in Model 1, experiencing one or more socioeconomic misfortunes ($b=0.078$, $p<.05$), risky adolescent behaviors ($b=0.170$, $p<.01$), and/or impairments ($b=0.107$, $p<.01$) are associated with more frailty. However, experiencing one or more infectious diseases was associated with *fewer* indicators of frailty ($b=-0.180$, $p<.01$). As expected, being older ($b=0.020$, $p<.001$), female ($b=0.169$, $p<.001$), and black or Hispanic ($b=0.164$, $p<.01$; $b=0.164$, $p<.01$, respectively) was associated with more frailty indicators. Because the prevalence of disease in frail adults is so pervasive (Vetrano et al., 2019), Model 2 further controls for multimorbidity. With this addition, the substantive findings regarding childhood experiences and covariates remain with only reduced effect sizes, except for socioeconomic misfortunes which no longer predicts frailty. As expected, morbidity in adulthood is associated

with more frailty: each additional chronic disease is associated with a 0.140 (p<.001) increase in frailty.

Finally, Model 3 further adjusts for adult resources and risks often associated with adult health outcomes. Again, the substantive findings regarding childhood experiences remain consistent. One or more risky adolescent behaviors (b=0.116, p<.05) and impairments (b=0.079, p<.05) are associated with increased frailty, whereas infectious disease is associated with reduced frailty (b=-0.131, p<.05). Among adult resources education (b=-0.018, p<.01), wealth (b=-0.051, p<.001), and having private insurance (b=-0.072, p<.05) are associated with less frailty; among risk factors, only current smoking (b=0.117, p<.05) and morbidity (b=0.129, p<.001) are associated with more frailty while controlling for all others. Finally, with the addition of these risks and resources, the effect of race is no longer significant.

2.6.3. Mediation Analysis

Table 2.3 presents the mediation analysis of the effects of the childhood experience domains on frailty at either 2006 or 2008. Each dash in the column labeled "Pathway" refers to a relationship between a Childhood Exposure Domain (CED) and an endogenous outcome or an indirect pathway between the CED and the outcome; indented rows involve one or more indirect effects.

As shown in the first rows of Table 2.3, there is some evidence that each of the childhood experience domains are associated with frailty, either directly or indirectly. However, infectious diseases in childhood (*b*=-0.138, p<.05) and risky adolescent behaviors (*b*=0.115, p<.05) are the only predictors *directly* associated with frailty, indicating that the effect of most of these early experiences are largely indirect through adult risks and resources. Additionally, though there is a significant total *indirect effect* of childhood chronic diseases (*b*=0.021, p<.01) and risky parental

behaviors (*b*=-0.016, p<.05), the *total effect* of these domains is not significant. These findings, in conjunction with the absence of significant relationships between these CEDs and frailty from the regression models in Table 2.2, provide evidence for relatively weak relationships for these domains. As such, in the following sections I discuss the indirect pathways only for those childhood exposure domains with significant total effects: SES, infectious diseases, risky adolescence behaviors, and impairments.

2.6.3.1. Childhood SES

The total effect of one or more childhood SES misfortunes on frailty was a 0.067 unit increase (p<.05). Among direct effects of childhood SES on adult resources, education (*b*= -1.394, p<.001) and wealth (*b*= -0.420, p<.001) were significant, but not private insurance (*b*= -0.006, p=0.726). In turn, education had direct effects on both morbidity (*b*=-0.028, p<.001) and frailty (*b*=-0.019, p<.001) as did wealth (*b*=-0.094, p<.001 and *b*=-0.051, p<.001, respectively).[3] Childhood SES was also directly associated with the adult risks of heavy drinking (*b*=-0.026, p<.001) and BMI (*b*=0.835, p<.001), but not current smoking or morbidity. However, none of these risks were directly associated with frailty, and only BMI was directly associated with morbidity (*b*=0.040, p<.001). Though there was no direct relationship between childhood SES and morbidity, the effect of morbidity on frailty was substantial as expected (*b*=0.129, p<.001). Additionally, the indirect effects associated with two adult resources were also significant, revealing that the effect of childhood SES on frailty was mediated by education (*b*=0.031, p<.001) and wealth (*b*=0.027, p<.001).

[3] Private Insurance was directly associated with frailty (b=-0.074,p<.01) but not morbidity.

2.6.3.2. Childhood Infectious Disease

In contrast to childhood SES, infectious diseases were associated with an overall 0.247 unit decrease in frailty (p<.001). In addition to its significant direct effect on frailty, one or more infectious diseases in childhood had a significant, direct effect on each adult resource: education (*b*=2.541, p<.001), wealth (*b*=0.802, p<.001), and private insurance (*b*=0.161, p<.001).[4] The indirect effects associated with each of these resources were also significant, indicating that the effect of early infectious disease on frailty was mediated by education (*b*=-0.058, p<.001), wealth (*b*=-0.051, p<.001), and having private insurance (*b*=-0.012, p.>05).[5] Infectious disease also had a direct effect on multiple adult risks including: smoking (*b*=0.042, p<.05), drinking (*b*=0.032, p<.05), and higher BMI (*b*=4.627, p<.001), but not morbidity. Despite these direct effects, however, there was no statistical evidence indicating that any adult risks mediated the effect of childhood infectious disease on frailty.

2.6.3.3. Risky Adolescent Behavior

Pathway analysis also revealed that experiencing risky adolescent behavior directly (*b*=0.115, p<.05) and indirectly (*b*=0.044, p<.01) increases frailty. In total, reporting one or more risky adolescent behaviors is associated a 0.159 unit increase in frailty (p<.01). However, unlike other childhood exposure domains, risky adolescent behavior was only directly associated with less wealth (*b*=-0.234, p<.05), higher BMI (*b*=0.877, p<.01), and morbidity (*b*=0.166, p<.05). Finally, morbidity appears to significantly mediate the effect of risky adolescent behaviors on frailty (*b*=0.021, p<.05).

[4] The direct effects associated with adult risks/resources and morbidity and frailty are stable across childhood exposure domains. As they have already been discussed for Childhood SES, they will not be listed further.
[5] It should be noted, however, that the direct effect of private insurance on morbidity was not significant.

2.6.3.4. Childhood Impairments

Among direct effects, one or more childhood impairments was not associated with any of the adult resources but was directly associated with the adult risks of higher adult BMI (b=0.767, p<.001) and morbidity (b=0.155, p<.01). Similar to risky adolescent behaviors, only adult morbidity mediates the effect of childhood impairments on frailty (b=0.020, p<.01).

To illustrate how the above coefficients map onto the proposed pathways in Figure 1.2, Figure 2.2 presents the relationships between these four CEDs, mediators, and frailty. For conciseness, only mediators with significant, direct relationships with the CED in each panel are included.

2.7. Discussion

The purpose of this study was to elucidate how a wide array of exposures during childhood influences the development of frailty— a state of increased vulnerability resulting from a decline in reserve and functioning across multiple physiologic systems associated with rapid health decline and mortality (Xue, 2011). These analyses reveal that some childhood exposures influence physical frailty decades after they are experienced, even after considering adult factors. Yet, this study finds support for cumulative inequality theory in that the effects of early misfortune are contingent on adult mediators Having access to resources in adulthood mitigates some of the noxious effects of a difficult childhood. This research contributes to the growing body of literature on the social predictors of frailty by assessing the direct and indirect effects of a wide variety of childhood exposures on condition in later life.

My first research question asked which childhood exposures were directly associated with later life frailty. Results from Table 2.2 reveal that one or more socioeconomic, risky adolescent behaviors, and/or impairments are associated with increased frailty in later life.

Though the association between SES misfortune is attenuated after considering adult risks, resources, and disease, risky adolescent behaviors remain significant. Additionally, and counterintuitively, one or more infectious diseases was associated with less frailty risk, net of adult factors. Furthermore, results from Table 2.3 indicate that each of the CEDs influenced frailty *indirectly*. Though the total effects for childhood chronic disease and risky parental behaviors were not significant, those for the other domains remained after adjusting for adult risks and resources.

Similar to work by Alvarado et al. (2008) and Gale et al. (2016), I found that one or more SES misfortunes was associated with more frailty in later life. Building on these studies, this study found that both risky adolescent behaviors and impairments also were associated with increased frailty. These findings highlight the importance of considering the effects of a wider array of childhood exposures. According to cumulative inequality theory, disparities in one domain of life (e.g., SES) can bleed over into other domains of life (e.g., health and risky behaviors) throughout the life course. This research demonstrates the effect of each CED, independent of one another. But it is likely that, in practice, these effects are cumulative. When combining the childhood exposure indicators into a cumulative measure (0-27, without infectious diseases; see Table S.2.1), the standardized effect of one additional misfortune during childhood ($B=0.041$, $p<.01$) is as nearly as detrimental to frailty as each additional year of education is protective ($B= -0.046$, $p<.01$).

Despite these noxious effects, some have suggested that some early misfortunes may actually be protective (Seery et al., 2010). Indeed, other studies found similar health protective effects of experiencing infectious disease in childhood (Kemp et al. 2018; Kubota et al., 2015; Smith et al., 2019). It is conceivable that experiencing infectious disease in childhood is an

impressionable experience that influences future health behaviors, of both parents and children, in a positive way to avoid future health problems. It is also possible that infectious diseases in childhood help to reduce future autoimmune and chronic diseases through the induction of regulatory T cells, in a kind of acquired immunity. For example, Kubota and colleagues (2015), found that the more infectious diseases contracted during childhood, the lower their risk was for mortality from cardiovascular disease. From another vantage point, the "hygiene hypothesis" (Strachan, 1989) suggests that a lack of exposure to infection (i.e., excessive hygiene) leads to defects in the establishment of an effective immune system (Kubota et al., 2015).

It should be noted, however, that this originating finding from HRS data does not imply that one should expose children to infectious diseases—the *youngest* respondents in the sample were born in 1943, twenty years before the first measles vaccine was licensed in 1963. Exposure to antigens, accomplished through vaccination, rather than the diseases themselves is an effective and safer method to train the immune system.

My second research question asked through which adult risks and resources do childhood exposures influence frailty in later life. As such, I investigated the pathways connecting the six CEDs to frailty through mediation analysis. Guided by cumulative inequality theory, I anticipated that, in addition to directly influencing frailty, childhood misfortunes would lead to increased frailty in later life through adult health, risks, and resources. Indeed, mediation results from Table 2.3 partially support this hypothesis by providing evidence for adult resources and health as significant mediators.

Among adult resources, increased education and wealth mediate the relationship between childhood SES and infectious diseases in later life. Education and wealth help to reduce the effect of the exposure by reducing frailty directly and indirectly through a reduction in

morbidity. What differs for these two pathways, however, is how the exposure itself is associated with each resource.

One or more SES exposures is associated with a decrease in wealth and education. According to cumulative inequality theory and supported by countless empirical studies (Szanton et al., 2010; Zang & Choi, 2020), poor socioeconomic status can have lasting impacts on adult health by reducing access to life course resources needed to overcome the disadvantage. Yet, if a person can manage to access those resources, through social support programs and/or human agency, the effects of early inequality can be mitigated. However, unlike SES exposure, one or more infectious diseases is associated with increased education and wealth. Given that only 7.35% of the sample did not experience any infectious diseases, it is possible that this relationship is spurious, whereby some other, unmeasured factor in childhood is influencing both infectious disease exposure and access to education.

Among adult risks, the effect of each CED was partially mediated by adult morbidity. This is largely unsurprising given the number of studies that have identified a relationship between various childhood experiences and comorbidity in later life (Ferraro et al., 2016; Henchoz et al., 2019; Pavela & Latham, 2016) as well as the established relationship between morbidity and frailty (Fried et al., 2001). In fact, though the overall indirect effects of risky adolescent behaviors and childhood impairments are significant, adult morbidity is the *only* mediator of the relationship between these two domains and frailty, making it the sole, universal mediator in these analyses among the childhood exposure domains.

More surprising, however, is that this study did not identify any of the other adult risks (current smoking, heavy drinking, or BMI) as significant mediators. As discussed in the background section, it is possible that increased BMI may be health protective for a number of

frailty indicators, particularly for unintentional weight loss. Additionally, there may be a U-shaped relationship for alcohol consumption, where moderate drinking is health protective and heavy drinking is detrimental.

Though illuminating, this study is not without its limitations, two of which are a result of the age of the sample. First, responses regarding childhood exposures are retrospective and subject to recall bias. However, this study accounts for socioeconomic resources and depressive symptoms (included in the frailty measure itself) and excludes respondents with low cognition scores as Vuolo and colleagues (2014) suggest. Additionally, I excluded respondents who had proxy responses for childhood questions to further preserve response reliability.

Second, given that one of the indicators of frailty, walking speed, is collected only for those HRS respondents 65 and older, the sample may be particularly vulnerable to selection bias. Respondents who experienced the most hardships early in life are likely not in this sample due to increased premature mortality and/or institutionalization (long-term care, incarceration, etc.). For example, in van der Linden, Sieber, and colleague's similar study of childhood misfortune and later life frailty using SHARE data (2020), they use a subjective measure of slowness to enable investigating frailty of respondents under 75 years of age. They found that more ACEs and ACHEs (adverse childhood health experiences) were both associated with higher odds of frailty. As such, the left censoring in this study is likely the reason for a lack of association between risky parental behaviors and childhood chronic diseases—two domains which most logically linked to frailty due to their traumatic nature and high association with adult disease, respectively.

Despite the acknowledged limitations, the findings of this study contribute to the understanding of the life course process of frailty development in several ways. First, this study

expands on current literature on frailty by considering the effects of a wider array of childhood experiences. Echoing the findings of others, I found poor childhood SES was one of the most consistent predictors of adult frailty—but much of the effect was due to its many indirect effects. Additionally, this study found evidence for the influence of each domain of childhood exposures, either directly and/or indirectly, and established infectious diseases as health protective for those born before the widespread use of the MMR vaccine. Second, this study elucidates the pathways from early life exposures to later life frailty. In doing so, I found that some of the effects of childhood exposures are masked when simply adjusting for adult risks and resources. Finally, given the emphasis on adult resources, rather than risks, in mediation analyses, this study contributes to CI theory's position that the noxious effects of early life are not guaranteed. Thus, for frailty, these effects can be reduced by providing improved access to education and increased wealth in adulthood.

Future studies can expand on these findings by considering the *biological* pathways that may underpin the *social* pathways identified in this study. Chronic inflammation has been a significant area of focus in studying the pathophysiology for frailty, because many frail (and pre-frail) adults have elevated levels of inflammatory markers such as IL-6 and C-reactive protein (Mooney et al., 2016; Soysal et al., 2016). Although this association suggests that stress from chronic health problems and/or noxious social conditions may be a driving force in developing frailty, longitudinal studies are needed to assess whether the resulting chronic inflammation is associated with the *development* of frailty, or simply a product of the condition.

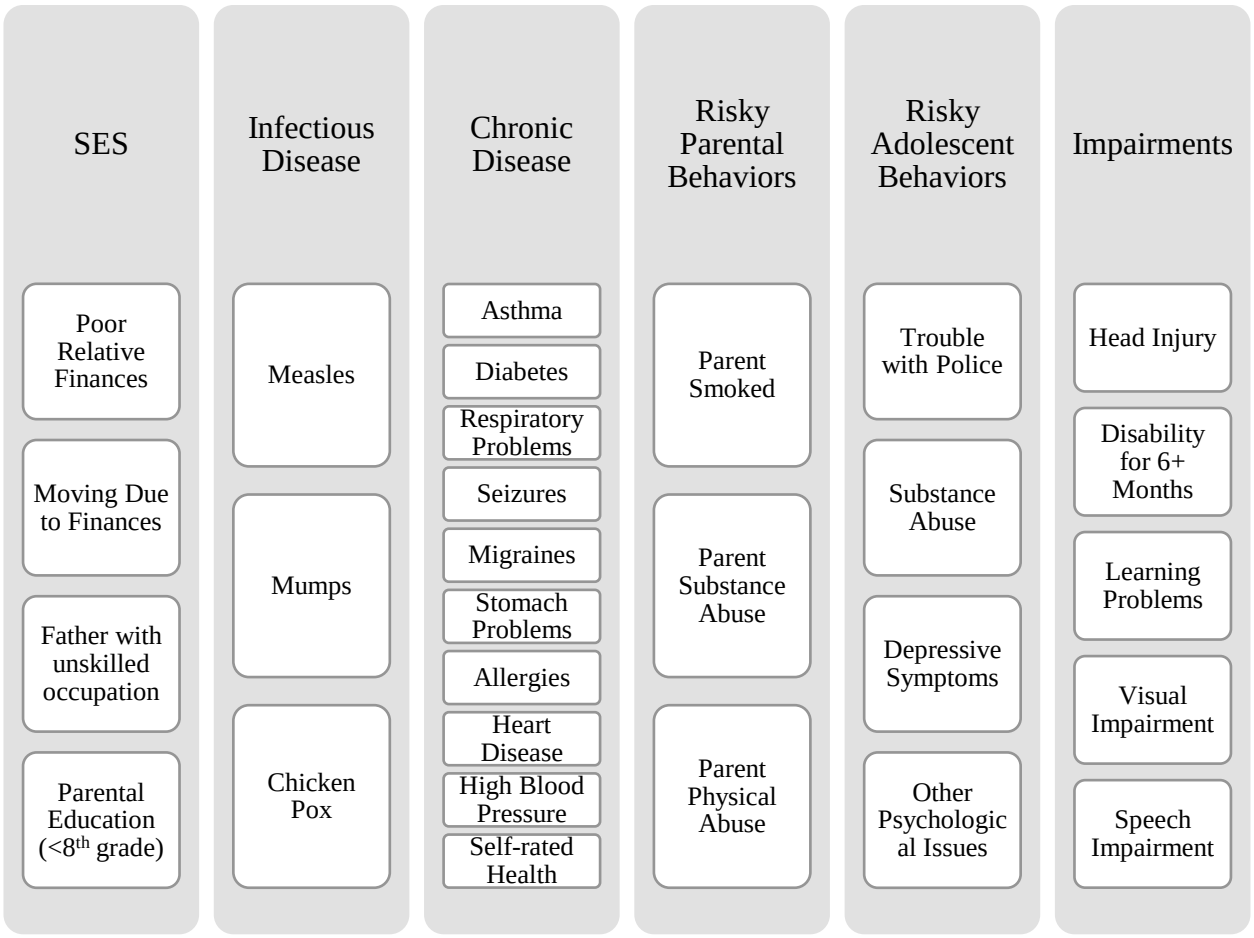


Figure 2.1. Childhood Exposures Indicators by Domain.

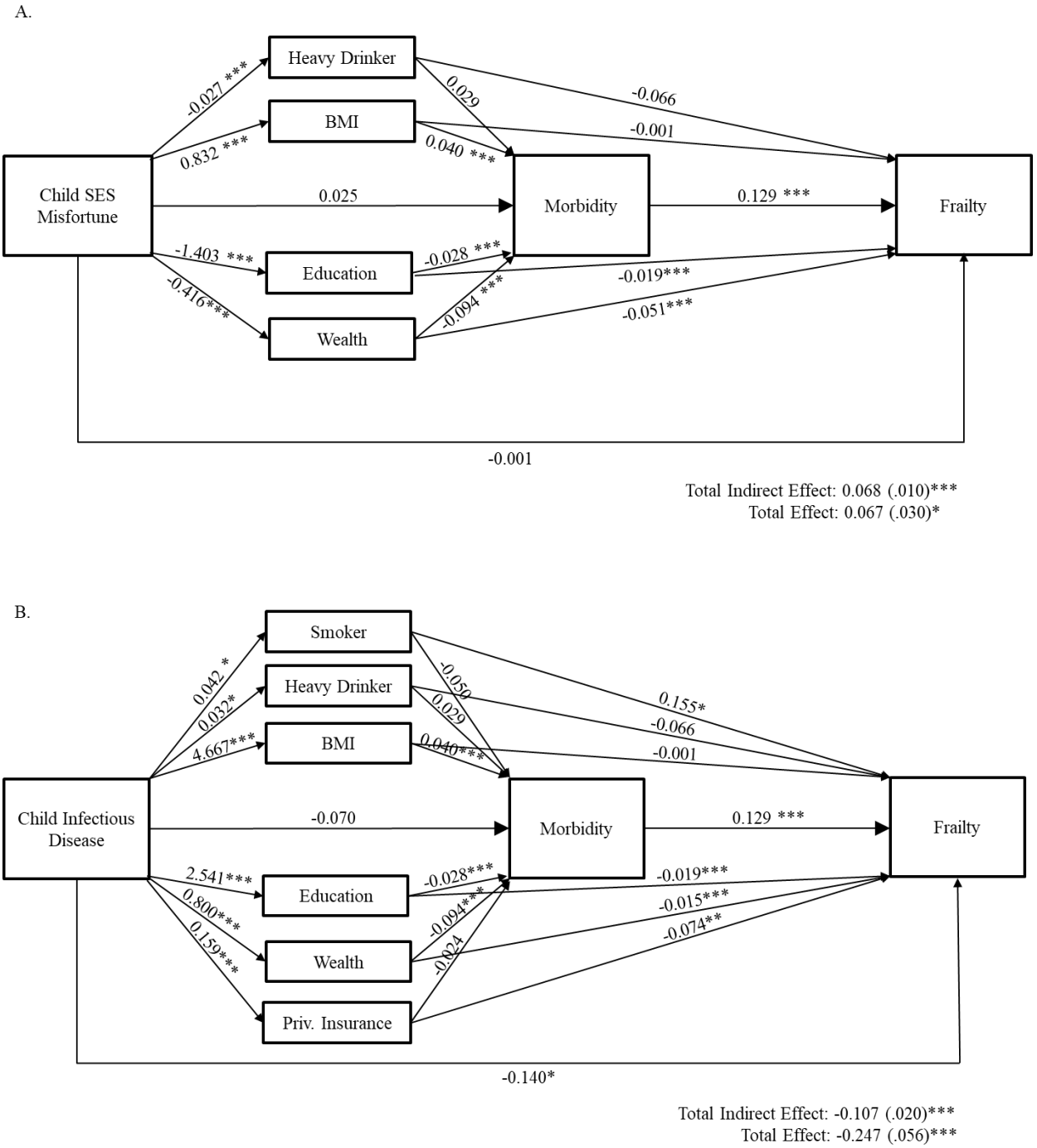
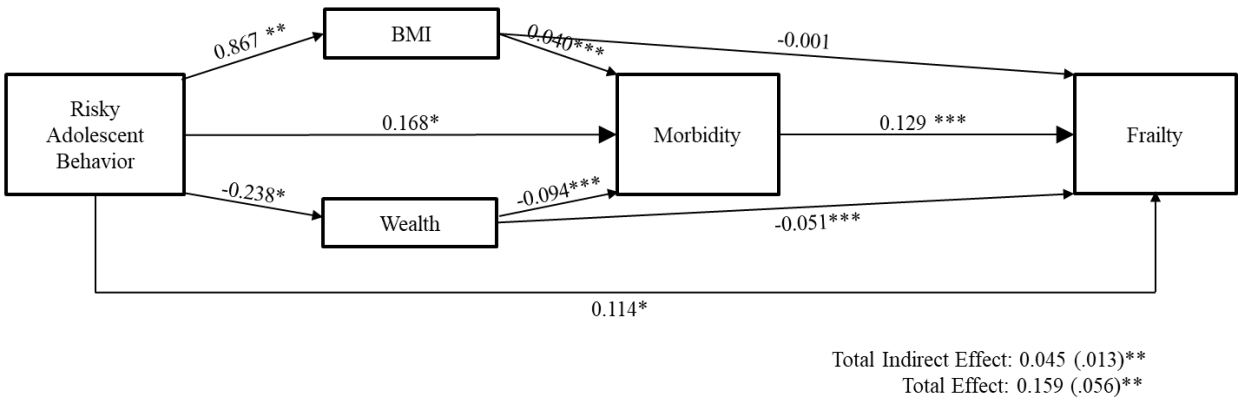


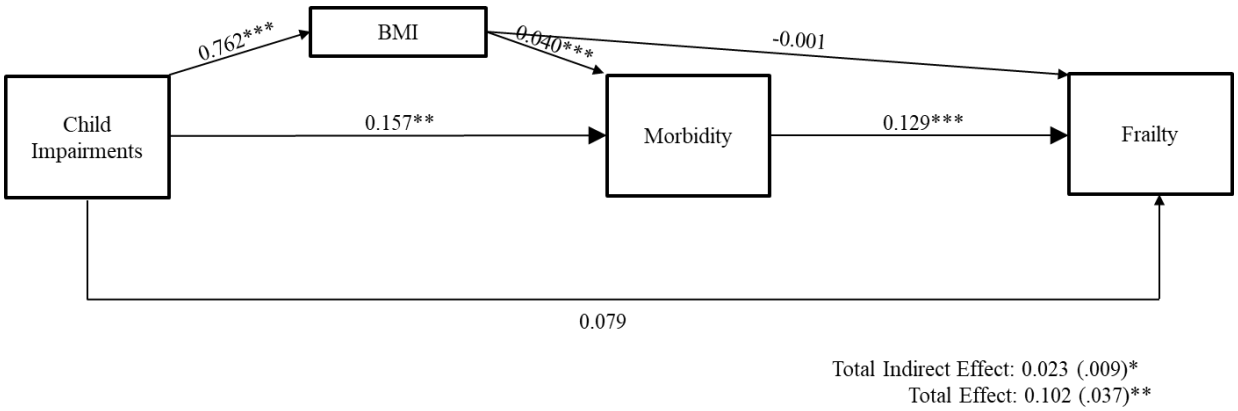
Figure 2.2. Relationships Among Select Childhood Experience Domains, Mediators, and Frailty A. Pathway Model from Childhood Socioeconomic Misfortune to Frailty. B. Pathway Model from Infectious Disease Misfortune to Frailty. Notes: *p<0.05; **p<0.01; ***p<0.001. Coefficients are unstandardized

Figure 2.2. Continued.

C.



D.



C. Pathway Model from Risky Adolescent Behavior to Frailty. D. Pathway Model from Childhood Impairments to Frailty. Notes: *p<0.05; **p<0.01; ***p<0.001. Coefficients are unstandardized.

Table 2.1. Descriptive Statistics of the Analytical Sample (N=6,805)

Variable	Range	Percent	Mean(SE)
Frailty (0-5, W1)	0-5		1.04 (1.00)
Robust (=0)		33.93%	
Pre-Frail (=1-2)		56.94%	
Frail (≥3)		9.13%	
Childhood Experiences			
1+ Socioeconomic	0,1	72.63%	
1+ Risky Parental behavior	0,1	66.74%	
1+ Infectious Disease	0,1	92.65%	
1+ Chronic Disease	0,1	29.66%	
1+ Impairments	0,1	16.51%	
1+ Risky Adolescent behavior	0,1	6.67%	
Demographics			
Age (years at baseline)	65-100		74.49 (6.89)
Female	0,1	55.77%	
Race/ethnicity			
White (ref)	0,1	83.06%	
Black	0,1	10.30%	
Hispanic	0,1	6.64%	
Adult Resources			
Education (in years)	0-17		12.57 (2.99)
Wealth (cube root in $10,000s)	-4.25-15.64		3.06 (1.74)
Private Insurance	0,1	56.93%	
Adult Risks			
Smoker	0,1	9.35%	
Heavy drinker	0,1	5.30%	
Body Mass Index	9.6-66.1		26.91 (4.86)
Morbidity	0-7		2.28 (1.29)

Table 2.2. Linear Regression of Frailty[a] During 2006 or 2008 on Predictors (N=4,655)

	Model 1	Model 2	Model 3
	b (SE)	b (SE)	b (SE)
Childhood Exposure[b]			
Socioeconomic Misfortune	0.078 (0.030)*	0.057 (0.030)	0.003 (0.031)
Infectious Disease	-0.180 (0.059)**	-0.165 (0.059)**	-0.131 (0.059)*
Chronic Disease	0.050 (0.030)	0.014 (0.030)	0.017 (0.030)
Risky Parental Behavior	0.018 (0.030)	0.010 (0.029)	0.009 (0.029)
Risky Adolescent Behavior	0.170 (0.056)**	0.148 (0.055)**	0.116 (0.055)*
Impairment	0.107 (0.037)**	0.079 (0.037)*	0.079 (0.037)*
Demographics			
Age	0.020 (0.002)***	0.016 (0.002)***	0.016 (0.002)***
Female	0.169 (0.028)***	0.189 (0.027)***	0.162 (0.028)***
Black	0.164 (0.049)**	0.142 (0.049)**	0.018 (0.051)
Hispanic	0.164 (0.061)**	0.189 (0.061)**	0.034 (0.064)
Adult Resources			
Education			-0.018 (0.005)**
Wealth			-0.051 (0.009)***
Private Insurance			-0.072 (0.028)*
Adult Risks			
Smoker			0.117 (0.048)*
Heavy Drinker			-0.064 (0.060)
BMI			0.000 (0.003)
Morbidity		0.140 (0.011)***	0.129 (0.011)***
Constant	-0.573 (0.172)**	-0.619 (0.171)***	-0.114 (0.228)

[a]Frailty= 0-5 indicators

[b]Each childhood exposure is measured as one or more indicators within each domain.

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

Table 2.3. Mediational Results for Each Childhood Exposure Domain (CED) and Frailty during 2006 or 2008 (N=4,655).

Pathway	Socioeconomic b(SE)[a]	Infectious Disease b(SE)[a]	Chronic Disease b(SE)[a]
CED—Frailty	-0.002 (0.031)	-0.138 (0.057)*	0.017 (0.030)
Total Indirect Effect	0.069 (0.010)***	-0.108 (0.020)***	0.021 (0.008)**
Total Effect	0.067 (0.030)*	-0.247 (0.056)***	0.037 (0.030)
CED—Education	-1.394 (0.094)***	2.541 (0.173)***	0.402 (0.094)***
Education—Frailty	-0.019 (0.005)***	-0.019 (0.005)***	-0.019 (0.005)***
Education—Morbidity	-0.028 (0.007)***	-0.028 (0.007)***	-0.028 (0.007)***
Indirect Effect	0.031 (0.007)***	-0.058 (0.014)***	-0.009 (0.003)**
CED—Wealth	-0.420 (0.053)***	0.802 (0.098)***	0.049 (0.053)
Wealth—Frailty	-0.051 (0.009)***	-0.051 (0.009)***	-0.051 (0.009)***
Wealth—Morbidity	-0.094 (0.012)***	-0.094 (0.012)***	-0.094 (0.012)***
Indirect Effect	0.027 (0.005)***	-0.051 (0.010)***	-0.003 (0.003)
CED—Private Ins.	-0.006 (0.016)	0.161 (0.029)***	-0.012 (0.016)
Private Ins.—Frailty	-0.072 (0.028)*	-0.072 (0.028)*	-0.072 (0.028)*
Private Ins.—Morbidity	-0.023 (0.037)	-0.023 (0.037)	-0.023 (0.037)
Indirect Effect	0.000 (0.001)	-0.012 (0.005)*	0.001 (0.001)
CED—Smoker	0.013 (0.009)	0.042 (0.017)*	-0.008 (0.009)
Smoker—Frailty	0.112 (0.047)*	0.112 (0.047)*	0.112 (0.047)*
Smoker—Morbidity	-0.053 (0.062)	-0.053 (0.062)	-0.053 (0.062)
Indirect Effect	0.001 (0.001)	0.005 (0.003)	-0.001 (0.001)
CED—Drinker	-0.026 (0.007)***	0.032 (0.013)*	0.001 (0.007)
Drinker—Frailty	-0.066 (0.060)	-0.066 (0.060)	-0.066 (0.060)
Drinker—Morbidity	0.028 (0.080)	0.028 (0.080)	0.028 (0.080)
Indirect Effect	0.002 (0.002)	-0.002 (0.002)	-0.000 (0.000)
CED—BMI	0.835 (0.177)***	4.627 (0.324)***	0.371 (0.176)*
BMI—Frailty	-0.001 (0.003)	-0.001 (0.003)	-0.001 (0.003)
BMI—Morbidity	0.040 (0.003)***	0.040 (0.003)***	0.040 (0.003)***
Indirect Effect	0.003 (0.002)	0.020 (0.012)	0.002 (0.001)
CED—Morbidity	0.027 (0.041)	-0.073 (0.075)	0.245 (0.039)***
Morbidity—Frailty	0.129 (0.011)***	0.129 (0.011)***	0.129 (0.011)***
Indirect Effect	0.004 (0.005)	-0.009 (0.010)	0.033 (0.006)***

Table 2.3. Continued.

	Risky Parent	Risky Adolescent	Impairments
	b(SE)[a]	b(SE)[a]	b(SE)[a]
CED—Frailty	0.007 (0.029)	0.115 (0.055)*	0.078 (0.037)
Total Indirect Effect	-0.016 (0.008)*	0.044 (0.013)**	0.022 (0.009)*
Total Effect	-0.013 (0.029)	0.159 (0.056)**	0.102 (0.037)**
CED—Education	0.507 (0.090)***	-0.041 (0.174)	0.213 (0.115)
Education—Frailty	-0.019 (0.005)***	-0.019 (0.005)***	-0.019 (0.005)***
Education—Morbidity	-0.028 (0.007)***	-0.028 (0.007)***	-0.028 (0.007)***
Indirect Effect	-0.011 (0.003)**	0.001 (0.004)	-0.005(0.003)
CED—Wealth	0.243 (0.051)***	-0.234 (0.098)*	-0.039 (0.065)
Wealth—Frailty	-0.051 (0.009)***	-0.051 (0.009)***	-0.051 (0.009)***
Wealth—Morbidity	-0.094 (0.012)***	-0.094 (0.012)***	-0.094 (0.012)***
Indirect Effect	-0.015 (0.004)***	0.015 (0.007)	0.003 (0.004)
CED—Private Ins.	0.044 (0.015)**	-0.057 (0.029)	-0.027 (0.019)
Private Ins.—Frailty	-0.072 (0.028)*	-0.072 (0.028)*	-0.072 (0.028)*
Private Ins.—Morbidity	-0.034 (0.037)	-0.023 (0.037)	-0.023 (0.037)
Indirect Effect	-0.003 (.002)*	0.004 (0.003)	0.002 (.002)
CED—Smoker	0.037 (0.009)***	-0.008 (0.017)	-0.012 (0.012)
Smoker—Frailty	0.112 (0.047)*	0.112 (0.047)*	0.112 (0.047)*
Smoker—Morbidity	-0.053 (0.062)	-0.053 (0.062)	-0.053 (0.062)
Indirect Effect	0.004 (0.002)	-0.001 (0.002)	-0.001 (0.001)
CED—Drinker	0.034 (0.007)***	0.008 (0.014)	-0.010 (0.009)
Drinker—Frailty	-0.066 (0.060)	-0.066 (0.060)	-0.066 (0.060)
Drinker—Morbidity	0.028 (0.080)	0.028 (0.080)	0.028 (0.080)
Indirect Effect	-0.002 (0.002)	-0.001 (0.001)	0.001 (0.001)
CED—BMI	1.512 (0.168)***	0.877 (0.326)**	0.767 (0.216)***
BMI—Frailty	-0.001 (0.003)	-0.001 (0.003)	-0.001 (0.003)
BMI—Morbidity	0.040 (0.003)***	0.040 (0.003)***	0.040 (0.003)***
Indirect Effect	0.006 (0.004)	0.004 (0.003)	0.003 (0.002)
CED—Morbidity	0.047 (0.038)	0.166 (0.073)*	0.155 (0.049)**
Morbidity—Frailty	0.129 (0.011)***	0.129 (0.011)***	0.129 (0.011)***
Indirect Effect	0.006 (0.005)	0.021 (0.010)*	0.020 (0.006)**

Note: Model adjusts for age, gender, and race.
[a]Unstandardized coefficient (standard error)
***p<.001, **p<.01, *p<.05

Table S.2.1. Linear Regression of Frailty[a] During 2006 or 2008 on Accumulated Misfortune.

	Model 1	Model 2	Model 3
	B[b] (SE)	B[b] (SE)	B[b] (SE)
ACM[c]	0.093 (0.007)***	0.064 (0.007)***	0.041 (0.007)**
Demographics			
Age	0.142 (0.002)***	0.115 (0.002)***	0.115 (0.002)***
Female	0.080 (0.026)***	0.092 (0.026)***	0.079 (0.027)***
Black	0.056 (0.047)***	0.058 (0.047)***	0.012 (0.048)
Hispanic	0.050 (0.058)***	0.189 (0.011)***	0.019 (0.060)
Adult Resources			
Education			-0.046 (0.005)**
Wealth			-0.090 (0.009)***
Medigap/Private			-0.044 (0.027)**
Adult Risks			
Smoker			0.045 (0.046)**
Heavy Drinker			-0.013 (0.059)
BMI			0.004 (0.003)***
Morbidity		1.276 (0.031)***	0.174 (0.011)***
Constant	-0.785 (0.150)***	-0.787 (0.149)***	-0.319 (0.210)

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

[a]Frailty: 0=robust, 1-2=pre-frail, 3+= frail

[b]Standardized coefficients

[c]ACM= accumulated childhood misfortune; 0-27 indicators excluding measles, mumps, and chicken pox indicators.

CHAPTER 3. TRAJECTORIES OF FRAILTY: LIFE COURSE PREDICTORS AND SOCIAL MEDIATING MECHANISMS

3.1. Introduction

Over the past few decades, empirical research has looked beyond proximal risk factors to illustrate the effect that early life experiences can have on health throughout the life course. Research on adverse childhood experiences (ACEs) has demonstrated the noxious effects that early abuse, neglect, and household dysfunction have on a host of adult health outcomes (Campbell et al., 2016; Felitti et al., 1998; Gilbert et al., 2015). Furthermore, parallel research on childhood misfortune has linked cancer (Morton et al., 2012; Kemp et al. 2018), stroke (Zaborenko et al., 2020), comorbidity (Schafer & Ferraro, 2012; Williams et al. 2019), and poor mental health (Lyu & Agrigoroaei, 2017; Morton & Ferraro, 2018), to less traumatic, but moderately disadvantageous experiences such as low SES, poor health, and impairments during childhood. As evidence for this relationship continues to compile, researchers have begun to try and explain the mechanisms by which these early experiences shape health.

Some researchers argue that the effects of childhood exposures are so potent that they can influence later life health directly via health programming. However, recent research on physical health outcomes, including frailty, suggests that these early experiences work largely indirectly throughout the life course via biological and social processes (Kendig et al, 2017; Lyu & Agrigoroaei, 2017). Theoretically, each of these mechanisms hold merit and are likely interrelated—social processes in everyday life can lead to biological changes under the skin. Yet, no one study can effectively assess the combined effects of each.

This study uses cumulative inequality theory and the stress process model to elucidate *one* mechanism—social relationships—by which early misfortune influences physical health

trajectories in later life. Guided by cumulative inequality theory I aim to address two empirical questions. First, do childhood exposures influence trajectories of physical frailty; second, do social relationships mediate, or reduce, the effects of early misfortune on frailty over time? I begin with an explanation of the theoretical background that frames the present study and move to a review of the prior empirical literature.

3.2. Theoretical Framework

The primary theoretical framework for this study comes from cumulative inequality theory (Ferraro & Shippee, 2009). This mid-range theory, developed to bridge the gap between empirical research with theory, links social systems, risk and resource accumulation, and human agency to individual health trajectories. Cumulative inequality (CI) theory is a useful framework for this study because it recognizes that childhood is a sensitive period for adult health and advocates for a systematic examination how of early-life events and exposures shaper later-life health outcomes. Three elements of CI theory inform this study and are elucidated below.

First, CI maintains that childhood exposures are important in influencing the health of older adults. Secondly, the theory states that whereas advantage increases exposures to opportunity, disadvantage increases exposure to risk. Combined these ideas suggest that while a privileged childhood can propel individuals along a favorable life trajectory, experiencing misfortunes or disadvantage during childhood can spark a seemingly unstoppable cascade of inequality which accumulates over the life course. In this way, CI theory identifies childhood exposure as a social precursor to later life health,

Indeed, empirical research has identified such a link. This research has revealed that poor health among older adults may be the result of exposures during childhood including socioeconomic misfortune (Cohen et al., 2010; Luo & Waite, 2005), poor health (Blackwell et al.

2001; Brandt et al., 2012), risky households (Williams et al. 2019), and disability or impairments (Stevenson et al., 2019). Furthermore, when one considers that disadvantage tends to diffuse across domains, these effects can be additive (Felitti et al., 1998; Zaborenko et al., 2020). Cumulative inequality theory provides a foundation from which to examine these studies of the life-long effects of early insults.

Third, though the effects of early misfortune may feel inexorable to those who experience them, CI theory maintains that the effects of early disadvantage are not deterministic. Rather, trajectories are modifiable through personal agency and resource mobilization. Relatedly, turning points, particularly those which provide access to such coping mechanisms, can alter consequences of risk accumulation (Ferraro & Shippee, 2009).

One such resource identified by researchers is social relationships. Social support is associated with health across the life course, in that those who are socially embedded, and experience supportive relationships are healthier than their isolated, and socially unsupported counterparts (Lyu & Agrigoroai, 2017). Researchers argue that social support may directly influence health but also may do so by helping to buffer the effects of stressful circumstances (House, Umberson, and Landis, 1988). In this way, one may hypothesize that high levels of social support may help to reduce the detrimental health effects of childhood misfortune. Additionally, it is conceivable that older adults who are married, have children, and have formed relationships with extended family and friends may be more equipped to combat a lifetime of stress accumulation, because these relationships represent more potential sources of support and coping.

However, it should be noted that although agency and resource mobilization can alter unfavorable trajectories, one must consider these processes within the overall structural, and

historical, context. Individual decisions and health behaviors are not made in a vacuum: behind every decision are structures and institutions that provide unequal access to risks and resources throughout the life course. To suggest that agency alone can combat these forces is misguided. Adversities early in life, such as abuse and family dysfunction, tend to undermine important psychosocial resources, including social support. For example, children from risky households, who are more likely to also have limited financial resources, have poor social competence and emotion regulation relative to children from more stable homes, which results in difficulties forming and maintaining meaningful social ties throughout the life course (Cohen et al, 2010; Miller et al., 2011). Though salient and supportive relationships may be harder to come by for those who experience early misfortune, this research investigates the potential ameliorating effects of social support in addition to the exacerbating effects of social strain.

3.3. Literature Review

3.2.1. Frailty over time

An established line of research over the past two decades has solidified childhood exposures as an early precursor to a later life health and mortality. Experiencing childhood disadvantages such as low SES, poor childhood health, and risky households has been linked to a host of health problems in later life from self-reported mental (Lyu & Agrigoroaei, 2017) and physical health (Umberson et al., 2014) to the major causes of death (Felitti et al., 1998; Williams et al., 2019).

More recently, there has been an explosion of multidisciplinary interest in discovering the etiology of frailty among older adults. In 2019 alone, over 2,900 articles with the keyword 'frailty' were published in indexed peer-reviewed journals. These studies have identified a

number of adult risk factors for the development of frailty, including chronic disease and comorbidity (Bandeen-Roche et al., 2015; Howrey et al., 2018; Klein et al., 2005; Walston et al., 2002) as well as related adult lifestyle factors such as smoking (Hubbard et al., 2009; Kojima, et al., 2015), poor nutrition (Kuh et al., 2006; León-Muñoz et al. 2015), and sedentariness (Bandeen-Roche et al., 2015).

Researchers interested in the early origins of adult health are among those that have turned their attention to this clinical state of physiologic vulnerability. Though limited in quantity and scope, the findings from the four peer-reviewed studies which directly assess the effects of childhood experiences on frailty are illuminating; each study identified poor childhood SES as a precursor to frailty among older adults (Alvarado et al., 2008; Gale et al., 2016; van der Linden, Cheval, et al., 2020; van der Linden, Sieber, et al., 2020). Additionally, in their European-based study, van der Linden and colleagues (2020) found that wider array of exposures, including adverse experiences, poor health, and poor SES during childhood, were all associated with frailty in older adults but that the effects diminished over time, particularly once controlling for adult economic resources.

As research continues to identify the early antecedents of frailty among older adults, it will become increasingly important to understand *how* and through what mechanisms these early exposures influence this physiological state of vulnerability. Prior research has found evidence that childhood exposures can have direct and independent relationships with frailty (Williams et al., 2019; Kemp et al., 2018; Zaborenko et al., 2020). In the previous chapter of this dissertation, I found evidence to suggest that childhood exposures also influence frailty prevalence indirectly though adult risks and resources. The current study aims to assess the mediating role of social relationships in later life, specifically the role of social support, strain, and roles.

3.2.2. Childhood Exposures, Social Relationships, and Frailty

3.2.2.1. Social Relationships and Frailty

The positive effects that social relationships can have on health outcomes throughout the life course have been well documented by sociologists and health researchers and acknowledged by policy makers and the general public alike. For example, social support has been linked to better self-rated health (Villalonga-Olives et a., 2020), slower growth of chronic conditions (Mazzella et al., 2010), and reduced risk of mortality (Holt-Lunstad et al., 2010; Holt-Lunstad et al., 2015). Specifically, social support was found to be related to less-steep increases in frailty over time (Peek et al., 2012; Woo et al., 2005). However, the effects of social support may not be universally beneficial to health. Some studies find that social support can lead to poorer outcomes for older adults with disabilities or impairments, potentially through poor health modeling behaviors or a through perceived "overprotection" and subsequent feelings of helplessness and loss of mastery (Cimarolli et al., 2006).[6] Furthermore, it is possible some studies may identify a case of reverse-causality. Stress activated coping—in this case the activation of social contacts to help cope with initial frailty—may masquerade as social support *causing* health decline rather than being activated to *combat* the decline.

In general, research shows that older adults report greater satisfaction in their social networks than younger people, often due to the measures they take to minimize negative interactions (see *socioemotional selectivity theory*; Carstensen, 2006). Yet, when older adults' relationships are characterized by frequent conflict or ambivalence, they can provide a steady stream of chronic stress that may be damaging to health, particularly among older minorities

[6] Overprotection is defined as "a perception on part of the ill adults that he/she is overhelped, induced to be dependent, shielded from stress, and generally not treated as an adult." Individuals who experience the phenomenon often report being helped when unnecessary and/or being overly restricted by support providers (Thomas & Sobolew-Shubin, 1993 a&b).

(Rook & Charles, 2017). However, as with social support, it is possible that strain may, in fact, be health promoting. For example, studies have found that social strain from spouses (Xu et al., 2016) and children (Thomas & Umberson, 2018; Thomas et al., 2019) may be protective against cognitive decline and increase physical activity over time, perhaps through social control mechanisms.[7]

Finally, while social strain and support tap into relationship quality, some studies have found that network quantity or number of social roles may also influence health. One would expect that having more social roles (i.e. being a spouse, parent, friend, etc.) would provide more opportunities for social interaction and support. Though older adults are often faced with declining social roles, research has demonstrated that greater role occupancy is related to better subjective health for both women and men in later life (Adelmann, 1994), perhaps through more opportunity for involvement in positive lifestyle behaviors (Matz-Costa et al., 2016). However, it is also possible that having and maintaining too many social roles can be straining (Portes, 1998), particularly for those dealing with physical health issues, and opens the possibility for more negative interactions (Rook & Charles, 2017).

3.2.2.2. Childhood Exposures and Social Relationships

Though social relationships can be beneficial to the health of older adults, positive and salient social relationships may be hard to achieve for those who experienced less than ideal childhoods. As discussed earlier in the theoretical framework, experiencing childhood misfortune often reduces access to important psychosocial resources, including social relationships.

[7] The social control model suggests that some actions taken by spouses in an effort to promote healthier behaviors among their partners may actually impose strain on their marital relationship (Xu, Thomas, & Umberson, 2016).

According to Bradley and Corwyn (2002), parents with more education can employ several resources—including better social connections, high quality of learning experiences, and access to safe neighborhoods— which can help their children to develop better social skills and to establish supportive social relations. In contrast, research has shown that individuals who grow up with poor SES during childhood are more likely to lack such resources (Poulton & Capsi, 2005). Furthermore, experiencing poor childhood SES increases the likelihood that families are characterized by conflict and neglectful relationships, as opposed to warm and attentive ones, failing to provide their children with a healthy model from which to frame their future relationships. Relatedly, children from risky families, particularly those with martial conflict, are more likely to display poor emotional regulation and risky behaviors in adolescence, hostility, and difficulties with social competence which are carried into adulthood (Cohen et al., 2010).

Additionally, health problems and impairments during childhood have also been linked to the development of social relationships. Though families with children suffering from chronic diseases, impairments, or disabilities are often supportive and nurturing, the strain of chronic stressors such as this can also create tense familial relationships (Rayan & Ahmad, 2016). As such, childhood health and impairments can influence the development of relationships in later life through the aforementioned mechanisms. Furthermore, children with chronic diseases and/or impairments are less likely to form quality relationships with friends than their peers (Geisthardt et al., 2002; Guralnik et al., 2007). This decreased access to non-familial relationships eventually reduces the likelihood of marriage or partnership in later life (Tumin, 2016). Though the mechanisms by which different early exposures influence the number and quality of

relationships in later life, evidence suggests that experiencing childhood misfortune often creates barriers to forming and maintaining quality relationships throughout the life course.

3.4. Research Questions

Drawing from cumulative inequality and stress process theories as well as prior empirical evidence, the present study asks two questions:

1. Do childhood exposures influence trajectories of physical frailty over time?

Two recently published European studies found that adverse childhood experiences and childhood socioeconomic conditions predict frailty trajectories over time (van der Linden, Cheval et al. 2020, van der Linden, Sieber et al., 2020). This study uses sister data from the HRS to assess the effects of a more fine-grained set of childhood exposure domains on frailty over the same time period (2004-2016) among older adults in the United States.

2. Do social relationships mediate the effects of childhood exposures on frailty over time?

Empirical and theoretical work on the effects of early childhood exposures provide evidence that older adults who experienced early disadvantage are less likely to form and maintain healthy social bonds throughout the life course (Cohen et al., 2010). And despite the assumptions about the universally positive effects of social support on health, research on social support, strain, and number of social roles and their effects on older adults' health are mixed. To examine the mechanisms by which childhood exposures influence later life frailty, this study investigates whether social relationships exacerbate or ameliorate the effects of early misfortune, as illustrated in Figure 3.1.

3.5. Methods

3.5.1. Sample

Data come from the Health and Retirement Study (HRS)—a multistage, probability study of adults aged 51+ years, with an oversampling of Florida residents and Black and Hispanic adults. Waves 7 to 14 (2004 to 2016) are used for this study, because several of the childhood exposure indicators were first measured on the full sample in 2004. Variables for both the dependent variable and mediators of interest for this study are collected from surveys conducted on half-samples of respondents every four years to reduce respondent burden. As such, baseline analyses for this study come from 2006/2008 and two subsequent waves come from 2010/2012 and 2014/2016 (see Figure 3.2).

Respondents eligible for this study include those 65 years and older at baseline who consented to physical measure testing during an enhanced face-to-face, in-home survey.[8] Those who had responses from proxies for childhood experiences and whose cognition scores indicate the presence of dementia (total cognition score <6) were excluded from the sample to preserve the validity of retrospective childhood data. Finally, those with missing frailty data for all three waves were excluded.[9]

3.5.2. Measures

3.5.2.1. Frailty

Frailty is a measure composed of five indicators: unintentional weight loss, exhaustion,

[8] Those under 65 years of age were not included in the study, because walking speed was only assessed for individuals 65 and older.

[9] By HRS survey design, those who were interviewed by proxy, lived in a nursing home, or who completed a telephone interview were also excluded, because they were not asked to complete the physical measure tests (Crimmins et al., 2008).

low energy expenditure, weakness, and slowness (Fried et al., 2001). Following the work of others (Cigolle et al. 2009), *weight loss* is a calculated or self-reported weight loss of 10% or more over a two year span or a having BMI of less than 18.5 kg/m^2.[10] *Exhaustion* is assessed by an affirmative answer to either "felt activities were efforts" or "could not get going" during the past week. *Low energy expenditure* is calculated using a gender stratified activity scale based on self-reported frequency and intensity of physical activity ranging from 0-17.6 (Williams et al., 2019; Umstattd Meyer et al., 2014).[11] Respondents with activity scores in the lowest 20% of the sample distributions are considered to have low energy expenditure. *Weakness* is defined as having an assessed grip strength in the lowest 20% of the sample distribution, adjusting for sex and BMI. *Slowness* is defined as having an assessed walking speed over 8 feet in the lowest 20% of the sample distribution, adjusting for gender and height.[12] Participants who refused or could not complete either the grip strength or walking speed tests because of safety concerns or physical limitation were also classified as "weak" or "slow," respectively (Cigolle et al., 2009). These indicators were summed to create a frailty scale (0-5), where 0 indicates no reported frailty.

[10] In Fried and colleagues (2001) initial study using the biological syndrome model of frailty used data from the Cardiovascular Health Study (CHS). In the CHS, weight loss was assessed using a measure of self-reported, unintentional weight loss with the question, "In the last year, have you lost more than 10 pounds unintentionally (i.e., not due to dieting or exercise)?" Follow-up weight loss was assessed as an unintentional loss of 5% or more over the past year. Because "unintentional" weight loss is not directly assessed in the HRS, I followed the work of Cigolle et al. (2009) and others by defining weight loss as a 10% or greater weight loss in the previous two years, or having a BMI classified as "underweight" (< 18.5 kg/m2).

[11] The self-reported physical activity scale accounts for both frequency and intensity of activities. Respondents were asked about the frequency of mild, moderate, and vigorous activity they participated in. Possible responses were never/rarely (=0), one to three times per month (=1), once per week (=2), two or more times per week (=3), and every day (=4). The scale was created weighting by intensity (mild = 1.2, moderate = 1.4, and vigorous = 1.8) based on metabolic equivalent recommendations for activity (Williams et al., 2019; Umstattd Meyer et al., 2014). Possible scores ranged from 0 (no physical activity) to 17.6 (mild, moderate, and vigorous physical activity every day).

[12] Data used to assess weakness and slowness come from the physical measures subsample of the HRS conducted in the enhanced face-to-face survey.

3.5.2.2. Childhood Exposures

Childhood exposures before the age of 18 were measured with responses to 29 questions, where each indicator is coded dichotomously (1 if the condition is reported, 0 if not). Based upon previous analyses, these indicators are categorized into six domains. *Socioeconomic status (SES)* consists of 4 indicators: reporting poor or fair finances in relation to others, moving due to finances, having a father with an unskilled labor occupation, and father (or mother if not available) having less than an eighth-grade education. *Infectious disease* includes measles, mumps, and/or chicken pox. *Chronic disease* includes asthma, diabetes, respiratory problems, seizures, migraines, stomach problems, allergies, heart disease, high blood pressure, and self-rated childhood health (poor or fair=1, good, very good, excellent=0). *Risky parental behavior* is assessed by having a parent who smoked, abused substances, and/or were physically abusive. *Risky adolescent behavior* is assessed by having trouble with police, substance abuse, depressive symptoms, and/or other psychological issues. Finally, reported childhood *impairment* includes head injury, disability for 6+ months, learning problems, visual impairment, and/or speech impairment. Childhood exposure indicators were summed to create a count of exposures within each domain, where 0 indicates no experiences in that domain and 1 indicates one or more exposures (van der Linden et al., 2020).

3.5.2.3. Social Relationships

Social Relationships are assessed using social support, negative social support (or strain), and number of social roles. Perceived *social support* from spouses, children, other family, and friends is measured separately with 3 items: “how much do they really understand the way you feel about things?”; “how much can you rely on them if you have a serious problem?”; “how much can you open up to them if you need to talk about your worries?” Reponses range from a

lot (=1) to not at all (=4). An index was created by reverse coding and averaging the scores across the items; social support scores from each domain were set to missing if there is more than one item missing as suggested by the HRS user guide (Smith et al., 2017). Similarly, negative social support, or *social strain*, is also measured separately for each domain of support with 4 items: "how often do they make too many demands on you?"; "how much do they criticize you?"; "how much do they let you down when you are counting on them?"; "how much do they get on your nerves?" Items are reverse coded and averaged to create an index; strain scores were set to missing if there are more than two items missing for each domain. Each index was constructed by taking the average of all items across *all* relationship domains for which the respondent reported so that respondents scores reflect only the social ties they had. As such, a count variable of *number of social roles* was created to reflect the presence of a spouse, children, family, and/or friends by summing the number of affirmative responses (0-4). Though this measure is referred to as a count of social roles, this is a relatively crude measure and is not meant to be an exhaustive list of roles that an older adult may hold (excludes the possibility of being a volunteer, church member, etc.). However, the simplicity of the measure makes it an effective control variable for the relationship quality variables, as well as one of substantive interest as it quantifies the social roles for which support and strain input can be measured in this sample. Finally, these four roles are relatively stable amongst older adults (Rook & Charles, 2017) and are, therefore, more likely to remain despite frailty onset or worsening frailty over time.

3.5.3.1. Covariates

Models adjust for demographic characteristics including age (in years, at baseline), gender (female=1), and race/ethnicity (non-Hispanic white, non-Hispanic black, and Hispanic).

Those who did not self-identify into one of the previous three categories were excluded from analysis as the number of cases was insufficient for meaningful comparison.

Adult risks include measures of health and lifestyle factors. Morbidity, or chronic disease count (0-7), is assessed by respondent reports of doctor diagnosed arthritis, diabetes, lung disease, stroke, heart problems, hypertension, and cancer (excluding skin). Current smoking was assessed with a dichotomous indicator (=1). Heavy drinking was also assessed dichotomously as 5+ drinks per day for men and 4+ drinks per day for women (Dawson, 2011). BMI is based on self-reports ranging from 9.6-66.1 kg/m^2. *Adult resources* include socioeconomic factors and social context. Education in years is top coded at 17+. Wealth comes from RAND HRS Longitudinal File (2016), is presented in tens of thousands of dollars, and cube rooted to correct for skewness. Health insurance status is a dichotomous indicator where 1= private insurance (with or without a Medigap plan) and 0=otherwise (no insurance, Medicaid and/or Medicare only).

3.5.3. Analysis

The analysis was conducted in two parts using Stata ST/SE 15.1. To examine the life course predictors of frailty over time, a series of latent growth curve models (GCM) were estimated. First, models were estimated without any adult risks or resources to establish a relationship between the childhood exposure domains and frailty over time. Next, adult morbidity was added because of its high association with frailty, followed by adult risks and resources, simultaneously. Finally, social relationship variables were added.

To further examine the potential mediating effect of social relationships on the association between childhood exposures and frailty over time, mediation analysis was conducted using Structural Equation Modeling (SEM) and the post-estimation command “estat

teffects" to calculate the total, direct, and indirect effects for each path, along with standard errors using the delta method (Sobel, 1987). To test the significance of *individual* paths from each CED to frailty intercept and slope (i.e. through each social relationship variable and adult morbidity), I used the "nlcom" command to calculate the products of the coefficients and their corresponding standard errors.

This study uses full-information maximum likelihood (FIML) to handle missing data for on both individual variables as well as panel attrition. FIML is a theory-based approach to missing data that allows all respondents in the dataset to contribute to analysis, regardless of whether they responded to every item or even participated in every wave of the study. Rather than "imputing" missing values, it borrows information from the observed scores to estimate parameters from incomplete data. Using an iterative optimization algorithm, FIML repeatedly auditions different population parameters until the algorithm arrives at parameters that maximize log-likelihood (Enders, 2011).

3.5.3.1. Supplemental Analysis

Some investigators advocate using exposure domains to investigate the effects of childhood misfortunes on later life health outcomes (Ferraro et al., 2016), but others advocate adding the exposure indicators into a global variable (Felitti et al., 1998). To test the effects of accumulated childhood exposure on frailty trajectories and the corresponding mediation effects of social relationships, indicators were added into a variable of accumulated childhood misfortune. Because previous research has found that infectious diseases during childhood affect the health outcomes of older adults differently than the other five exposure domains (Williams et al., 2019; Zaborenko et al., 2020), the indicators for chicken pox, measles, and mumps were excluded for a final variable ranging from 0 to 27 exposures (see Appendix B).

In addition, analyses were conducted to determine who was removed from the analytical sample due to mortality during the eight-year observation of this study. Respondents who died were more likely to be older, male, current smokers with lower reported social support. Additionally, those who reported more chronic conditions and those who experienced no infectious diseases in childhood were also more likely to have died (results not shown).

3.6. Results

3.6.1. Sample Characteristics

The unadjusted descriptive statistics for all variables are presented in Table 3.1; all statistics are measured at baseline, except for frailty for which I present statistics at each wave. At wave 1 (2006/2008) the average frailty score was 1.04 (SE=1.00) on a 0-5 scale. Among respondents remaining in the study 8 years later (wave 3), the average frailty score was 1.26 (SE=1.07). For childhood exposures, respondents were most likely to experience one or more infectious diseases (92.86%) and socioeconomic misfortunes (72.63%), and many respondents experienced at least one risky parental behavior (66.74%). Just under 30% suffered from a chronic disease in childhood, but far fewer experienced impairments (16.41%) or risky adolescent behaviors (6.34%).

Respondents ranged from 65 to 100 years old at wave 1, with an average age of 74.21 (SE=6.81). There were more female respondents (58.64%) than male and the sample was majority white (11.4% black, 6.93% Hispanic). Respondents reported moderately high social support (3.17 on a scale of 1-4), with less reported social strain (2.39 on the same scale). Additionally, the average respondent reported 3.44 social roles, with the most common "missing" role being that of a spouse (results not shown). In terms of adult resources, the sample

was relatively privileged. The average respondent reported more assets than debt ($574,164.50; SD= $1,261,527.00), had a little more than a high school education (12.52 years; SE=2.99), and the majority (56.56%) had private insurance in addition to Medicare. Among adult health risks, few respondents were current smokers (9.35%) or heavy drinkers (5.19%), but respondents were "overweight" on average (BMI=27.94; SE=5.28) and reported doctor-diagnosed multimorbidity (2.31 diseases, SE=1.29).

3.6.2. Frailty Trajectories

Table 3.2 presents results of latent growth curve models that test the associations of life course predictors with initial frailty among older adults and the growth rate in frailty over time. When adjusting for only demographic characteristics in Model 1, all childhood exposure domains, apart from risky parental behaviors, are associated with initial levels of frailty at Wave 1. Exposure to one or more SES misfortunes (b=0.076, p<.01), chronic diseases (b=0.077, p<.01), risky adolescent behaviors (b=0.184, p<.001), and impairments (b=0.115, p<.001) during childhood is associated with higher initial levels of frailty, whereas one or more infectious diseases are associated with lower levels of frailty (b=-0.267, p<.001). Additionally, older adults (b=0.015, p<.001), females (b=0.174, p<.001), and black and Hispanic adults (b=0.146, p<.001; b=0.130, p<.01, respectively) also had higher initial levels of frailty. Yet, none of the childhood exposure domains were associated with the slope of frailty *over time*. The older a respondent was at Wave 1, the faster the increase in frailty over time (b=0.004, p<.001); Hispanic adults also saw a faster increase in frailty over the 8-year observation period (b=0.082, p<.05). Finally, though women had higher initial levels of frailty, they experienced a slower increase in frailty over time than their male counterparts (b=-0.038, p<.05).

With the addition of disease morbidity to the analysis in Model 2, many of the

substantive results remain, with only modest reductions in effect sizes. Not surprisingly, however, chronic disease in childhood was no longer associated with initial levels of frailty. Additionally, more doctor-diagnosed chronic diseases in later life was associated with higher initial levels of frailty (b=0.158, p<.001), but not over time.

Model 3 introduces two of the social relationship variables: social support and social strain. Again, substantive results among demographics, childhood exposure domains, and morbidity remain stable with minor reductions in effect sizes. Social support is associated with lower levels of initial frailty at Wave 1 (b=-0.160, p<.001) but not growth over time; social strain is not significantly associated with either frailty intercept or slope.

Model 4 introduces the final social relationship variable. More reported social roles (i.e., spouse, children, family, and friends) is associated with lower initial levels of frailty *and* frailty over time (b=-0.118, p<.001; -0.043, p<.05, respectively).

Model 5 builds on Model 3 by including adult risks and resources as predictors of initial frailty and frailty over time.[13] With the addition of these covariates, one or more risky adolescent behaviors and impairments continue to be associated with higher initial frailty, while infectious diseases continue to be associated with lower initial frailty. However, socioeconomic misfortunes in childhood are no longer associated with initial frailty, but one or more chronic childhood diseases is associated with higher initial frailty (b=0.049, p<.05). As expected, inclusion of adult risks and resources reduces the effect of being black or Hispanic, relative to white, to non-significance for both initially, and over time. All proposed adult resources are associated with lower initial levels of frailty, and higher education continues to be associated with slower frailty growth over time (b=-0.013, p<.001). Among adult risks, current smoking is

[13] In Table 2, Models 3 and 5 do not include the social roles variable; Models 4 and 6 do.

associated with higher levels of frailty initially (b=0.167, p<.001) and over time (b=0.082, p<.01). Heavy drinking does not influence initial levels of frailty but is associated with steeper frailty growth over time (b=-0.082, p<.05). All other substantive results remain, with minor changes to effect sizes.

Finally, Model 6 builds on Model 4 by adding adult risks and resources. Models 5 and 6 are similar, however, the latter includes number of social roles (see Figure 3.3 for a visual explanation of all Table 3.2 models). As such, Models 5 and 6 produce similar results with one exception; higher BMI is associated with higher initial frailty (b=0.006, p<.01). More social roles continue to be associated with lower initial frailty (b=-0.082, p<.001) and frailty over time (b=-0.031, p<.05).

3.6.3. Mediation Analysis

Table 3.3 presents the mediation analysis of the effects of the childhood exposure domains by social relationships on frailty growth (intercept and slope). Apart from socioeconomic misfortunes, there is some evidence that each of the childhood experience domains are associated with frailty trajectories, either directly or indirectly when controlling for demographics and adult risks/resources (shown in the first rows). Additionally, despite its detrimental, direct effect on social strain (b=0.072, p<.05) and total indirect effect on initial frailty (b=0.017, p<.01), the *total effect* of risky parental behaviors is not significant. Accordingly, in the following sections I discuss the pathways to frailty trajectories for the four remaining childhood exposures domains (infectious disease, chronic disease, risky adolescent behaviors, and impairments) first discussing initial frailty followed by growth over an 8-year period.

3.6.3.1. Initial Frailty

One or more infectious diseases was associated with a *direct* 0.115 unit decrease in frailty ($p<.01$) with a *total* 0.124 unit decrease ($p<.01$), when accounting for indirect relationships. Infectious diseases had a direct and positive effect on social support ($b=0.054$, $p<.05$) and number of social roles ($b=0.127$, $p<.001$). In turn, social support was directly associated with a reduction in both morbidity ($b=-0.101$, $p<.01$) and frailty ($b=-0.102$, $p<.001$); number of social roles was directly associated with frailty ($b=-0.078$, $p<.001$). Conversely, there was no direct relationship between infectious disease and social strain or morbidity. However, strain was associated with higher morbidity ($b= 0.099$, $p<.001$) and morbidity was directly associated with frailty ($b=0.136$, $p<.001$).[14] In total, though several indirect pathways between infectious disease and initial frailty were significant, the relationship appears to be largely direct (total indirect effect= 0.009, $p=0.326$).

Reporting at least one chronic disease during childhood was associated with a *total* 0.089 unit increase initial frailty ($p<.001$) with significant direct and indirect effects ($b=0.051$, $p<.05$; $b=0.038$, $p<.001$, respectively). Mediation analysis reveals that chronic diseases during childhood is indirectly associated with higher initial frailty through its effects on social support ($b=-0.031$, $p<.05$) and morbidity ($b=0.253$, $p<.001$) in later life.

Risky adolescent behavior was also directly ($b=0.121$, $p<.05$) and indirectly ($b=0.180$, $p<.001$) associated with initial frailty, with a total unit increase of ($b=0.180$, $p<.001$). Like chronic diseases, one or more risky adolescent behaviors is indirectly associated with higher initial frailty through reduced social support (-0.188, $p<.001$) and morbidity ($b=0.190$, $p<.01$),

[14] The direct effects associated with the three social environment variables, morbidity, and initial frailty are not dependent on the childhood exposure domains and are, therefore, the same for each panel of Table 3. As they have already been reported here, they will not be reported in subsequent paragraphs in this section.

but also through fewer social roles (b=-0.143, p<.001).

Finally, as with risky adolescent behaviors, reporting any impairments during childhood influenced initial frailty directly (b=0.076, p<.05) and indirectly (b= 0.033, p<.001) through less social support (b=-0.048, p<.01), fewer support domains (b= -0.013, p<.001), and disease morbidity (b=0.150, p<.001). In total, impairments are associated with a 0.110 unit decrease in frailty at Wave 1 (p<.001).

3.6.3.2. Frailty Over Time

Results from this analysis also reveal an indirect influence of childhood exposures on frailty over time through number of social roles. One or more chronic diseases (b=0.002, p<.01), risky adolescent behaviors (b= 0.009, p<.05), and impairments (b= 0.004, p<.05) are associated with a more rapid growth in frailty over 8 years, whereas having at least one infectious disease is associated with slower growth over time (b=-0.005, p<.05).

3.7. Discussion

The purpose of this study was to examine the effects of childhood exposures on frailty over time and to identify if and how social relationships contribute to that process. Numerous studies have established the notable effects that childhood misfortune can have on later life health outcomes, and researchers interested in the antecedents of frailty have followed suit. However, with the exception of one study (van der Linden et al, 2020), this research has yet to examine the pathways by which this relationship unfolds over time. Furthermore, this study is the first among the early origins of frailty studies to systematically examine the effect of social relationships, both as an independent variable and potential mediator.

Guided by prior empirical studies and cumulative inequality theory, I anticipated that

childhood exposures would, by and large, be associated with higher initial frailty and frailty growth over time. Specifically, I anticipated that poor SES, chronic disease, impairments, and risky parental and adolescent behaviors would be associated with worse frailty trajectories, while infectious diseases during childhood may be health protective. Each childhood exposure domain, apart from risky parental behavior, was associated with initial levels of frailty. When controlling for adult factors, exposure to one or more chronic diseases, risky adolescent behaviors, and impairments continued to be associated with higher initial levels of frailty, whereas one or more infectious diseases was associated with lower initial levels of frailty. However, none of the childhood exposure domains were *directly* associated with frailty growth, providing evidence that the effects of childhood exposures are on frailty over time work through intermediary factors throughout the life course.

I also anticipated that social relationships would have independent and mediating effects on frailty trajectories. I expected that misfortune during childhood would be associated with worse social relationships (less support, fewer social roles, and more strain). Additionally, though evidence on the directionality of the proposed relationships was mixed, I anticipated what social support and number of social roles would, in turn, be associated with more favorable frailty trajectories, whereas social strain would be associated with more frailty over time. Though I found no evidence for an independent or mediating effect of social strain, I found that social support was independently associated with lower initial frailty and helped diminish the effects of childhood chronic diseases, impairments, and risky adolescent behaviors on baseline frailty. Additionally, I found that having more social roles was independently associated with lower initial frailty and slower frailty growth over time. Finally, results revealed that infectious and chronic disease, risky adolescent behaviors, and impairments were indirectly associated with

frailty over time through number of social roles.

The above findings highlight two important conclusions about predicting frailty trajectories and, ultimately, about how to create more favorable trajectories for older adults. First, childhood exposures influence frailty in later life directly and by their influence on mid- and later-life factors over time. Similarly, van der Linden and colleagues (2020) found associations between adverse exposures, poor health, and poor SES during childhood and frailty at baseline, but that differences in frailty became smaller over time after adjusting for adult resources. Though it is possible that age is acting as a "leveler," this convergence of frailty trajectories in later life may be explained by mortality selection in older, disadvantaged adults. Indeed, supplemental analyses revealed that older males who smoke, those with low social support, more diseases, and those who experienced no early infectious diseases were more likely to exit the analytical sample due to mortality over eight years. As such, it is likely that the effects of childhood exposures on frailty trajectories are underestimated for those who died during the observation period of this study and for those who died or were institutionalized, and therefore are not represented in the HRS. According to cumulative inequality theory, as persons with the most health problems are removed from a sample, either through earlier mortality or institutionalization, the population (and sample) composition will change, resulting in a cohort inversion (Ferraro & Shippee, 2009).

Second, social relationships can be an important later life resource to help reduce the effects of childhood misfortune on frailty. Not only are social support and number of social roles independently implicated in frailty trajectories, but more support reduces the effect of a number of childhood exposures on initial frailty and more social roles reduce baseline frailty and its trajectory over time. To my knowledge, the present study is the first to analyze the mediating

effects of social relationships in the relationship between childhood exposures and frailty, but studies examining related health outcomes have reported similar findings regarding the salubrious effects of social support (Lyu & Agrigoroaei, 2017; Umberson et al., 2014). Additionally, empirical research on multiple social roles and health among older adults more generally (Adelmann, 1994) seems to confirm the role accumulation hypothesis, that roles provide the opportunity to gain access to social supports, resources, and social stimulation needed to maintain health (Thoits, 1983). From a cumulative inequality perspective, social relationships represent an important resource that may accumulate over a lifetime.

Though social relationships in later life can be health protective, particularly for those who experienced early disadvantage, this study also found that many childhood exposures are associated with lower social support and holding fewer social roles. Indeed, cumulative inequality states that while advantage (i.e., a "privileged" childhood) increases exposure to opportunity (i.e., positive social relationships), disadvantage (i.e., poor childhood health, risky behaviors, impairments) increases exposure to risk (i.e., fewer social roles and strain). Findings from this research suggest that better social support in later life may help older adults avoid frailty; and among those who may become frail, increased support may help reduce its progression over time. In fact, one randomized controlled trial aimed at improving outcomes in pre-frail and frail older adults found that providing social support, in the form of twice weekly "buddy" visits, improved overall frailty scores. Importantly, they also found that the social support intervention alone was just as effective in reducing frailty as the group assigned to a physical training and nutrition intervention (Luger et al., 2016). Admittedly, it would be challenging, if not impossible, for interventions to attempt to address the number of social roles from some salient domains (i.e., spouse and children), but studies such as this indicate that

encouraging friendship among older adults may be particularly important for improving frailty trajectories.

Yet, among these results are the rather "counterintuitive" findings regarding childhood infectious diseases and its relationship with frailty both directly and through social relationships. Other studies have found infectious diseases during childhood to be health protective for a number of health conditions in later life including stroke (Kubota et al., 2015; Zaborenko et al., 2020), handgrip-strength (Smith et al., 2019), and remaining disease free (Williams et al., 2019). Researchers propose that infectious diseases in childhood may help to reduce future disease through the induction of regulatory T cells, in a kind of acquired immunity (Kubota et al., 2015). Additionally, given the proportion of the sample who reported one or more infectious diseases (.9286), it is likely the positive association between infectious disease and social support reflects the rarity of not experiencing *any* infectious disease, particularly among this older sample. Perhaps avoiding all infectious disease during childhood is indicative of a relatively socially isolated childhood, which influenced the development and maintenance of social relationships in later life.

In addition to the main findings, I briefly discuss those which are notable among demographic characteristics and adult risks and resources. Firstly, being female was associated with higher initial frailty, but slower growth in frailty over time. This finding is echoed by much of the literature which finds that, although women may suffer from more chronic conditions, they appear to be better equipped to handle such conditions (Reiker & Bird, 2005). Second, although being a minority was associated with higher initial frailty (and frailty slope for Hispanic adults), these effects became nonsignificant after accounting for adult risks and resources—providing evidence that these factors can ameliorate or exacerbate racial disparities. Though personal

agency can be employed, structural inequality due to systemic racism reduces access to these resources and increases exposure to these risks, de facto. Education, wealth, and access to private insurance were associated with *lower* initial frailty, whereas current smoking, BMI, and morbidity are associated with higher *initial* frailty. However, among adult resources, only education was associated with slower growth over time, perhaps indicating the importance of cognitive reserve in combating frailty over time.

This study should be interpreted with the following limitations in mind. First, data used for the childhood exposure indicators was self-reported and measured retrospectively. As such, these measures may be subject to recall bias and social desirability particularly for older adults (Carstensen, 2006). However, studies on recalled measures of childhood exposures have found adequate validity (Barboza Solis et al., 2015) and this study accounts for socioeconomic resources and depressive symptoms and excludes respondents with low cognition scores as Vuolo and colleagues (2014) suggest.

Second, though the findings regarding the mediating and long-lasting effects of number of social roles are illuminating, the measure is a relatively crude one, and does not consider an exhaustive list of potential roles. As previously stated, however, these four roles are the most stable for older adults, relatively unphased by changes in health including frailty. As such, this operationalization may be more potent than other, more thorough, measures for predicting health outcomes independently and as a mediator.

Despite the acknowledged limitations, the findings of this study contribute to the literature in a number of ways. First, this study expands on current literature on frailty by considering the effects of a more fine-grained list of childhood exposures on frailty growth over time in the United States. Echoing the most recent literature, I found that childhood chronic

disease, impairments, and risky adolescent behaviors increase frailty risk in later life. Second, this study identifies the social pathways by which early exposures influence frailty trajectories. I found that both social support and more social roles mediated the relationship between childhood exposures and frailty, and that the effect of more social roles continued over time. This research has implications for interventions aimed at improving the social relationships among non-frail and pre-frail adults.

Future studies could build on these findings by considering which specific sources of social support (i.e., spouses, children, family or friends) are the most effective in reducing frailty among older adults. Furthermore, studies have found that the mediating mechanisms by which social relationships influence the relationship between childhood disadvantage and later life health may differ by race and ethnicity (Umberson et al., 2014). As such, future studies should take an intersectional approach to investigate the full spectrum of effects that social relationships may have on later life frailty trajectories.

M1= CE Domains + Demos
M2= CE Domains + Demos + Morbidity
M3= CE Domains + Demos + Morbidity + Support/Strain
M4= CE Domains + Demos + Morbidity + Support/Strain **&** Network Comp.
M5= CE Domains + Demos + Morbidity + Support/Strain + Adult Risks/Resources
M6= CE Domains + Demos + Morbidity + Support/Strain **&** Network Comp. + Adult Risks/Resources

Figure 3.1. Explanation of Table 2 models. CE= Childhood Exposure; Demos= Demographics; Comp.=Composition.

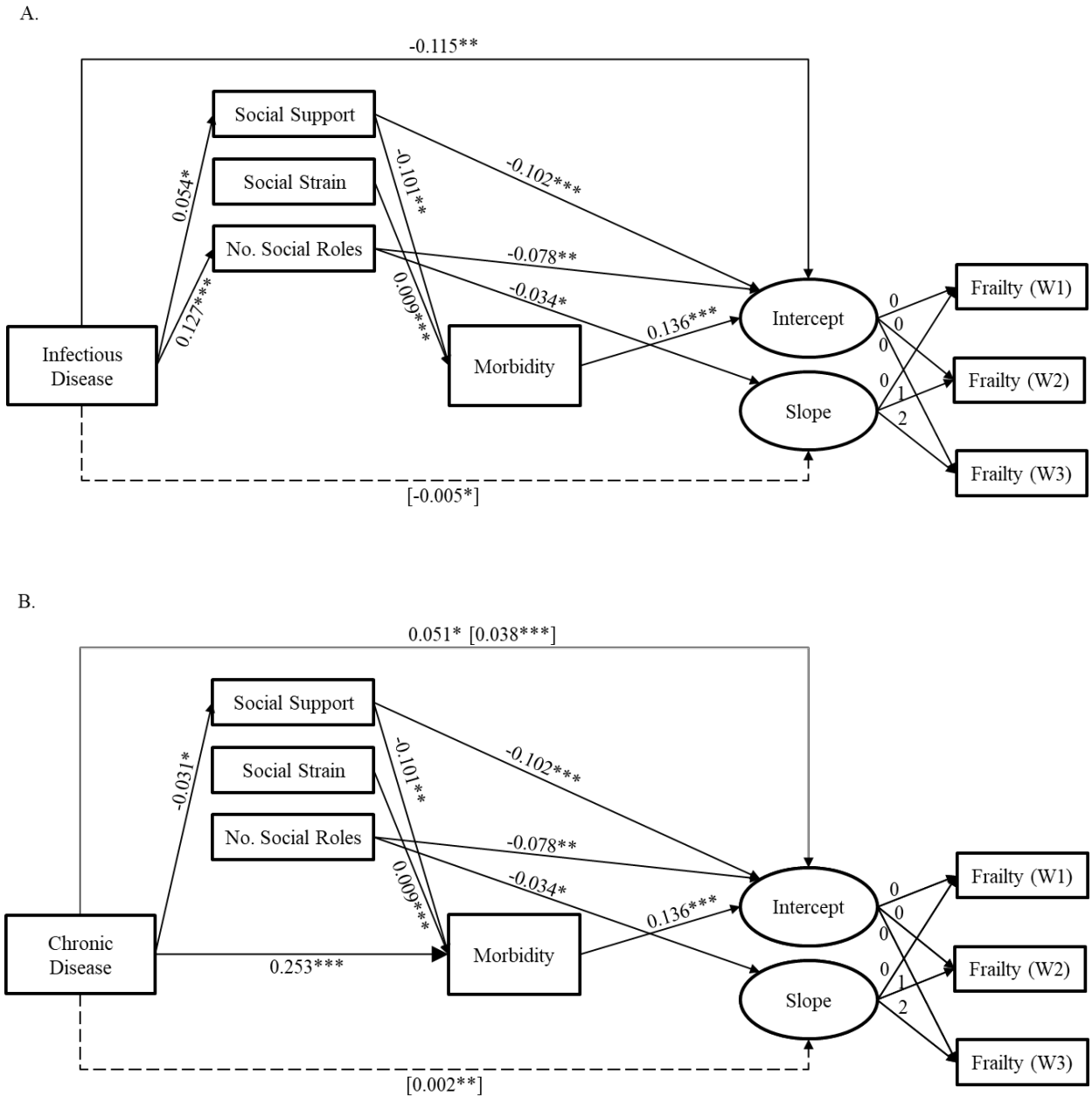
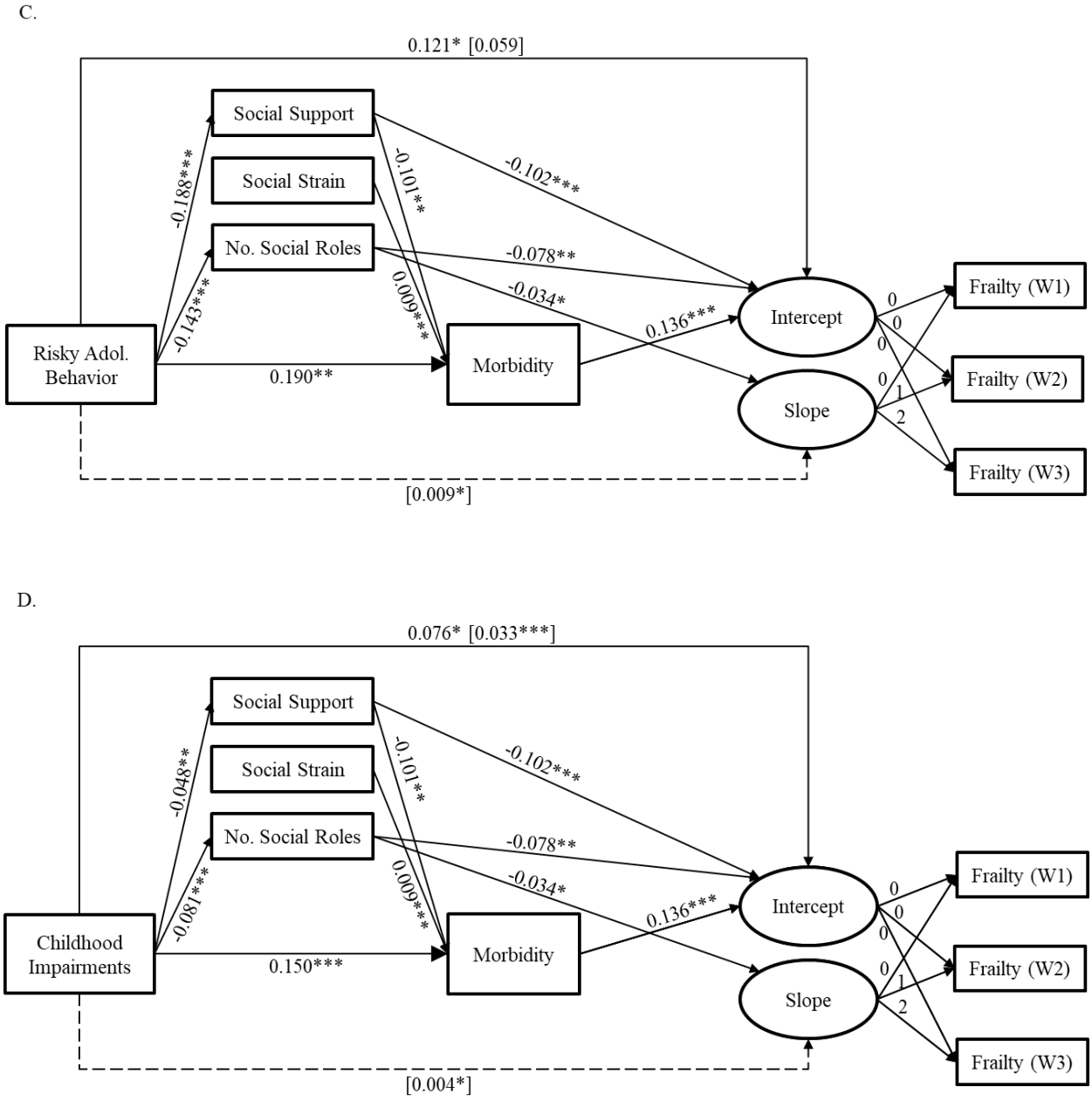


Figure 3.2. Relationships Among Select Childhood Exposure Domains, Social Relationships, and Frailty Trajectories. A. Pathway model from childhood infectious disease to frailty. B. Pathway model from childhood chronic disease to frailty. Direct effects presented as solid arrows. Total indirect effects of exposure domains on frailty are presented in brackets with dashed arrows or next to reported direct effects. Notes: *p<0.05; **p<0.01; ***p<0.001. Coefficients are unstandardized.

Figure 3.2. Continued.



C. Pathway model from risky adolescent behavior to frailty. D. Pathway model from childhood impairments to frailty. Direct effects presented as solid arrows. Indirect effects of exposure domains on frailty are presented in brackets with dashed arrows or next to reported direct effects. Notes: *p<0.05; **p<0.01; ***p<0.001. Coefficients are unstandardized.

Table 3.1. Descriptive Statistics of the Analytical sample (N=7,712)

Variable	Range	Percent	Mean(SE)
Frailty			
Wave 1	0-5		1.04 (1.00)
Wave 2	0-5		1.17 (1.04)
Wave 3	0-5		1.26 (1.07)
Childhood Exposures			
1+ Socioeconomic	0,1	72.63%	
1+ Infectious Disease	0,1	92.86%	
1+ Chronic Disease	0,1	29.78%	
1+ Risky Parental Behavior	0,1	66.79%	
1+ Risky Adolescent Behavior	0,1	6.34%	
1+ Impairments	0,1	16.41%	
Demographics			
Age (years at baseline)	65-100		74.21 (6.81)
Female	0,1	58.64%	
Race/ethnicity			
White (ref)	0,1	81.66%	
Black	0,1	11.4%	
Hispanic	0,1	6.93%	
Social Relationships			
Social Support	1-4		3.17 (0.52)
Social Strain	1-4		2.39 (0.94)
Number of Social Roles	0-4		3.44 (0.69)
Adult Resources			
Education (in years)	0-17		12.52 (3.01)
Wealth (cube root in $10,000s)	-4.25-15.64		3.00 (1.75)
Private Insurance	0,1	56.56%	
Adult Risks			
Smoker	0,1	9.35%	
Heavy drinker	0,1	5.19%	
Body Mass Index	12.3-67.8		27.49 (5.28)
Morbidity	0-7		2.31 (1.29)

Note: unadjusted values presented.

Table 3.2. Growth Curve Model Estimates of the Effects of Life Course Predictors on Frailty (N=7,712)

	Model 1		Model 2	
	Intercept	Slope	Intercept	Slope
	Coef. (SE)	Coef. (SE)	Coef. (SE)	Coef. (SE)
Childhood Exposure				
Socioeconomic	0.076 (0.027)**	-0.003 (0.020)	0.052 (0.026)*	-0.003 (0.020)
Infectious Disease	-0.267 (0.044)***	-0.069 (0.036)	-0.267 (0.043)***	-0.063 (0.036)
Chronic Disease	0.077 (0.026)**	-0.003 (0.019)	0.037 (0.025)	-0.004 (0.019)
Risky Parental B.	-0.033 (0.025)	-0.010 (0.019)	-0.048 (0.024)	-0.013 (0.019)
Risky Adol. B.	0.184 (0.052)***	-0.049 (0.038)	0.146 (0.051)**	-0.049 (0.038)
Impairment	0.115 (0.031)***	-0.011 (0.024)	0.085 (0.031)**	-0.014 (0.024)
Demographics				
Age	0.015 (0.001)***	0.004 (0.001)***	0.010 (0.001)***	0.003 (0.001)***
Female	0.174 (0.023)***	-0.038 (0.018)*	0.191 (0.023)***	-0.040 (0.018)*
Black	0.146 (0.037)***	0.007 (0.029)	0.110 (0.037)**	-0.001 (0.029)
Hispanic	0.130 (0.047)**	0.082 (0.035)*	0.142 (0.046)**	0.077 (0.035)*
Adult Resources				
Education				
Wealth				
Private Insurance				
Adult Risks				
Morbidity			0.158 (0.009)***	0.013 (0.007)
Smoker				
Heavy Drinker				
BMI				
Social Relationships				
Social Support				
Social Strain				
No. Social Roles				
Model fit index	CFI= 0.927; RMSEA= 0.040		CFI= 0.936; RMSEA= 0.039	

	Model 3		Model 4	
	Intercept	Slope	Intercept	Slope
	Coef. (SE)	Coef. (SE)	Coef. (SE)	Coef. (SE)
Childhood Exposure				
Socioeconomic	0.056 (0.026)*	-0.002 (0.020)	0.061 (0.026)*	-0.003 (0.020)
Infectious Disease	-0.206 (0.043)***	-0.055 (0.036)	-0.135 (0.044)**	-0.040 (0.037)
Chronic Disease	0.039 (0.025)	-0.003 (0.019)	0.044 (0.025)	0.000 (0.019)
Risky Parental B.	-0.032 (0.024)	-0.012 (0.019)	-0.007 (0.024)	-0.008 (0.019)
Risky Adol. B.	0.124 (0.051)*	-0.051 (0.038)	0.126 (0.051)*	-0.048 (0.037)
Impairment	0.083 (0.030)**	-0.014 (0.024)	0.081 (0.030)**	-0.016 (0.024)
Demographics				
Age	0.016 (0.001)***	0.005 (0.001)***	0.019 (0.001)***	0.006 (0.001)***
Female	0.210 (0.023)***	-0.037 (0.018)*	0.176 (0.023)***	-0.047 (0.018)**
Black	0.137 (0.037)***	0.005 (0.029)	0.124 (0.037)**	-0.003 (0.029)
Hispanic	0.167 (0.046)***	0.080 (0.035)*	0.183 (0.046)***	0.083 (0.035)*
Adult Resources				

Table 3.2. Continued.

Education				
Wealth				
Private Insurance				
Adult Risks				
Morbidity	0.155 (0.009)***	0.012 (0.007)	0.153 (0.009)***	0.012 (0.007)
Smoker				
Heavy Drinker				
BMI				
Social Relationships				
Social Support	-0.160 (0.021)***	-0.025 (0.017)	-0.116 (0.022)***	-0.017 (0.017)
Social Strain	0.008 (0.013)	-0.009 (0.010)	0.009 (0.013)	-0.008 (0.010)
No. Social Roles			-0.118 (0.016)***	-0.043 (0.013)**
Model fit index	CFI= 0.961; RMSEA= 0.029		CFI= 0.985; RMSEA= 0.018	

	Model 5		Model 6	
	Intercept	Slope	Intercept	Slope
	Coef. (SE)	Coef. (SE)	Coef. (SE)	Coef. (SE)
Childhood Exposure				
Socioeconomic	-0.006 (0.027)	-0.023 (0.021)	0.003 (0.027)	-0.022 (0.021)
Infectious Disease	-0.143 (0.044)**	-0.045 (0.037)	-0.114 (0.044)*	-0.037 (0.037)
Chronic Disease	0.049 (0.025)*	0.005 (0.019)	0.051 (0.025)*	0.007 (0.019)
Risky Parental B.	-0.014 (0.024)	-0.013 (0.019)	-0.004 (0.024)	-0.011 (0.019)
Risky Adol. B.	0.121 (0.050)*	-0.058 (0.037)	0.119 (0.050)*	-0.055 (0.037)
Impairment	0.079 (0.030)**	-0.012 (0.024)	0.076 (0.030)*	-0.013 (0.023)
Demographics				
Age	0.020 (0.001)***	0.007 (0.001)***	0.021 (0.001)***	0.007 (0.001)***
Female	0.172 (0.023)***	-0.046 (0.018)*	0.156 (0.023)***	-0.054 (0.018)**
Black	0.028 (0.038)	-0.018 (0.030)	0.025 (0.038)	-0.021 (0.030)
Hispanic	0.002 (0.048)	0.029 (0.037)	0.025 (0.048)	0.036 (0.037)
Adult Resources				
Education	-0.021 (0.004)***	-0.013 (0.003)***	-0.019 (0.004)***	-0.012 (0.003)***
Wealth	-0.053 (0.007)***	-0.011 (0.006)	-0.045 (0.007)***	-0.008 (0.006)
Private Insurance	-0.072 (0.023)**	0.015 (0.018)	-0.066 (0.023)**	0.017 (0.018)
Adult Risks				
Morbidity	0.138 (0.009)***	0.006 (0.007)	0.136 (0.009)***	0.005 (0.007)
Smoker	0.167 (0.038)***	0.082 (0.031)**	0.173 (0.038)***	0.084 (0.031)**
Heavy Drinker	-0.072 (0.049)	0.082 (0.038)*	-0.070 (0.049)	0.083 (0.038)*
BMI	0.003 (0.002)	0.002 (0.002)	0.006 (0.002)**	0.002 (0.002)
Social Relationships				
Social Support	-0.120 (0.022)***	-0.021 (0.017)	-0.102 (0.022)***	-0.016 (0.017)
Social Strain	0.006 (0.013)	-0.007 (0.010)	0.006 (0.013)	-0.007 (0.010)
No. Social Roles			-0.082 (0.017)***	-0.032 (0.013)*
Model fit index	CFI= 0.973; RMSEA= 0.022		CFI= 0.982; RMSEA= 0.017	

Note: Frailty measured 0-5 scale. Each childhood exposure is measured as 1+ indicators within each domain.; CFI= comparative fit index; RMSEA= root mean square error of approximation.***p<.001, **p<.01, *p<.05

Table 3.3. Social Relationship Mediation Results from Growth Curve Model Estimates of the Effects of Life Course Predictors on Frailty (N=7,712).

	Socioeconomic Misfortunes		Infectious Diseases	
	Intercept	Slope	Intercept	Slope
CED—Frailty	0.003 (0.027)	-0.022 (0.021)	-0.115 (0.044)**	-0.036 (0.037)
Total Indirect Effect	0.007 (0.005)	0.001 (0.001)	-0.009 (0.009)	-0.005 (0.002)*
Total Effect	0.010 (0.027)	-0.021 (0.021)	-0.124 (0.045)**	-0.041 (0.037)
CED—Support	-0.009 (0.014)	-	0.054 (0.026)*	-
Support—Morbidity	-0.101 (0.029)**	-	-0.101 (0.029)**	-
Support—Frailty	-0.102 (0.022)***	-0.016 (0.017)	-0.102 (0.022)***	-0.016 (0.017)
Indirect Effect	0.001 (0.002)	0.000 (0.000)	-0.006 (0.003)	-0.001 (0.001)
CED—Strain	-0.047 (0.026)	-	0.077 (0.047)	-
Strain —Morbidity	0.099 (0.016)***	-	0.099 (0.016)***	-
Strain —Frailty	0.007 (0.013)	-0.007 (0.010)	0.007 (0.013)	-0.007 (0.010)
Indirect Effect	-0.001 ((0.001)	0.000 (0.000)	0.001 (0.001)	-0.000 (0.001)
CED—Roles	-0.009 (0.019)	-	0.127 (0.034)***	-
Roles— Morbidity	-0.013 (0.024)	-	-0.013 (0.024)	-
Roles— Frailty	-0.078 (0.027)***	-0.034 (0.013)*	-0.078 (0.027)***	-0.034 (0.013)*
Indirect Effect	0.001 (0.002)	0.000 (0.001)	-0.010 (0.003)**	-0.004 (0.002)*
CED—Morbidity	0.047 (0.035)		0.041 (0.060)	
Morbidity—Frailty	0.136 (0.009)***	0.005 (0.007)	0.136 (0.009)***	0.005 (0.007)
Indirect Effect	0.006 (0.005)	0.000 (0.000)	0.006 (0.008)	0.000 (0.000)

	Chronic Diseases		Risky Parental Behaviors	
	Intercept	Slope	Intercept	Slope
CED—Frailty	0.051 (0.025)*	0.007 (0.019)	-0.005 (0.024)	-0.011 (0.019)
Total Indirect Effect	0.038 (0.005)***	0.002 (0.002)**	0.017 (0.005)**	0.000 (0.001)
Total Effect	0.089 (0.025)***	0.009 (0.019)	0.012 (0.025)	-0.011 (0.019)
CED—Support	-0.031 (0.014)*	-	-0.023 (0.013)	-
Support— Morbidity	-0.101 (0.029)**	-	-0.101 (0.029)**	-
Support— Frailty	-0.102 (0.022)***	-0.016 (0.017)	-0.102 (0.022)***	-0.016 (0.017)
Indirect Effect	0.004 (0.002)*	0.001 (0.001)	0.003 (0.002)	0.000 (0.000)
CED—Strain	-0.027 (0.025)	-	0.072 (0.024)**	-
Strain — Morbidity	0.099 (0.016)***	-	0.099 (0.016)***	-
Strain — Frailty	0.007 (0.013)	-0.007 (0.010)	0.007 (0.013)	-0.007 (0.010)
Indirect Effect	-0.001 (0.001)	0.000 (0.000)	0.001 (0.001)	-0.000 (0.001)
CED—Roles	-0.007 (0.018)	-	0.013 (0.018)	-
Roles— Morbidity	-0.013 (0.024)	-	-0.013 (0.024)	-
Roles— Frailty	-0.078 (0.027)***	-0.034 (0.013)*	-0.078 (0.027)***	-0.034 (0.013)*
Indirect Effect	0.001 (0.001)	0.000 (0.001)	-0.001 (0.001)	0.000 (0.001)
CED—Morbidity	0.253 (0.032)***		0.102 (0.31)**	
Morbidity—Frailty	0.136 (0.009)***	0.005 (0.007)	0.136 (0.009)***	0.005 (0.007)
Indirect Effect	0.035 (0.005)***	0.001 (0.002)	0.014 (0.004)**	0.005 (0.001)

(Continued on next page.)

Table 3.3. Continued.

	Risky Adolescent Behaviors		Impairments	
	Intercept	Slope	Intercept	Slope
CED—Frailty	0.121 (0.050)*	-0.055 (0.037)	0.076 (0.030)*	-0.014 (0.023)
Total Indirect Effect	0.059 (0.011)***	0.009 (0.004)*	0.033 (0.006)***	0.004 (0.002)*
Total Effect	0.180 (0.051)***	-0.046 (0.037)	0.110 (0.031)***	-0.009 (0.023)
CED—Support	-0.188 (0.027)***	-	-0.048 (0.017)**	-
Support— Morbidity	-0.101 (0.029)**	-	-0.101 (0.029)**	-
Support— Frailty	-0.102 (0.022)***	-0.016 (0.017)	-0.102 (0.022)***	-0.016 (0.017)
Indirect Effect	0.022 (0.005)***	0.003 (0.003)	0.006 (0.0020*	0.001 (0.001)
CED—Strain	-0.004 (0.049)	-	0.043 (0.030)	-
Strain — Morbidity	0.099 (0.016)***	-	0.099 (0.016)***	-
Strain — Frailty	0.007 (0.013)	-0.007 (0.010)	0.007 (0.013)	-0.007 (0.010)
Indirect Effect	-0.000 (0.001)	0.000 (0.000)	0.001 (0.001)	-0.000 (0.000)
CED—Roles	-0.143 (0.036)***	-	-0.081 (0.022)***	-
Roles— Morbidity	-0.013 (0.024)	-	-0.013 (0.024)	-
Roles— Frailty	-0.078 (0.027)***	-0.034 (0.013)*	-0.078 (0.027)***	-0.034 (0.013)*
Indirect Effect	0.011 (0.004)**	0.005 (0.002)*	0.007 (0.002)**	0.003 (0.001)*
CED—Morbidity	0.190 (0.064)**		0.150 (0.039)***	
Morbidity—Frailty	0.136 (0.009)***	0.005 (0.007)	0.136 (0.009)***	0.005 (0.007)
Indirect Effect	0.026 (0.008)**	0.001 (0.001)	0.020 (0.005)***	0.001 (0.001)

Note: Frailty is measured on a scale of 0-5 indicators. Each childhood exposure is measured as one or more indicators within each domain. Model adjusts for demographic characteristics and adult risks and resources. Unstandardized coefficients are presented; standard errors are in parentheses. Each dash (-) in first column refers to a relationship between a Childhood Exposure Domain and an endogenous outcome or an indirect pathway between the CED and the outcome; indented rows involve one or more indirect effects. Comparative fit index (CFI)= 0.856; Root mean square error of approximation (RMSEA)= 0.049. CED= childhood exposure domain. ***p<.001, **p<.01, *p

CHAPTER 4. CONCLUSION

4.1. Summary and Discussion of Findings

A now well-established body of literature has demonstrated the effects that early life exposures can have on a host of later life conditions from self-rated health (Irving & Ferraro, 2006; Lyu & Agrigoroaei, 2017) to premature mortality (Chen et al., 2016; Lee & Ryff, 2019). Research has demonstrated that childhood exposures can influence these specific conditions both directly and indirectly through later life risks (Bandeen-Roche et al., 2015; Howrey et al., 2018; Hubbard et al., 2009; Klein et al., 2005; Kojima et al., 2015; Song et al., 2015; Walston et al., 2002) and resources (Hoogendijk et al., 2014; Lyu & Agrigoroaei, 2017; Szanton et al., 2010; Umberson et al., 2014; Woo et al., 2005). Frailty, a clinical state of increased, and often extreme, vulnerability to stressors affecting multiple physiologic systems represents a unique public health challenge, because the current US healthcare system is inadequately designed to address such a complex syndrome (Kojima, 2019). Yet, research on the childhood origins of frailty is limited, and studies addressing the corresponding mediating pathways are rare. To further understand the etiology of frailty, and to address these notable literature gaps, the primary goal of this dissertation was to examine the life course predictors of frailty and to elucidate how childhood exposures influence frailty trajectories among older adults.

Guided generally by the life course perspective, and more specifically cumulative inequality theory, I conducted two empirical studies with the goal of addressing three primary aims. In the following sections I summarize and discuss the findings from chapters 2 and 3 as they relate to the overarching goals of this dissertation.

4.1.1. Aim 1: Life Course Predictors of Frailty Trajectories

For aim 1, I investigated *the early and later-life predictors of initial frailty and frailty growth over time among older, US adults.* Existing studies on the effects of childhood exposures on frailty most often investigated the effects of early-life maltreatment, poor socioeconomic status, and poor health (Alvarado et al., 2008; Gale et al., 2016; van der Linden, Cheval, et al., 2020; van der Linden, Sieber, et al., 2020). Furthermore, these studies often fail to consider the *cumulative* effects of experiencing misfortunes in more than one domain—e.g. SES and health. Even the most recent, inclusive study by van der Linden and colleagues (2020) using SHARE data did not consider the effects of adolescent experiences or distinguish between impairments and chronic/infectious diseases. My research added to prior studies by using data from the United States to investigate a wider array of childhood exposures and the combined effects of those misfortunes.

This aim was partially addressed in the first study (chapter 2) by investigating the effects of six domains of childhood exposure on frailty prevalence. Controlling for only demographic characteristics, I found that one or more SES, risky adolescent behaviors, and impairments was associated with higher baseline frailty while one or more infectious diseases was associated with lower baseline frailty. When controlling for adult disease, risks, and resources, effect sizes were attenuated, to non-significance for SES, but childhood exposures remained predictors of frailty prevalence. This study also examined the effects of later life morbidity, risks, and resources on baseline frailty. I found that each proposed resource—education, wealth, and private insurance—was independently associated with lower baseline frailty; however, among adult risks, only smoking and morbidity were associated with higher frailty.

Building on prevalence results, study 2 (chapter 3), further addressed this aim by examining the same life course predictors on frailty trajectories over eight years. Like the

previous chapter, I found that in addition to SES, risky adolescent behaviors, and impairments, one or more chronic diseases in childhood was associated with higher initial frailty trajectories while one or more infectious diseases was associated with lower initial frailty. Again, the addition of all adult risks and resources reduced the effects of childhood exposures on frailty; however, all but SES remained significant predictors of initial frailty. Each adult resource was associated with lower initial frailty levels and more education reduced the slope of frailty growth over time. Among adult risks, morbidity, smoking and BMI were associated with higher initial frailty, but only smoking and heavy drinking were associated with steeper frailty growth over time.

Taken together, these findings highlight some of the life course predictors of later life frailty. Beginning with those adult risks and resources most proximal to frailty, a few findings are particularly notable. First, each proposed resource—wealth, education, and private insurance—was associated with lower frailty prevalence; however, education was the most salient as it was also associated with a reduction in frailty growth over time. Indeed, other studies have found more education to be associated with less frailty (Leigh, 1998; Santos-Eggimann et al., 2009) and other related outcomes such handgrip strength (Hairi et al., 2009) and disability (Montez et al., 2017). Based on previous literature, it is possible that education helps to reduce frailty through increased cognitive reserve, its effect on learned health behaviors, employment opportunities, and subsequent wealth (Brunello et al., 2016). Though more research is needed on the mechanisms by which education reduces frailty initially and over time, this is an important finding; unlike some other potential resources (e.g. wealth), education may be more easily targeted through interventions.

Among adult risks, there were surprisingly few statistically significant predictors of initial frailty and frailty over time. Though morbidity was associated with initial frailty, it surprisingly did not predict growth over time. Only current smoking and heavy drinking were associated with steeper frailty growth over the eight-year observation period. Though illuminating, this is certainly not a shocking discovery. Further, when evaluating the policy implications of such a finding, one must consider the levels of mastery and correlating sense of illness trajectories among older, frail adults who are current smokers and/or heavy drinkers. From a cumulative inequality lens, people generally have a sense of how they are faring health-wise, which influences their subsequent actions (Ferraro & Shippee, 2009). These older, frail adults may hold a negative evaluation of their future health trajectories, and feel it is out of their hands. Indeed, research has found that personal mastery attenuates the effect of frailty on physical decline trajectories and is theorized to do so, in part, through its positive influence on health behaviors (Lee et al., 2016). So, it may be possible to assume that older adults who continue to engage in these activities *despite* a rapid and devastating health decline have low levels of mastery and are unlikely to benefit from a focused intervention to improve health behaviors.

Among the early life predictors of frailty, impairments, risky adolescent behaviors, and infectious disease exposures were most salient in predicting frailty. Though this is the first study to consider the effects of these domains on frailty simultaneously, previous empirical studies found similar links between these early exposures and frailty-related outcomes. Researchers have found that experiencing a childhood impairment is associated with lower handgrip strength among men (Smith et al., 2019) and childhood depression is associated with stroke incidence (Zaborenko et al., 2020). These findings support CI theory's proposition that childhood

conditions are indeed important for adult health. Moreover, the effects of these misfortunes are likely additive when experienced simultaneously—which is likely considering the cumulative nature of childhood exposures (Ferraro & Shippee, 2009). For example, studies have found greater risk-taking behaviors among adolescents with learning disabilities (McNamara et al. 2010). Future studies should consider the potential for multiplicative effects of numerous domains of childhood misfortune on later life frailty.

Although childhood impairments and risky adolescent behaviors consistently predicted higher frailty scores, one or more infectious diseases appeared to be health protective—a finding echoed by studies of other, later life health outcomes (Kemp et al. 2018; Kubota et al., 2015; Smith et al., 2019; Williams et al., 2019). As noted in previous chapters, it is important to interpret this finding considering this study's sample. Respondents ranged from 65 to 100 years old, meaning the youngest respondents were born years before the first measles vaccine was licensed in 1963. Though one explanation for this association is acquired immunity, exposure to antigens, through vaccination, is safer method to train the immune system.

Despite the importance of these findings, perhaps equally as illuminating were the null findings regarding the other childhood exposures domains. Based on the findings of other life course health studies, I anticipated that SES, risky parental behaviors, and chronic diseases would be particularly important for predicting frailty—yet such a relationship was not observed. Cumulative inequality highlights the importance of human agency and resource mobilization to reduce the effects of early disadvantage. As such, it was important to consider these pathways.

4.1.2. Aim 2: The Mediating Role of Adults Risks and Resources

In addressing the first aim of this dissertation, I found that a number of childhood exposures independently influenced frailty in later life. As previously discussed, prior studies

had also identified a link between those same early exposures and the mid- and later- life risks and resources associated with frailty (Bandeen-Roche et al., 2015; Hoogendijk et al., 2014; Howrey et al., 2018; Hubbard et al., 2009; Klein et al., 2005; Kojima et al., 2015; Song et al., Szanton et al., 2010; 2015; Walston et al., 2002; Woo et al., 2005). As such, it was also important to consider more indirect paths from early exposures to frailty to fully understand the true impact of these exposures. Accordingly, aim 2 builds on the findings of Aim 1 by examining *if childhood exposures influence frailty directly and/or indirectly through adult risks and resources.*

Using mediational analysis in study 1, I found evidence that each of the childhood experience domains were associated either directly or indirectly with frailty prevalence. However, only the infectious diseases and risky adolescent behavior domains were *directly* associated with frailty, confirming that early exposure domains influence frailty largely indirectly—through adult risks and resources. Among adult resources I found, specifically, that education and wealth mediate the relationship between childhood SES and infectious diseases and frailty in later life. Education and wealth help to reduce frailty directly and indirectly through a reduction in morbidity. Among adult risks, I found that each childhood exposure domain was mediated by adult morbidity—for risky adolescent behavior and impairments, morbidity was the only significant mediator.

This research highlights the importance of adult resources in helping to reduce the effects of early misfortunes. Education is particularly important for frailty in older adults as it *directly* reduces initial frailty and growth over time, *indirectly* reduces frailty through reduced morbidity, and *mediates* the relationship between two domains of childhood exposures and frailty. As previously discussed, the effect of more years of education and educational attainment on health

outcomes throughout the life course is well-established. But these results further establish receiving advanced education as a potential "turning point" to alter chains of risk set forth by early disadvantage (Ferraro & Shippee, 2009). According to prior empirical and theoretical research, education is thought to reduce the effects of early misfortune on later life health through one of three mechanisms: (1) "learned effectiveness," (2) employment opportunities, and (3) accumulated wealth. In the first mechanism, the health protective effect of education on health, net of income, is the result of education-induced healthy lifestyles and giving individuals a feeling of personal agency or mastery (Mirowsky & Ross, 2003; Ross & Wu, 1995; Taylor, 2011). In this way, it is the education in and of itself which reduces health risk. In the second, having higher education—specifically receiving a degree—reduces unemployment and increases the likelihood of having a full-time, rewarding career which is in turn associated with better health (Backé et al., 2012; Hibbard & Pope, 1993). Additionally, more education is typically associated with higher income; thus, work and better socioeconomic conditions of the well-educated may protect their health.

Third, and related to my previous statement, wealth accumulation can be viewed as a resource that serves to *protect* individuals from a host of life course stressors. Furthermore, research has also found that wealth *buffers* the effects of negative health events through multiple forms of "financial-based capital" (e.g. assets, formal and informal care, health information, etc.; Taylor, 2011). It should be noted, however, that while education increases access to wealth, familial wealth earlier in the life course also increases access to these forms of capital, independent of higher education. As such, possessing life course wealth *and* more education represents a particularly potent form of accumulated health capital. Though the HRS collects detailed information about wealth, it does not collect data on degree conferral, so determining the

mechanisms by which education helps to reduce the effects of childhood exposures is challenging. Future studies should investigate these proposed pathways.

The findings from aim 2 also confirm that failing to account for indirect relationships between childhood exposures and frailty through adult risks and resources would underestimate their effects. As hypothesized following the results discussed in aim 1, childhood SES, chronic diseases, and risky parental behaviors were indeed associated with frailty, but indirectly *through* a reduction in wealth and education. According to cumulative inequality theory, advantage increases exposure to opportunity, but disadvantage also increases exposure to risk (Ferraro & Shippee, 2009). In this case, experiencing childhood exposures most often linked to poor health outcomes in later life was associated with more frailty because those experiences may constrain opportunities in adulthood.

4.1.3. Aim 3: The Mediating Role of Social Relationships

The third aim, tested in the study 2 (Chapter 3), examined *the role that social relationships play in frailty trajectories among older adults.* Empirical and theoretical work on the effects of early childhood exposures suggests that older adults who experienced early disadvantage are less likely to form and maintain healthy social relationships throughout the life course (Cohen et al., 2010; Geisthardt et al., 2002; Guralnik et al., 2007; Rayan & Ahmad, 2016) furthermore, research on the direction of the relationship between social support, strain, and number of social roles and their effects on older adults' health is mixed (Adelmann, 1994; Cimarolli et al., 2006; Peek et al., 2012; Rook & Charles, 2017; Thomas & Umberson, 2018; Thomas et al., 2019; Xu et al., 2016). Using mediational analysis, I found that social support was independently associated with lower initial frailty and helped to reduce the effects of childhood chronic diseases, impairments, and risky adolescent behaviors on initial frailty.

Additionally, I found that the number of close social roles older adults claim was independently associated with lower initial frailty and slower frailty growth over time. Finally, results revealed that infectious and chronic disease, risky adolescent behaviors, and impairments were indirectly associated with frailty over time through number of social roles.

Prior studies on self-rated physical health, morbidity, and functional limitation have reported similar findings regarding the positive, mediating effects of social support (Lyu & Agrigoroaei, 2017; Umberson et al., 2014). This dissertation was the first to analyze the mediating effects of social relationships in the link between childhood exposures and frailty and results point to an overall positive mediating effect on initial frailty. Analyses did not reveal an effect for frailty growth over time; however, it is possible that any benefits of social support over time are camouflaged by stress-activated coping processes, whereby more frail adults receive increasing support from support networks. To address this possibility, future studies could assess the differential effects of emotional versus instrumental or informational support on frailty among older adults.

This study also found support for the "role accumulation process," finding that more social roles help to protect against frailty onset *and* growth over time. This finding is particularly important given that many older adults are faced with a loss of salient social roles due to illness and mortality. However, as discussed in chapter 3, this measure of social roles is relatively crude and excludes other roles of particular importance to many older adults (including volunteer, church member, and grandparent) that are less susceptible to aging-related loss. Though the role accumulation hypothesis would expect to see further health benefits of these additional roles, it may be that a more comprehensive measure of social roles would uncover a role strain effect due to too many roles (i.e., a nonlinear relationship). Intervention studies

aiming to increase the number of social roles of older adults in an effort to reduce frailty should consider this possibility.

Though social support and social roles are important for reducing frailty trajectories, particularly for those who experienced early disadvantage, it is also true that childhood exposures reduce access to both resources. According to cumulative inequality theory, disadvantage increases exposure to risk (Ferraro & Shippee, 2009). Indeed, experiencing poor childhood health and impairments and engaging in risky adolescent behaviors were all associated with fewer social roles and less social support in adulthood. In order to intervene in these mechanisms, however, more research is needed on the relationship between childhood exposures and social relationships earlier in the life course. Despite the established effects of social relationships on frailty and the transmission of social skills from childhood to adulthood, it is possible that childhood and mid-life social relationships would have an independent effect on frailty. If so, it would present additional opportunities to help prevent frailty decades prior.

4.2. Data Limitations

Although Chapters 2 and 3 each acknowledge study-specific limitations, there are two overarching limitations which warrant further discussion. One important limitation to consider when interpreting the findings from this dissertation is the age of the sample and the selection bias that it likely introduces. Because one of the indicators of frailty (walking speed) is collected only for respondents 65 and older, the sample for this dissertation may be particularly vulnerable to survivorship bias; potential respondents who experienced the most early- and mid-life hardships are less likely to survive long enough to qualify as an HRS participant and are even less likely to make it into my older sample (due to institutionalization or early mortality). This study finds a relative lack of evidence for higher frailty among minorities and/or those who

experienced arguably the harshest of childhood experiences (risky parental behaviors). But instead of this being a substantive finding, it is likely that this dissertation suggests a cross-over effect whereby these disadvantaged subgroups experience systematically higher mortality at younger ages, leaving a more robust group of disadvantaged individuals for observation and study. As age increases, the composition of the population is weighted toward the robust members of the disadvantaged subgroup, that gives the illusion of a cross-over effect in overall health.

A second limitation related to the age of the sample is recall bias. Respondents in this sample are 65 and older and are asked to recall events from a nearly a half century prior (at a minimum). Research suggests, however, that bias most often manifests as false negatives (Hardt & Rutter, 2004), indicating that, if anything, retrospective studies of childhood exposures are conservative estimates of early disadvantage. Nonetheless, this study addresses this possibility by accounting for socioeconomic resources and depressive symptoms (included in the frailty measure itself) and excludes respondents with low cognition scores as Vuolo and colleagues (2014) suggest. Additionally, I excluded respondents who had proxy responses for childhood questions and used respondents' earliest responses about these experiences to further preserve response reliability.

Yet, even when considering these limitations, the HRS is a fantastic dataset by which to study the life course etiologies of many health outcomes among older US adults. Response rates are relatively high (85% or higher for each core wave) and the existence of seventeen sister studies[15] across the world, lends itself for meaningful cross-national comparison.

[15] Sister studies include those conducted in Brazil (ELSI), China (CHARLS), Costa Rica (CRELES), England (ELSA), Europe (SHARE), India (LASI), Indonesia (IFLS), Ireland (TiLDA), Japan (JSTAR), Korea (KLoSA), Malaysia (MARS), Mexico (MHAS), Northern Ireland (NICOLA), South Africa (HAALSI), Scotland (HAGIS), Thailand (HART), and SAGE conducted by the World Health Organization.

4.3. Future Directions

Prior research has established frailty as an important area of interest for life course health scholars across disciplines. Recently, researchers have begun to understand how early life exposures influence the development and progression of this syndrome. This dissertation builds on the current literature by investigating the life course predictors of frailty prevalence and growth over time and discovering how childhood exposures directly and indirectly affect frailty, through adult risks and resources, including social relationships. Guided by recent findings, including those from this dissertation, there are endless future directions for studying the life course etiologies of frailty among older adults, several of which I propose in Table 4.1. Here, I focus on five—addressing three substantive and two methodological questions.

First, future research should differentiate between relationship domains of social support when assessing potential benefits. This research suggests that more social support is associated with less frailty but using an averaged measure across all domains may mask variability. For example, Lyu and Agrigoroaei (2017) found that only social support from family reduces the effect of childhood misfortune on later life health, whereas others find spousal support to be most health protective. Furthermore, it may be of particular importance to assess the effect of social support from friends, especially among older adults as spouses and family become less available due to illness and death. Relatedly, difference in the effects of domain specific strain may be responsible for the null findings in this dissertation. It is possible that while strain from one's partner may *reduce* frailty, strain from children may *exacerbate* frailty (or vice-versa) and averaging these effects is masquerading as a null finding. Domain specific findings are important to inform intervention studies and should be examined further.

In addition to social environment, future studies of frailty should examine how the physical environment influences frailty trajectories over time. The effects of many aspects of the

physical environment, such as pollution and crime, on later life health are clear. Yet, what remains to be seen is how these ecological factors, usually measured by adult neighborhood characteristics, interact with lifestyle factors, ascribed statuses, and particularly early life conditions to influence frailty.

Third, though this research finds few predictors of frailty growth over time, future studies should examine the extent to which frailty is reversible and what factors aid in its reduction over time. Variations in frailty measurement make this question a complex endeavor, but some research using phenotypic frailty seem to suggest that early-stage frailty, what some call "pre-frailty", may be reversible but unlikely for those with three or more indicators (Gill et al., 2006; Lee, Auyeung, et al., 2014). Like the findings of this dissertation regarding the positive benefits of wealth and education, Lee and colleagues (2014) found that higher cognitive function and SES are associated with improved frailty status. Though illuminating, more research is needed on the potential benefits of other adult resources.

Relatedly, a future direction for researchers would be to identify which indicators of the frailty phenotype are most consequential to frailty as a latent concept. Like many similar health measurement scales, it is likely that unintentional weight loss, exhaustion, weakness, slowness, and low energy expenditure do not equally "load" onto frailty as a concept. It is also likely that those loading factors would vary based on frailty progression (robust vs prefrail vs frail). Intervention studies aiming to reduce frailty growth over time, or even reverse frailty, would benefit from this knowledge; it would be easier to design an intervention which addresses one indicator of frailty than one that meant to address five.

Finally, Fried and colleagues' phenotypic frailty is one of the most commonly used measurement tools used to assess frailty and makes for easier cross-study comparison. However,

fully addressing the etiology of frailty is impeded at the start by a lack of standardized conceptualization and operationalization of this physical state. Though once synonymous with disability, comorbidity, and simply old age, frailty is now defined as a distinct biologic syndrome; yet, the markers of this syndrome are still debated. A future study comparing the effects of childhood exposures across multiple measurement tools such as the Deficit Accumulation Index (Rookwood et al., 2005) and FRAIL scale (van Kan et al., 2008), in addition to phenotypic frailty (Fried et al., 2001), would help to advance our understanding of the true impact of early disadvantage on frailty.

Frailty among older adults presents a significant public health problem now and for the foreseeable future. This condition is a notable indication of senescence in hyperdrive, signaling a rapid decline towards disability and ultimately mortality. This dissertation has highlighted a number of predictors of frailty across the life course and has subsequently identified areas for potential interventions—particularly those aimed at providing equal access to higher education and quality social relationships over the life course. Most importantly, this dissertation has demonstrated that frailty prevention should not be a task delegated exclusively to older adults. Effective prevention of this often devastating and costly syndrome should indeed begin early in life.

Table 4.1. Future Directions for Research on the Life Course Origins of Later Life Frailty

Type	Topic Area	Research Questions
Methodological	Fried Phenotype	Which of the frailty phenotype indicators are most consequential to frailty as a latent concept? Are the results the same when predicting frailty versus pre-frailty?
		Are subjective measures of slowness and weakness as effective as objective measures in creating a measure of phenotypic frailty (how can we measure frailty among adults <65 years of age in the HRS)?
	Alt. Frailty Measures	Which measurement of frailty (phenotypic, frailty index, etc.) is most predictive of mortality among older adults?
	Childhood Exposures	When childhood exposures are considered as a latent concept, which indicators are most predictive of later life frailty?
Substantive	General Frailty	Is frailty reversible? If so, what social and environmental factors help to reverse frailty?
	Social Support	What sources of social support (spouses, children, family, or friends) are most effective in preventing frailty or reducing its growth over time?
		Do the mediating effects of social support on the relationship between childhood exposures and later life frailty vary by gender?
		Do the mediating effects of social support on the relationship between childhood exposures and later life frailty vary by race?
		Is there a buffering effect of social support on childhood stressors and frailty (i.e. is the effect of social support stronger for those who experienced more misfortune during childhood)?
		Does social network embeddedness reduce the growth of frailty over time?
	Additional Factors	How does physical environment (i.e. neighborhood factors) mediate or moderate the relationship between childhood exposures and frailty?
		Which measures of neighborhood environment most associated with frailty trajectories: *subjective* or *objective*?

APPENDIX A. CHAPTER 2 SUPPLEMENTAL ANALYSES

Table A.1. Extended Descriptive Statistics at Baseline (N=6,805)

Variable	Range	Percent	Mean(SE)
Frailty (0-5, W1)	0-5		1.04 (1.00)
Robust (=0)		*33.93%*	
Pre-Frail (=1-2)		*56.94%*	
Frail (≥3)		*9.13%*	
Frailty Indicators			
Weight Loss	*0,1*	*6.75%*	
Exhaustion	*0,1*	*28.86%*	
Low Energy Expenditure	*0,1*	*21.09%*	
Slowness	*0,1*	*21.21%*	
Weakness	*0,1*	*26.45%*	
Childhood Experiences			
1+ Socioeconomic	0,1		
1+ Risky Parental Behavior	0,1		
1+ Infectious Disease	0,1		
1+ Chronic Disease	0,1		
1+ Impairments	0,1		
1+ Risky Adolescent Behavior	0,1		
Childhood Experiences			
Socioeconomic	*0-2*		*1.13 (0.81)*
Risky Parental behavior	*0-2*		*0.80 (0.65)*
Infectious Disease	*0-2*		*1.73 (0.59)*
Chronic Disease	*0-2*		*0.40 (0.67)*
Impairments	*0-2*		*0.19 (0.44)*
Risky Adolescent behavior	*0-2*		*0.07 (0.28)*
Demographics			
Age (years at baseline)	65-100		74.49 (6.89)
Female	0,1	55.77%	
Race/ethnicity			
White (ref)	0,1	83.06%	
Black	0,1	10.30%	
Hispanic	0,1	6.64%	
Adult Resources			
Education (in years)	0-17		12.57 (2.99)
Wealth (cube root in $10,000s)	-4.25-15.64		3.06 (1.74)
Private Insurance	0,1	56.93%	
Insurance			
None (ref)	*0,1*	*0.51%*	
Medicaid	*0,1*	*5.41%*	
Medicare	*0,1*	*37.30%*	
Medigap/Private	*0,1*	*56.78%*	

Table A.1. Continued.

Adult Risks			
Smoker	0,1	9.35%	
Heavy drinker	0,1	5.30%	
Body Mass Index	9.6-66.1		26.91 (4.86)
Morbidity	0-7		2.28 (1.29)

Note: Statistics presented in italics are alternate forms of variables considered or are presented to provide additional information; these alternate variables were not used in analyses.

Table A.2. Ordered Logistic Regression of Frailty[a] During 2006 or 2008 on Predictors

	Model 1	Model 2	Model 3
	OR (SE)	OR (SE)	OR (SE)
Childhood Exposure			
1 SES	1.037 (0.076)	1.030 (0.077)	0.978 (0.074)
2+ SES	1.229 (0.089)**	1.180 (0.087)*	1.070 (0.083)
1 Infectious	0.620 (0.093)**	0.634 (0.098)**	0.664 (0.103)**
2+ Infectious	0.714 (0.093)*	0.715 (0.095)*	0.772 (0.104)*
1 Chronic	1.092 (0.081)	1.047 (0.079)	1.058 (0.080)
2+ Chronic	1.013 (0.098)	0.927 (0.092)	0.924 (0.092)
1 Risky Parent B.[b]	0.972 (0.063)	0.967 (0.064)	0.968 (0.064)
2+ Risky Parent B.[b]	1.159 (0.122)	1.119 (0.111)	1.118 (0.111)
1 Risky Adolescent B.[b]	1.257 (0.159)	1.236 (0.159)	1.242 (0.162)
2+ Risky Adolescent B.[b]	2.005 (0.751)	2.016 (0.755)	1.412 (0.533)
1 Impairment	1.190 (0.099)*	1.167 (0.099)	1.195 (0.102)*
2+ Impairments	1.306 (0.273)	1.078 (0.236)	1.012 (0.225)
Demographics			
Age	1.030 (0.005)***	1.025 (0.005)***	1.025 (0.005)***
Female	1.346 (0.079)***	1.411 (0.085)***	1.338 (0.083)***
Black	1.169 (0.125)	1.136 (0.124)	0.897 (0.103)
Hispanic	1.200 (0.161)	1.250 (0.171)	0.928 (0.136)
Adult Resources			
Education			0.976 (0.012)*
Wealth			0.923 (0.018)***
Medicaid			1.692 (0.710)
Medicare			1.021 (0.400)
Medigap/Private			0.922 (0.360)
Adult Risks			
Smoker			1.170 (0.126)
Heavy Drinker			0.974 (0.127)
BMI			0.995 (0.007)
Morbidity		1.276 (0.031)***	1.254 (0.031)***

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

[a]Frailty: 0=robust, 1-2=pre-frail, 3+= frail

[b]B.=behavior

Table A.3. Linear Regression of Frailty[a] During 2006 or 2008 on Predictors (N=4,650)

	b (SE)
Childhood Exposure[b]	
Socioeconomic Misfortune	-0.001 (0.031)
Infectious Disease	-0.120 (0.059)*
Chronic Disease	0.017 (0.030)
Risky Parental Behavior	0.015 (0.029)
Risky Adolescent Behavior	0.124 (0.055)*
Impairment	0.076 (0.037)*
Demographics	
Age	0.016 (0.002)***
Female	0.134 (0.028)***
Black	0.018 (0.050)
Hispanic	0.043 (0.064)
Adult Resources	
Education	-0.019 (0.005)**
Wealth	-0.044 (0.009)***
Private Insurance	-0.073 (0.028)**
Adult Risks	
Smoker	0.105 (0.048)*
Drinks per Week	-0.011 (0.003)**
Drinks per Week^2	0.000 (0.000)*
BMI	-0.119 (0.020)***
BMI^2	0.002 (0.000)***
Morbidity	0.128 (0.011)***
Constant	1.631 (0.361)***

[a]Frailty= 0-5 indicators

[b]Each childhood exposure is measured as one or more indicators within each domain.

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

Table A.4. Linear Regression of Frailty[a] During 2006 or 2008 on Predictors (N=6,805)

	Model 1	Model 2	Model 3
	b (SE)	b (SE)	b (SE)
Childhood Exposure[b]			
Socioeconomic Misfortune	0.076 (0.028)**	0.051 (0.028)	-0.007 (0.028)
Infectious Disease	-0.170 (0.049)***	-0.168 (0.047)***	-0.137 (0.047)**
Chronic Disease	0.081 (0.027)**	0.040 (0.026)	0.045 (0.025)
Risky Parental Behavior	0.013 (0.027)	-0.001 (0.026)	0.002 (0.026)
Risky Adolescent Behavior	0.199 (0.055)***	0.161 (0.054)**	0.140 (0.053)**
Impairment	0.119 (0.033)***	0.090 (0.032)**	0.083 (0.032)**
Demographics			
Age	0.025 (0.002)***	0.020 (0.002)***	0.021 (0.002)***
Female	0.168 (0.024)***	0.190 (0.024)***	0.158 (0.024)***
Black	0.192 (0.040)***	0.156 (0.039)***	0.039 (0.040)
Hispanic	0.174 (0.060)***	0.190 (0.049)***	0.021 (0.051)
Adult Resources			
Education			-0.019 (0.005)***
Wealth			-0.052 (0.008)***
Private Insurance			-0.079 (0.024)**
Adult Risks			
Smoker			0.184 (0.041)***
Heavy Drinker			-0.052 (0.052)
BMI			0.002 (0.003)
Morbidity		0.161 (0.009)***	0.145 (0.010)***
Constant	-0.901 (0.146)***	-0.619 (0.142)***	-0.458 (0.190)*

[a]Frailty= 0-5 indicators

[b]Each childhood exposure is measured as one or more indicators within each domain.

Note: Analyses conducted using SEM with FIML option.

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

Table A.5. Mediational Results for Each Childhood Exposure Domain (CED) and Frailty during 2006 or 2008 (N=6,803).

Pathway	Socioeconomic b(SE)[a]	Infectious Disease b(SE)[a]	Chronic Disease b(SE)[a]
CED—Frailty	-0.013 (0.028)	-0.160 (0.045)***	0.042 (0.026)
Total Indirect Effect	0.086 (0.010)***	-0.115 (0.017)***	0.030 (0.007)***
Total Effect	0.073 (0.028)*	-0.275 (0.045)***	0.072 (0.027)**
CED—Education	-1.485 (0.086)***	2.509 (0.136)***	0.375 (0.081)***
Education—Frailty	-0.023 (0.004)***	-0.023 (0.004)***	-0.023 (0.004)***
Education—Morbidity	-0.024 (0.006)***	-0.024 (0.006)***	-0.024 (0.006)***
Indirect Effect	0.039 (0.007)***	-0.066 (0.011)***	-0.010 (0.003)***
CED—Wealth	-0.486 (0.047)***	0.823 (0.076)***	-0.011 (0.045)
Wealth—Frailty	-0.053 (0.008)***	-0.053 (0.008)***	-0.053 (0.008)***
Wealth—Morbidity	-0.106 (0.010)***	-0.106 (0.010)***	-0.106 (0.010)***
Indirect Effect	0.033 (0.005)***	-0.056 (0.008)***	-0.001 (0.003)
CED—Private Ins.	-0.026 (0.014)	0.125 (0.022)***	-0.013 (0.013)
Private Ins.—Frailty	-0.081 (0.024)**	-0.081 (0.024)**	-0.081 (0.024)**
Private Ins.—Morbidity	-0.030 (0.032)	-0.030 (0.032)	-0.030 (0.032)
Indirect Effect	0.002 (0.001)	-0.011 (0.004)**	0.001 (0.001)
CED—Smoker	0.010 (0.008)	0.047 (0.013)***	-0.001 (0.008)
Smoker—Frailty	0.161 (0.040)***	0.161 (0.040)***	0.161 (0.040)***
Smoker—Morbidity	-0.041 (0.053)	-0.041 (0.053)	-0.041 (0.053)
Indirect Effect	0.002 (0.001)	0.007 (0.003)*	-0.000 (0.001)
CED—Drinker	-0.030 (0.006)***	0.026 (0.010)*	0.001 (0.006)
Drinker—Frailty	-0.057 (0.052)	-0.057 (0.052)	-0.057 (0.052)
Drinker—Morbidity	0.013 (0.068)	0.013 (0.068)	0.013 (0.068)
Indirect Effect	0.002 (0.002)	-0.001 (0.001)	-0.000 (0.000)
CED—BMI	0.808 (0.161)***	4.453 (0.253)***	0.405 (0.152)**
BMI—Frailty	-0.001 (0.002)	-0.001 (0.002)	-0.001 (0.002)
BMI—Morbidity	0.041 (0.003)***	0.041 (0.003)***	0.041 (0.003)***
Indirect Effect	0.004 (0.002)*	0.023 (0.010)*	0.002 (0.001)
CED—Morbidity	0.028 (0.037)	-0.076 (0.060)	0.244 (0.034)***
Morbidity—Frailty	0.147 (0.009)***	0.147 (0.009)***	0.147 (0.009)***
Indirect Effect	0.004 (0.005)	-0.011 (0.009)	0.036 (0.006)***

(Continued on next page.)

Table A.5. Continued.

	Risky Parent	Risky Adolescent	Impairments
	b(SE)[a]	b(SE)[a]	b(SE)[a]
CED—Frailty	-0.018 (0.025)	0.133 (0.053)*	0.080 (0.032)*
Total Indirect Effect	-0.020 (0.008)*	0.051 (0.014)***	0.029 (0.008)***
Total Effect	-0.038 (0.026)	0.183 (0.055)**	0.109 (0.033)**
CED—Education	0.654 (0.078)***	-0.027 (0.166)	0.080 (0.100)
Education—Frailty	-0.023 (0.004)***	-0.023 (0.004)***	-0.023 (0.004)***
Education—Morbidity	-0.024 (0.006)***	-0.024 (0.006)***	-0.024 (0.006)***
Indirect Effect	-0.017 (0.003)***	0.001 (0.004)	-0.001(0.004)
CED—Wealth	0.301 (0.044)***	-0.191 (0.092)*	-0.074 (0.055)
Wealth—Frailty	-0.053 (0.008)***	-0.053 (0.008)***	-0.053 (0.008)***
Wealth—Morbidity	-0.106 (0.010)***	-0.106 (0.010)***	-0.106 (0.010)***
Indirect Effect	-0.021 (0.004)***	0.013 (0.006)*	0.013 (0.006)*
CED—Private Ins.	0.052 (0.013)***	-0.051 (0.026)	-0.033 (0.016)*
Private Ins.—Frailty	-0.081 (0.024)**	-0.081 (0.024)**	-0.081 (0.024)**
Private Ins.—Morbidity	-0.030 (0.032)	-0.030 (0.032)	-0.030 (0.032)
Indirect Effect	-0.004 (.002)**	0.004 (0.003)	0.004 (0.003)
CED—Smoker	0.042 (0.008)***	-0.005 (0.016)	-0.009 (0.010)
Smoker—Frailty	0.161 (0.040)***	0.161 (0.040)***	0.161 (0.040)***
Smoker—Morbidity	-0.041 (0.053)	-0.041 (0.053)	-0.041 (0.053)
Indirect Effect	0.007 (0.002)**	-0.001 (0.003)	-0.001 (0.003)
CED—Drinker	0.032 (0.006)***	0.000 (0.012)	-0.002 (0.007)
Drinker—Frailty	-0.057 (0.052)	-0.057 (0.052)	-0.057 (0.052)
Drinker—Morbidity	0.013 (0.068)	0.013 (0.068)	0.013 (0.068)
Indirect Effect	-0.002 (0.002)	-0.000 (0.001)	0.000 (0.001)
CED—BMI	1.615 (0.145)***	0.949 (0.311)**	0.691 (0.186)***
BMI—Frailty	-0.001 (0.002)	-0.001 (0.002)	-0.001 (0.002)
BMI—Morbidity	0.041 (0.003)***	0.041 (0.003)***	0.041 (0.003)***
Indirect Effect	0.008 (0.004)*	0.005 (0.003)	0.005 (0.003)
CED—Morbidity	0.061 (0.033)	0.181 (0.068)**	0.144 (0.042)**
Morbidity—Frailty	0.147 (0.009)***	0.147 (0.009)***	0.147 (0.009)***
Indirect Effect	0.009 (0.005)	0.027 (0.010)**	0.027 (0.010)**

[a]Unstandardized coefficient (standard error)
Note: Model controls for age, gender, and race. Analyses conducted using SEM with FIML option; gray highlights indicate a difference in significance level or large change in effect size compared to list-wise deletion models.
***p<.001, **p<.01, *p<.05

APPENDIX B. CHAPTER 3 SUPPLEMENTAL ANALYSES

Table B.1. Growth Curve Model Estimates of the Effects of Accumulated Childhood Exposures and Adult Social Relationships on Frailty (N=7,712)

	Model 1		Model 2	
	Intercept	Slope	Intercept	Slope
Accumulated CE	0.041 (0.007)***	-0.004 (0.005)	0.023 (0.007)**	-0.004 (0.005)
Demographics				
Age	0.011 (0.000)***	0.003 (0.000)***	0.007 (0.000)***	0.002 (0.000)***
Female	0.151 (0.023)***	-0.038 (0.018)*	0.167 (0.022)***	-0.040 (0.017)*
Black	0.160 (0.037)***	0.012 (0.028)	0.128 (0.037)***	0.004 (0.029)
Hispanic	0.155 (0.046)**	0.095 (0.035)**	0.176 (0.045)***	0.090 (0.035)**
Adult Resources				
Education				
Wealth				
Private Insurance				
Adult Risks				
Morbidity			0.157 (0.009)***	0.013 (0.007)
Smoker				
Heavy Drinker				
BMI				
Social Relationships				
Social Support				
Social Strain				
No. Social Roles				
Model Fit Index	CFI= 0.896; RMSEA= 0.059		CFI= 0.910; RMSEA= 0.057	

	Model 3		Model 4	
	Intercept	Slope	Intercept	Slope
Accumulated CE	0.025 (0.007)**	-0.004 (0.005)	.033(0.007)***	-0.004 (0.005)
Demographics				
Age	0.014 (0.001)***	0.004 (0.001)***	0.018 (0.001)***	0.006 (0.001)***
Female	0.194 (0.023)***	-0.036 (0.018)*	0.159 (0.023)***	-0.045 (0.018)*
Black	0.154 (0.036)***	0.010 (0.028)	0.129 (0.036)***	0.000 (0.028)
Hispanic	0.193 (0.045)***	0.092 (0.035)**	0.191 (0.045)***	0.093 (0.034)
Adult Resources				
Education				
Wealth				
Private Insurance				
Adult Risks				
Morbidity	0.153 (0.009)***	0.012 (0.007)	0.150 (0.009)***	0.012 (0.007)
Smoker				
Heavy Drinker				
BMI				
Social Relationships				
Social Support	-0.182 (0.021)***	-0.026 (0.016)	-0.123 (0.022)	-0.018 (0.017)

Table B.1. Continued.

Social Strain	0.005 (0.013)	-0.011 (0.010)	0.008 (0.013)	-0.009 (0.010)
No. Social Roles			-0.113 (0.015)***	-0.044 (0.012)***
Model Fit Index	CFI= 0.947; RMSEA= 0.040		CFI= 0.981; RMSEA= 0.023	

	Model 5		Model 6	
	Intercept	Slope	Intercept	Slope
Accumulated CE	0.018 (0.007)*	-0.007 (0.005)	0.023 (0.007)**	-0.007 (0.005)
Demographics				
Age	0.019 (0.001)***	0.005 (0.001)***	0.020 (0.001)***	0.007 (0.001)***
Female	0.160 (0.022)***	-0.044 (0.018)*	0.143 (0.023)***	-0.051 (0.018)**
Black	0.031 (0.038)	-0.015 (0.029)	0.026 (0.038)	-0.020 (0.029)
Hispanic	0.007 (0.047)	0.036 (0.037)	0.028 (0.047)	0.043 (0.037)
Adult Resources				
Education	-0.021 (0.004)***	-0.013 (0.003)***	-0.017 (0.004)***	-0.012 (0.003)***
Wealth	-0.055 (0.007)***	-0.011 (0.005)	-0.045 (0.007)***	-0.008 (0.006)
Private Insurance	-0.077 (0.023)**	0.015 (0.018)	-0.069 (0.023)**	0.017 (0.018)
Adult Risks				
Morbidity	0.139 (0.009)***	0.007 (0.007)	0.136 (0.009)***	0.006 (0.007)
Smoker	0.154 (0.039)***	0.080 (0.031)*	0.162 (0.038)***	0.082 (0.031)**
Heavy Drinker	-0.074 (0.049)	0.081 (0.038)*	-0.070 (0.049)	0.082 (0.038)*
BMI	0.002 (0.002)	0.002 (0.002)	0.005 (0.002)*	0.002 (0.002)
Social Relationships				
Social Support	-0.131 (0.022)***	-0.022 (0.017)	-0.109 (0.022)***	-0.017 (0.017)
Social Strain	0.005 (0.013)	-0.008 (0.010)	0.007 (0.013)	-0.008 (0.010)
No. Social Roles			-0.092 (0.017)***	-0.032 (0.013)*
Model Fit Index	CFI= 0.967; RMSEA= 0.027		CFI= 0.980; RMSEA= 0.021	

Note: Frailty is measured on a scale of 0-5 indicators. Accumulated Childhood Exposures is measured on a scale of 0-27 exposures. Unstandardized coefficients are presented with standard errors in parentheses. CE= Childhood Exposures; Comp.= composition.
***p<.001, **p<.01, *p<.05

Table B.2. Social Relationship Mediation Results from Growth Curve Model Estimates of the Effects of Accumulated Childhood Exposures on Frailty (N=7,712).

	Intercept		Slope	
	b	SE	b	SE
Accumulated CE—Frailty	0.023**	0.007	-0.007	0.005
Total Indirect Effect	0.018***	0.002	0.002	0.001
Total Effect	0.041***	0.007	-0.005	0.005
Accumulated CE—Support	-0.028***	0.004	-	-
Support—Morbidity	-0.097**	0.029	-	-
Support—Frailty	-0.109***	0.022	-0.016	0.017
Indirect Effect	0.003***	0.001	0.000	0.000
Accumulated CE—Strain	-0.008	0.007	-	-
Strain —Morbidity	0.103	0.016	-	-
Strain —Frailty	0.007	0.013	-0.008	0.010
Indirect Effect	-0.000	0.000	0.000	0.000
Accumulated CE—Roles	-0.014**	0.005	-	-
Roles— Morbidity	-0.018	0.023	-	-
Roles— Frailty	-0.088***	0.017	-0.034**	0.013
Indirect Effect	0.001*	0.001	0.000	0.000
Accumulated CE—Morbidity	0.099***	0.009	-	-
Morbidity—Frailty	0.136***	0.009	-	-
Indirect Effect	0.013***	0.002	0.001	0.001
Model Fit Index	CFI= 0.853; RMSEA= 0.052			

Note: Frailty is measured on a scale of 0-5 indicators. Accumulated childhood exposure is measured on a scale of 0-27 indicators. Model controls for demographic characteristics and adult risks and resources. Each dash (-) in first column refers to a relationship between accumulated childhood exposures and an endogenous outcome or an indirect pathway between the accumulated CE and the outcome; indented rows involve one or more indirect effects. CE= childhood exposure; CFI= comparative fit index; RMSEA= root mean square error of approximation.***p<.001, **p<.01, *p<.05

Table B.3. Mediation Results from Growth Curve Model Estimates for Each Childhood Exposure Domain (CED) and Frailty Over Time (N=7,712).

	Socioeconomic Misfortunes		Infectious Diseases	
	Intercept	Slope	Intercept	Slope
CED—Frailty	-0.012 (0.027)	-0.023 (0.021)	-0.169 (0.044)***	-0.051 (0.036)
Total Indirect Effect	0.097 (0.009)***	0.027 (0.006)***	-0.029 (0.011)*	-0.010 (0.003)**
Total Effect	0.085 (0.027)**	0.004 (0.020)	-0.198 (0.045)***	-0.061 (0.036)
CED—Education	-1.623 (0.072)***	-	0.500 (0.127)***	-
Education —Morbidity	-0.012 (0.006)*	-	-0.012 (0.006)*	-
Education —Frailty	-0.024 (0.004)***	-0.013 (0.003)***	-0.024 (0.004)***	-0.013 (0.003)***
Indirect Effect	0.041 (0.007)***	0.022 (0.005)***	-0.013 (0.004)**	-0.007 (0.002)**
CED—Wealth	-0.524 (0.044)***	-	0.247 (0.077)**	-
Wealth —Morbidity	-0.094 (0.009)***	-	-0.094 (0.009)***	-
Wealth —Frailty	-0.057 (0.007)***	-0.012 (0.006)*	-0.057 (0.007)***	-0.012 (0.006)*
Indirect Effect	0.037 (0.005)***	0.007 (0.003)*	-0.017 (0.006)**	-0.003 (0.002)
CED—Private Ins.	-0.043 (0.013)**	-	0.042 (0.023)	-
Private Ins.— Morbidity	-0.012 (0.030)	-	-0.012 (0.030)	-
Private Ins.— Frailty	-0.077 (0.023)**	0.016 (0.018)	-0.077 (0.023)**	0.016 (0.018)
Indirect Effect	0.003 (0.001)*	-0.001 (0.001)	-0.003 (0.002)	-0.001 (0.001)
CED—Smoker	0.010 (0.008)	-	-0.021 (0.014)	-
Smoker—Morbidity	0.037 (0.051)	-	0.037 (0.051)	-
Smoker—Frailty	0.152 (0.038)***	0.081 (0.031)**	0.152 (0.038)***	0.081 (0.031)**
Indirect Effect	0.002 (0.001)	0.001 (0.002)	-0.003 (0.002)	-0.002 (0.001)
CED—Drinker	-0.029 (0.006)***	-	0.001 (0.010)	-
Drinker—Morbidity	0.031 (0.064)	-	0.031 (0.064)	-
Drinker—Frailty	-0.072 (0.049)	0.082 (0.038)*	-0.072 (0.049)	0.082 (0.038)*
Indirect Effect	0.002 (0.002)	-0.002 (0.001)*	-0.000 (0.001)	-0.000 (0.001)
CED—BMI	0.630 (0.139)***	-	0.211 (0.242)	-
BMI—Morbidity	0.051 (0.003)***	-	0.051 (0.003)***	-
BMI—Frailty	0.001 (0.002)	0.002 (0.002)	0.001 (0.002)	0.002 (0.002)
Indirect Effect	0.005 (0.002)**	0.001 (0.001)	0.002 (0.002)	0.000 (0.001)
CED—Morbidity	0.042 (0.035)	-	0.044 (0.059)	-
Morbidity—Frailty	0.141 (0.009)***	0.007 (0.007)	0.141 (0.009)***	0.007 (0.007)
Indirect Effect	0.006 (0.005)	0.000 (0.000)	0.006 (0.008)	0.000 (0.001)

	Chronic Diseases		Risky Parental Behaviors	
	Intercept	Slope	Intercept	Slope
CED—Frailty	0.049 (0.025)*	0.004 (0.019)	-0.018 (0.024)	-0.015 (0.019)
Total Indirect Effect	0.033 (0.006)***	-0.001 (0.002)	0.012 (0.006)	0.004 (0.002)
Total Effect	0.083 (0.025)**	0.003 (0.019)	-0.007 (0.025)	-0.011 (0.019)
CED—Education	0.170 (0.069)*	-	-0.043 (0.068)	-

Table B.3. Continued.

Education —Morbidity	-0.012 (0.006)*	-	-0.012 (0.006)*	-
Education —Frailty	-0.024 (0.004)***	-0.013 (0.003)***	-0.024 (0.004)***	-0.013 (0.003)***
Indirect Effect	-0.004 (0.002)*	-0.002 (0.001)*	0.001 (0.002)	0.001 (0.001)
CED—Wealth	-0.031 (0.042)	-	0.092 (0.042)*	-
Wealth —Morbidity	-0.094 (0.009)***	-	-0.094 (0.009)***	-
Wealth —Frailty	-0.057 (0.007)***	-0.012 (0.006)*	-0.057 (0.007)***	-0.012 (0.006)*
Indirect Effect	0.002 (0.003)	0.000 (0.001)	-0.006 (0.003)*	-0.001 (0.001)
CED—Private Ins.	-0.021 (0.012)	-	0.013 (0.012)	-
Private Ins.— Morbidity	-0.012 (0.030)	-	-0.012 (0.030)	-
Private Ins.— Frailty	-0.077 (0.023)**	0.016 (0.018)	-0.077 (0.023)**	0.016 (0.018)
Indirect Effect	0.002 (0.001)	-0.000 (0.000)	-0.001 (0.002)	0.000 (0.000)
CED—Smoker	-0.008 (0.007)	-	0.020 (0.007)**	-
Smoker—Morbidity	0.037 (0.051)	-	0.037 (0.051)	-
Smoker—Frailty	0.152 (0.038)***	0.081 (0.031)**	0.152 (0.038)***	0.081 (0.031)**
Indirect Effect	-0.001 (0.001)	-0.001 (0.001)	0.003 (0.001)*	0.001 (0.001)
CED—Drinker	0.000 (0.006)	-	0.020 (0.006)***	-
Drinker—Morbidity	0.031 (0.064)	-	0.031 (0.064)	-
Drinker—Frailty	-0.072 (0.049)	0.082 (0.038)*	-0.072 (0.049)	0.082 (0.038)*
Indirect Effect	-0.000 (0.000)	0.000 (0.000)	-0.001 (0.001)	0.002 (0.001)
CED—BMI	-0.041 (0.132)	-	0.104 (0.130)	-
BMI—Morbidity	0.051 (0.003)***	-	0.051 (0.003)***	-
BMI—Frailty	0.001 (0.002)	0.002 (0.002)	0.001 (0.002)	0.002 (0.002)
Indirect Effect	-0.000 (0.001)	-0.000 (0.000)	0.001 (0.001)	0.000 (0.000)
CED—Morbidity	0.263 (0.032)***		0.111 (0.031)***	-
Morbidity—Frailty	0.141 (0.009)***	0.007 (0.007)	0.141 (0.009)***	0.007 (0.007)
Indirect Effect	0.036 (0.005)***	0.002 (0.002)	0.016 (0.005)**	0.001 (0.001)

	Risky Adolescent Behaviors		Impairments	
	Intercept	Slope	Intercept	Slope
CED—Frailty	0.139 (0.050)**	-0.057 (0.037)	0.082 (0.030)**	-0.013 (0.024)
Total Indirect Effect	0.062 (0.013)***	0.008 (0.004)*	0.040 (0.008)***	0.003 (0.002)
Total Effect	0.201 (0.052)***	-0.049 (0.037)	0.122 (0.031)***	-0.010 (0.024)
CED—Education	-0.307 (0.121)*	-	-0.086 (0.085)	-
Education —Morbidity	-0.012 (0.006)*	-	-0.012 (0.006)*	-
Education —Frailty	-0.024 (0.004)***	-0.013 (0.003)***	-0.024 (0.004)***	-0.013 (0.003)***
Indirect Effect	0.008 (0.004)*	0.004 (0.002)	0.002 (0.002)	0.001 (0.001)
CED—Wealth	-0.276 (0.085)**	-	-0.164 (0.051)**	-
Wealth —Morbidity	-0.094 (0.009)***	-	-0.094 (0.009)***	-
Wealth —Frailty	-0.057 (0.007)***	-0.012 (0.006)*	-0.057 (0.007)***	-0.012 (0.006)*
Indirect Effect	0.019 (0.006)**	0.003 (0.002)	0.012 (0.004)**	0.002 (0.002)
CED—Private Ins.	0.013 (0.012)	-	-0.041 (0.015)	-

Private Ins.— Morbidity	-0.012 (0.030)	-	-0.012 (0.030)	-
Private Ins.— Frailty	-0.077 (0.023)**	0.016 (0.018)	-0.077 (0.023)**	0.016 (0.018)
Indirect Effect	0.005 (0.002)	-0.001 (0.001)	0.003 (0.002)*	-0.001 (0.001)
CED—Smoker	-0.006 (0.015)	-	-0.009 (0.009)	-
Smoker—Morbidity	0.037 (0.051)	-	0.037 (0.051)	-
Smoker—Frailty	0.152 (0.038)***	0.081 (0.031)**	0.152 (0.038)***	0.081 (0.031)**
Indirect Effect	-0.001 (0.002)	-0.000 (0.001)	-0.001 (0.001)	-0.001 (0.001)
CED—Drinker	0.004 (0.011)	-	-0.005 (0.007)	-
Drinker—Morbidity	0.031 (0.064)	-	0.031 (0.064)	-
Drinker—Frailty	-0.072 (0.049)	0.082 (0.038)*	-0.072 (0.049)	0.082 (0.038)*
Indirect Effect	-0.000 (0.001)	0.000 (0.001)	0.000 (0.001)	-0.000 (0.001)
CED—BMI	0.127 (0.265)	-	0.240 (0.162)	-
BMI—Morbidity	0.051 (0.003)***	-	0.051 (0.003)***	-
BMI—Frailty	0.001 (0.002)	0.002 (0.002)	0.001 (0.002)	0.002 (0.002)
Indirect Effect	0.001 (0.002)	0.000 (0.001)	0.002 (0.001)	0.001 (0.001)
CED—Morbidity	0.216 (0.064)**	-	0.159 (0.039)***	-
Morbidity—Frailty	0.141 (0.009)***	0.007 (0.007)	0.141 (0.009)***	0.007 (0.007)
Indirect Effect	0.030 (0.009)**	0.001 (0.002)	0.022 (0.006)***	0.001 (0.001)

Note: Frailty is measured on a scale of 0-5 indicators. Each childhood exposure is measured as one or more indicators within each domain. Model adjusts for demographic characteristics. Unstandardized coefficients are presented; standard errors are in parentheses. Each dash (-) in first column refers to a relationship between a Childhood Exposure Domain and an endogenous outcome or an indirect pathway between the CED and the outcome; indented rows involve one or more indirect effects. Comparative fit index (CFI)=0.830; Root mean square error of approximation (RMSEA)=0.077. CED= childhood exposure domain. ***p<.001, **p<.01, *p<.05

Table B.4. Probit Regression of Mortality from 2006/2008- 2014/2016 on Predictors (N=1,358)

	Coef.	SE
Childhood Exposure		
Socioeconomic Misfortune	0.010	0.061
Infectious Disease	-0.240*	0.107
Chronic Disease	-0.011	0.058
Risky Parental Behavior	-0.031	0.057
Risky Adolescent Behavior	0.008	0.110
Impairment	-0.050	0.072
Demographics		
Age	0.067***	0.004
Female	-0.350***	0.055
Black	-0.038	0.010
Hispanic	-0.076	0.132
Adult Resources		
Education	-0.012	0.010
Wealth	-0.034	0.018
Private Insurance	-0.016	0.054
Adult Risks		
Smoker	0.454***	0.088
Heavy Drinker	0.009	0.119
BMI	0.003	0.003
Morbidity	0.190***	0.021
Social Relationships		
Social Support	-0.112*	0.052
Social Strain	-0.046	0.029
Number of Social Roles	-0.016	0.040
Constant	-5.409***	0.495

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

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Williams, M. M., Kemp, B. R., Ferraro, K. F., & Mustillo, S. A. (2019). Avoiding the major causes of death: does childhood misfortune reduce the likelihood of being disease free in later life? *Journal of Gerontology: Social Sciences*, *74*(1), 170-180. https://doi.org/10.1093/geronb/gby039.

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Xue, Q. L. (2011). The frailty syndrome: Definition and natural history. *Clinics in Geriatric Medicine*, *27*(1), 1-15. https://doi.org/10.1016/j.cger.2010.08.009.

Zaborenko, C. J., Ferraro, K. F., & Williams-Farrelly, M. M. (2020). Childhood misfortune and late-life stroke incidence, 2004–2014. *The Gerontologist*. Advance online publication. https://doi.org/10.1093/geront/gnaa007.

Zhang, Z., Liu, H., & Choi, S. W. (2020). Early-life socioeconomic status, adolescent cognitive ability, and cognition in late midlife: Evidence from the Wisconsin Longitudinal Study. *Social Science & Medicine, 244*, 112575. https://doi.org/10.1016/j.socscimed.2019.112575.

VITA

Monica M. Williams-Farrelly

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POSITION

Current	Ph.D. Candidate Sociology and Gerontology, Purdue University
September 2020	Postdoctoral Research Fellow, Survey Research Center, Institute for Social Research, University of Michigan

EDUCATION

2020	Ph.D. in Sociology and Gerontology- Purdue University *Dissertation Title*: Life Course Origins of Frailty in Later Life *Committee:* Kenneth F. Ferraro (Chair), Shawn Bauldry, Elizabeth Richards (Nursing), & Patricia Thomas
2015	M.A. in Sociology- Indiana University Purdue University Indianapolis *Medical Sociology (concentration)*
2011	B.A. Criminology and Chemistry- Butler University *High Departmental Honors, Criminology*

RESEARCH INTERESTS

- Health and Aging
- Early Origins of Health
- Health Disparities
- Social Networks and Relationships

TEACHING INTERESTS

- Medical Sociology
- Sociology of Aging
- Sexuality
- Social Gerontology
- Quantitative Methods
- Introduction to Statistics

PUBLICATIONS

Zaborenko, Callie, Ferraro, Kenneth F., and **Monica M. Williams-Farrelly**. "Childhood Misfortune and Late-life Stroke Incidence, 2004-2014." *The Gerontologist.* https://doi.org/10.1093/geront/gnaa007.

Williams, Monica M., Blakelee R. Kemp, Kenneth F. Ferraro, and Sarah A. Mustillo. 2019. "Avoiding the Major Causes of Death: Does Childhood Misfortune Reduce the Likelihood of Being Disease Free in Later Life?" *Journal of Gerontology: Social Sciences* 74(1): 170-180. https://doi.org/10.1093/geronb/gby039.

Ferraro, Kenneth F., Blakelee R. Kemp, and **Monica M. Williams**. 2017. "Diverse Aging and Health Inequality by Race and Ethnicity." *Innovation in Aging* 1(1): igx002, 1-11. https://doi.org/10.1093/geroni/igx002.

Latham, Kenzie and **Monica M. Williams**. 2015. "Does Neighborhood Disorder Predict Recovery from Mobility Limitation? Findings from the Health and Retirement Study." *Journal of Aging and Health* 27(8): 1415–1442. https://doi.org/10.1177/0898264315584328.

MANUSCRIPTS IN PROGRESS OR UNDER REVIEW

Thomas, Patricia A., **Williams-Farrelly, Monica M**., Ferraro, Kenneth F., and Sauerteig, Madison. "Childhood Stressors, Relationship Quality, and Cognitive Health in Later Life." In preparation, draft available

Sauerteig, Madison, Ferraro, Kenneth F., and **Monica M. Williams-Farrelly**. "Origins of Adult Waist Circumference: Childhood Misfortune and Adult Physical Activity." In preparation, draft available

Williams-Farrelly, Monica M. and Kenzie Latham-Mintus. "Neighborhood Disorder, Mobility Disability, and Meeting Up with Friends" In preparation, draft available

HONORS AND AWARDS

2019-2020	Purdue Research Foundation Research Grant, "Life Course Origins of Frailty in Later Life." College of Liberal Arts, Purdue University, $20,000
2019	Outstanding Graduate Student Paper Award, "Avoiding the Major Causes of Death: Does Childhood Misfortune Reduce the Likelihood of Being Disease Free in Later Life?" Department of Sociology, Purdue University
2019	Summer Research Training Grant, Department of Sociology, Purdue University, $2,000
2018	Promoting Research Opportunities to Maximize Innovation and Scholarly Excellence (PROMISE) Award, College of Liberal Arts Purdue University, $750
2017	Robert L. Eichhorn Fellowship Award in Medical Sociology, Department of

	Sociology, Purdue University, $5,000
2017	Promoting Research Opportunities to Maximize Innovation and Scholarly Excellence (PROMISE) Award, College of Liberal Arts, Purdue University, $750
2016	Summer Research Enhancement Award, Purdue University, $878
2015-2017	Frederick N. Andrews Fellowship, Purdue University, $19,000 yearly
2015	Suzanne K. Steinmetz Scholarship, Department of Sociology, Indiana University-Purdue University Indianapolis, $500

RESEARCH AND TEACHING EXPERIENCE

Research Assistant

2017-2020	Childhood, Social Support, and Cognition, Dr. Patricia Thomas, Purdue University
2015-2020	Childhood Misfortune and Adult Health, Dr. Kenneth Ferraro, Purdue University (NIA, # R01AG043544)
2013-2015	Neighborhoods and Mobility Limitation, Dr. Kenzie Latham, IUPUI
2010-2011	Homelessness in Indianapolis, Dr. Kenneth Colburn, Butler University

Instructor of Record (Full Responsibility)

Summer 2018	SOC 100, Introduction to Sociology, Purdue University
Fall 2018	SOC 100, Introduction to Sociology, Purdue University

Teaching Assistant

Spring 2016	SOC 328, Criminal Justice, Purdue University *Temporary Instructor, February 23-March 24, 2016* *Guest Lecturer, "Juvenile Justice," April 28, 2016*
Fall 2015	SOC 576, Health and Aging in a Social Context, Purdue University *Guest Lecturer, "Chronic Disease," September 10, 2015; "Ethical Issues in Public Health and Aging, November 10, 2015*
Spring 2015	SOC R-359, Introduction to Sociological Statistics, IUPUI
Spring 2014	SOC R-320, Human Sexuality, Instructor, IUPUI *Guest Lecturer, "Sex Dolls and Sexual Expression," March 2nd, 2014.*

PROFESSIONAL DEVELOPMENT

Paper Presentations

Williams-Farrelly, Monica M. and Kenneth Ferraro. "Life Course Origins of Frailty in Later Life." Presented November 13, 2019 at the Gerontological Society of America (GSA) Annual Scientific Meeting.

Sauerteig, Madison, Ferraro, Kenneth F., and **Monica M. Williams-Farrelly**. "Origins of Adult Waist Circumference: Childhood Misfortune and Adult Physical Activity." Presented

November 13, 2019 at the Gerontological Society of America (GSA) Annual Scientific Meeting.

Williams, Monica M., Patricia Thomas, and Kenneth Ferraro. "Does Social Support Mediate the Influence of Childhood Exposures on Cognition in Later Life?" Presented November 16, 2018 at the Gerontological Society of America (GSA) Annual Scientific Meeting.

Williams, Monica M., Blakelee Kemp, and Kenneth Ferraro. "Does Childhood Misfortune Increase the Likelihood of Mobility Limitation in Later Life?" Presented July 27, 2017 at the International Association of Gerontology and Geriatrics' 21st annual World Conference.

Zaborenko, Callie, **Williams, Monica M.**, and Kenneth F. Ferraro. "Childhood Misfortune and Late-life Stroke Incidence, 2004-2014." Presented November 17, 2016 at the Gerontological Society of America (GSA) Annual Scientific Meeting.

Williams, Monica M., Blakelee Kemp, and Kenneth Ferraro. "Escaping Disease: Does Childhood Disadvantage Reduce the Likelihood of Being Disease Free in Later Life?" Presented November 16, 2016 at The Gerontological Society of America (GSA) Annual Scientific Meeting.

Latham, Kenzie and **Monica M. Williams**. "Do Subjective Neighborhood Assessments Predict Recovery From Mobility Limitation? Findings from the Health and Retirement Study." Presented August 23, 2015 at the Society for the Study of Social Problems (SSSP) annual conference.

Williams, Monica M. "Social Support and Sexual Well-being" Presented April 17, 2015 at IUPUI's Center for Research and Learning's Annual Research Day.

Pedagogical and Methodological Training

- Certificate of Foundations in College Teaching, Center for Instructional Excellence, Purdue University (Summer 2019)
- Group-based Trajectory Modeling for the Medical and Social Sciences, ICPSR Summer Institute (Summer 2019)
- Examining the Health and Retirement Study (HRS) Workshop, ICPSR Summer Institute (Summer 2016)
- Seminar in Teaching Sociology, Purdue (Spring 2016)
- Graduate Teaching Assistant Training, Purdue (Fall 2015)
- Preparing Future Faculty and Professionals (PFFP) annual Pathways Conference, IUPUI (Fall 2014)
- SPSS: The Basics, IT Training, IUPUI (Summer 2014)
- NVivo Essentials, QSR International (Spring 2014)
- Educational Training for Teaching Associates (ETTA) Fall Conference, Center for Teaching and Learning, IUPUI (Fall 2013)
- How are the Children? Five Steps to Excellence in Child Welfare, Indiana Department of

Child Services (Fall 2012)

Professional Memberships

- American Sociological Association
  - Section on Aging and the Life Course
  - Section on Medical Sociology
- Gerontological Society of America

PROFESSIONAL SERVICE

2016-2017	Communications Officer, Sociology Graduate Organization, Purdue University
2014-2015	Co-Graduate Student Representative, IUPUI *"Curriculum Vitae Workshop" and "Applying to PhD Programs"*
2011-2015	Case Manager, New Hope of Indiana, Indianapolis, IN *Assist parents and children involved with the Indiana Department of Child Services in finding employment, stable housing, and health insurance*
Spring 2014	Panelist, School of Liberal Arts, Butler University *"Why Study Liberal Arts at Butler University?"*
2011-2012	Intern Volunteer Coordinator, Marion County Juvenile Detention Center, Indianapolis, IN *Juvenile Detention Alternatives Initiative (JDAI) grant; Organized "Career Options Fair," "Man Up!" and "Defeating Domestic; Head of Career Exploration volunteer group*