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## Gender and Health Over the Life Course: Temporal, Contextual, and Intersectional Considerations

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Graduate Program in Sociology

A thesis submitted in partial fulfillment of the requirements for the degree in Doctor of Philosophy

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## Abstract

Research has continuously demonstrated differences in health between men and women and emphasized a “gender paradox” whereby women live longer than men, but have higher rates of morbidity. Still, relatively little attention has been given to the underlying mechanisms and processes involved within groups of women and men that may provide greater insight into the patterns of health experienced among each group rather than simply between them. Specifically, there has been an over-reliance on cross-sectional and retrospective data; inattention to multiple resources and health conditions; limited consideration of various age ranges and time spans; and an over-emphasis on comparing women and men rather than what contributes to their respective health outcomes separately. This dissertation examines contributing factors to heterogeneity in the health of women and men, incorporating principles from the life course perspective and intersectionality theory. Each integrated chapter uses data from the U.S. Panel Study of Income Dynamics. Methods utilized include latent growth curve modelling, latent class analysis, discrete-time hazard models, and ordinary least squares and logistic regression. Findings contribute to the emerging body of literature that seeks to challenge traditional approaches to the conceptualization and measurement of gender and health through revealing the role of historical context, dynamic early life experiences, and intersecting dimensions of inequality across multiple health outcomes.

## Keywords

gender differences in health; PSID; life course; cumulative disadvantage; childhood economic hardship; self-rated health; chronic conditions; intersectionality; quantitative methods

## Co-Authorship Statement

Chapter 3 was co-authored by Dr. Andrea Willson (supervisor) and Dr. Kim Shuey. These individuals were involved in the conceptualization and creation of the latent class models used and consulted on manuscript development.

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# Chapter 1

## 1 Introduction

Extensive medical, epidemiological, and social science research has demonstrated differences in health between men and women and emphasized a “gender paradox” whereby women live longer than men, but have higher rates of morbidity (e.g. Macintyre, Hunt & Sweeting, 1996; Denton & Walter, 1999; Rieker & Bird, 2005; Case & Paxson, 2005; Bird & Rieker, 2008; Oksuzyan et al., 2009; del Mar et al., 2012; Luy & Minagawa, 2014; Pongiglione, Stavola, & Ploubidis, 2015). For example, women tend to experience higher rates of chronic debilitating disorders and low rates of life-threatening diseases, such as arthritis, while men generally experience more life-threatening chronic diseases at younger ages, such as coronary heart disease (for a full review, see Bird and Rieker 2008). With continued interest in the morbidity-mortality paradox between women and men, there also has been a strong focus within the literature on “explaining away” these apparent differences through controlling for factors that might account for differential health observations, such as socioeconomic status (e.g. Denton, Prus & Walters 2004; Ross & Mirowsky, 2010; Cherepanov et al., 2010), health behaviours (e.g. Wardle et al., 2004), biology (e.g. Crimmins et al., 2002); and family roles (e.g. Emslie, Hunt & MacIntyre, 1999; Denton & Walters, 1999). Factors such as these have been central to proposed theories concerning the gender paradox in health (for a review, see Bird & Reiker, 2008), yet none has been found to be adequate (Read & Gorman, 2010).

In recent years, scholars have attempted to move beyond some of these more traditional approaches in favour of contextualizing experiences of gender and health within multidimensional, person-centered, and dynamic approaches that draw attention to such important factors as early life conditions (e.g. Hamil-Luker & O’Rand, 2007; Pudrovska, 2014), intersecting lines of stratification (e.g. Warner & Brown, 2011; Brown & Hargrove, 2013), and inter-related life course processes (e.g. Erving, 2011; Suen, 2011; Pudrovska & Anishkin, 2013). Still, relatively little attention has been given to the underlying mechanisms and processes involved within groups of women and men that

may provide greater insight into differences in the patterns of health experienced among each group rather than simply between them. Specifically, there has been an over-reliance on cross-sectional and retrospective data; inattention to multiple resources and health conditions; limited consideration of various age ranges and time spans; and an over-emphasis on comparing women and men rather than what contributes to their respective health outcomes separately.

The integrated articles contained in this dissertation seek to challenge traditional approaches to gender and health through examining factors contributing to heterogeneity among women and men rather than looking only at differences between them. A comprehensive approach to gender and health is taken, incorporating principles from the life course perspective and intersectionality theory.

Paper 1 (Chapter 2) examines the role of cohort in shaping apparent differences in trajectories of self-rated health between women and men. Prior research on gender and self-rated health (SRH) typically has not distinguished between age and cohort-related changes in the health of men and women over time. Through the use of longitudinal panel data, this study finds gender differences in SRH may actually be an artifact of cohort. Prior to examining health across cohorts, women reported worse health than men. With the introduction of cohort to the models, no gender difference was found except in the earliest cohort (born 1924-1933). Thus, inequalities in health between men and women appear to decrease in recent cohorts, suggesting research turn instead to the potential larger differences that exist among them.

Paper 2 (Chapter 3) advances existing research on gender and health by examining how the timing and duration of childhood economic hardship differentiates between those at low and high risk of chronic disease onset in midlife for women and men across four different health outcomes. Results indicate that while women and men experience similar rates of the chronic conditions examined, the processes leading to these outcomes may actually be quite different. Specifically, women who experienced long-term economic hardship in childhood, or began life in poverty but moved out of poverty in childhood, were more likely to experience the onset of diabetes, arthritis, and

cardiovascular diseases in midlife, net of other factors (e.g. adult resources). On the contrary, childhood economic hardship experiences did not differentiate between men and low and high risk of these diseases. This study draws attention to the importance of conceptualizing and measuring childhood disadvantage as dynamic, and reveals that the process of cumulative disadvantage may be more integral to women's health outcomes.

Paper 3 (Chapter 4) further examines differences in health among women through attention to the relationship between social status and both the development of psychosocial resources and good health among and between black and white women. While black women developed the interpersonal rewards of self-efficacy and self-esteem through educational attainment, they did not experience the same degree of health benefits with these advantages as white women. Models estimating self-rated health and chronic conditions suggested that, instead, highly educated black women were at a persistent health disadvantage relative to whites, even at the same levels of psychosocial resources. That being said, black women with higher self-efficacy, and particularly, higher self-esteem, were more likely than black women with lower levels of these resources to report being in better health. Thus, resources may improve health within a disadvantaged group while still not bringing them up to the level of health experienced by their advantaged counterparts. Overall, the findings demonstrate that research should not treat women as a homogenous group, assuming that mechanisms affecting health operate the same for women regardless of their race.

## **1.1 Core theoretical frameworks**

A brief overview of the key theoretical concepts shaping each article will be presented here, with more detailed discussion of specific frameworks used provided in each subsequent chapter. This dissertation takes primarily a life course approach to gender and health, supplemented with intersectional considerations.

### **1.1.1 A life course perspective**

The life course perspective emerged out of changes in history, social demography, and scientific inquiry in North America and Europe, which resulted in widespread recognition that lives are influenced by historical context and/or social conditions as well

as personal biography (see Elder, Johnson & Crosnoe, 2003). Early life course research pointed to the importance of trajectories, or the succession of roles and experiences that occurs within individuals' lives. Individual trajectories can be characterized by periods of change and stability, and are situated within historical and cultural context (George, 1993). Of course, individuals are not limited to one trajectory as they occupy various social roles, which intersect to form social pathways. Social pathways involve the "trajectories of education, work, family, and residences that are followed by individual groups through society" (Elder, Johnson & Crosnoe, 2003, p. 8). In addition, social pathways are influenced by historical forces and "often structured by social institutions" (Elder, Johnson & Crosnoe, 2003, p. 8). Time and place are therefore critical in the study of the life course, as are processes of change, given the life course principle that human development and aging are lifelong processes and individual lives are embedded in both historical and biographical contexts. Later years of life are inextricably linked to early years, and individual development is something that occurs over time and in relation to contextual changes, with individuals' life trajectories located in and shaped by historical times and places. Trajectories also vary according to when particular events or experiences occur in the life course and are influenced by the interdependency of human lives.

The development and principles of the life course perspective make it clear that macro-, meso-, and micro-level factors interact to shape individuals' life course outcomes (George 1993). For example, policy (macro level) can impact the allocation of resources in a community (meso level) which can in turn influence the life conditions and activities of individuals (micro level). With regard to health, the life course perspective postulates that inequalities in health are generated over time by disparities in resources, opportunities, and sources of adversity, for example (Ferraro & Shippee, 2009; O'Rand, 2006). Accordingly, health trajectories and the contexts in which they emerge are best understood as inter-related processes over time rather than single points, and are influenced by multiple domains (e.g. social structure, institutions, individual characteristics). Thus, health, as well as many of its determinants, are not static, but rather, long-term trajectories that extend from childhood to late life. A life course conception of health is therefore particularly relevant for examining the role of historical

context (macro), unequal distributions of resources in early life (meso), and the intersection of gender with other biographical elements (micro). Indeed, gender operates across the life course and in accordance with many additional factors to which the life course perspective draws attention.

The cumulative, contextual, and long-term nature of inequality is referred to in the life course literature as cumulative disadvantage (see Diprete & Elrich, 2006 for a review). Within the life course trajectories of individuals, advantage provides opportunities to obtain access to resources as well as to avoid many sources of adversity, while disadvantage increases exposure to risk (O’Rand, 2006). The first manifestation of advantage and disadvantage, which become magnified with age, appears in childhood. That is, “the unequal provision of physical, social, and economic resources by parents to their children conditions lifelong patterns of inequality” (O’Rand 2006: 149). Initial advantage or disadvantage is therefore compounded or amplified over time to produce heterogeneity in life course outcomes.

In analyzing the connection between early life circumstances and adult health, the effects of timing and duration must also be considered given evidence of their often significant effects on individual outcomes. When childhood poverty is experienced, for example, it can produce variable results. For instance, Guo (1998) reports that early childhood exposure to poverty is most detrimental for cognitive ability, but poverty experienced during early adolescence is what impacts achievement. Longer exposure to childhood disadvantage also appears to have the strongest negative effects on socioeconomic outcomes in comparison to short-term exposure (Wagmiller et al. 2006). Related to health, recent work indicates that remaining in poverty throughout childhood and moving into poverty in later childhood negatively impact health over the life course, while advantaged childhood experiences or moving out of poverty result in a lower risk of poor health outcomes (Shuey & Willson, 2014). That is, individuals who moved out of poverty had health trajectories similar to those who had never experienced poverty at all, while long-term or moving into poverty have deleterious effects on health. Finally, many emerging studies increasingly point to the importance of early childhood experiences of poverty in shaping children’s subsequent cognitive, social, emotional, and physical health

outcomes (see Duncan & Magnuson, 2013 for a review). When and how long experiences of disadvantage occur is thus an essential to understanding life course processes of health as the long-term impacts of events, experiences, and transitions on health are conditional on their timing in individuals' lives (Halfon et al., 2014).

### 1.1.2 Intersectional considerations

In considering the differential likelihood of experiencing advantage and disadvantage, it is important to examine individuals' social locations given that such trajectories are deeply rooted in class, gender, race, and other structured social relations. Individuals simultaneously occupy multiple disadvantaged and/or advantaged statuses, with aspects of social identity (e.g. gender, race/ethnicity, class) intersecting and interacting with systems of oppression (e.g. sexism, racism, capitalism). It is here that intersectionality comes into play in its attention to multiple axes of inequality, rather than privileging one over others (Crenshaw, 1989).

Intersectionality also draws attention to the importance of examining differences among groups through rejecting essentialist assumptions that all members of a particular group are the same (McCall, 2005). Individuals' lives are multi-dimensional and complex, with outcomes such as health inadequately understood through single-factor explanations, such as gender, precisely because life course trajectories are influenced by the simultaneous operation of micro and macro social processes, structures, and dynamics (Hankivsky, 2012). Accordingly, an intersectional approach to health complements the life course perspective through highlighting the importance of intragroup differences, multiple dimensions of stratification, the relevance of context, and a focus on the "whole person" (Creek & Dunn, 2011).

Increasingly, research has begun to consider how intersecting socially-located dimensions, such as gender, race/ethnicity, age, and class, shape individual outcomes and in turn, are essential to the conceptualization and study of health (see Hankivsky, 2012). While the literature here is much less developed than that of cumulative disadvantage and health, recent studies have emerged which apply an intersectional perspective to long-term health trajectories (see, for example Lincoln, 2016; Kapilashrami, Hill, & Meer,



2015; Corus & Saatcioglu, 2015; Brown & Hargrove, 2013; Viruell-Fuentes, Miranda & Abdulrahim, 2012). Specifically, the literature positions an intersectional perspective as relevant to health inequalities research because it challenges researchers to move beyond the examination of single social positions (e.g. class only, race only) in assessing the social determinants of health. Further, intersectionality recognizes that experience of advantage and disadvantage reflect multiple axes of inequality which operate simultaneously.

### 1.1.3 An overview of women's and men's health

To further contextualize the findings of each chapter to follow, I will also provide an overview of gender differences in health. Further information on the specific health outcomes used in this dissertation are located within each chapter.

While the nature of and extent to which gender differences in health exist has been – and continues to be – the subject of debate, women and men indisputably differ in their health profiles (Read & Gorman, 2010). Gender differences in health are most clearly demonstrable regarding mortality, with women experiencing greater life expectancies than men in every developed country across the world. In the United States and Canada, life expectancy at birth for women is 81.2 years and 83.3 years, respectively, and 76.4 years and 78.8 years for men (CDC, 2015; Statistics Canada, 2012). Though the gap is large, it has been declining, with the most frequent explanation centering around men's more rapid reduction in smoking (Read & Gorman, 2010). Finally, it should be noted that heart disease and cancer are the top two leading causes of death for both women and men, even though women have a higher life expectancy than men (Read & Gorman, 2010).

Unlike differences in mortality, differences in morbidity between women and men are not as straightforward, particularly because the gender gap in disease tends to vary by disease outcome and stage of life (Gorman & Read, 2006; Bird & Rieker, 2008). In general, women experience more acute conditions such as upper respiratory infections as well as higher rates of chronic debilitating disorders, including autoimmune diseases, thyroid conditions, migraines, and arthritis (Walters, McDonough, and Strohschein 2002;

Denton, Prus, and Walters 2004; Rieker and Bird 2005). Conversely, men experience more severe chronic diseases at younger ages, such as coronary heart disease, emphysema, cancer, kidney disease, and cirrhosis (Bird and Rieker 2008; Cherepanov et al. 2010). Nevertheless, studies often find varied results regarding the prevalence of chronic conditions, in addition to reports of self-rated health (e.g. Rohlfen & Kronenfeld, 2014, Schnittker, 2007; Idler, 2003), providing further support for a life course account of gender and health that examines multiple health outcomes. In other words, findings of differences in the health conditions of men and women are not consistent across the literature given the use of differing health measures and data (i.e. cross-sectional vs. longitudinal). To move toward a comprehensive understanding of gender and health, various dimensions of health should be considered, especially given the complex nature of what constitutes health and for whom.

## 1.2 A note on methodology

The studies contained in each of the following chapters each use the U.S. Panel Study of Income Dynamics (PSID) and multivariate quantitative methods of analysis. The specific samples and methods used are discussed in each chapter, but a general overview of the dataset will be provided here.

### 1.2.1 Data

The PSID is an ongoing survey that began in 1968 with a nationally representative sample of approximately 5,000 families. The latest wave of available data at the time these chapters were completed was 2011. The PSID is the world's longest running nationally representative household survey. Data were collected annually from 1968 to 1997, and then biennially from 1999 onward (McGonagle et al., 2013). As many as four generations within given families are represented, as children are interviewed as their own family unit after leaving their parents' household, making the PSID advantageous in its multi-generational design in addition to its long span of data collection.

The PSID is administered via computer-assisted telephone interviews, and has a consistently high response rate (~97%) in each wave as well as a high continuation rate. This is particularly important for the success of a long panel study. The PSID is a

valuable source of data for the present study given that health is dynamic, as are a number of its predictors (e.g. income, education, and employment). Survey weights contained in the PSID have also been found to preserve sample representativeness (Fitzgerald, 2011).

Chapter 3 uses data from the Child Development Supplement (CDS) component of the PSID. In 1997, the PSID supplemented main data collection with additional data on a random subsample of parents and their 0-12 year old children. The CDS contains some information on primary caregivers not found in the main PSID, such as measures of self-efficacy, self-esteem, and social support, which were necessary for addressing the objectives of this chapter.

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## Chapter 2

### 2 Re-evaluating gender differences in self-rated health: The importance of cohort<sup>1</sup>

Research on gender differences in self-rated health (SRH) has yielded mixed findings and unanswered questions for over a decade. While many studies suggest that women report worse self-assessed health than men (e.g., Arber & Ginn, 1993; Idler, 2003), others find that this difference has declined in recent years (Cummings & Jackson, 2008; Schnittker, 2007), and many report no gender differences at all (e.g., Gold, Malmberg, McClearn, Pedersen, & Berg, 2002; Macintyre, Hunt, & Sweeting, 1996; Leinonen, Heikkinen, & Jylha, 1998; Prus & Gee, 2003; Rohlfen & Kronenfeld, 2014). Mixed results concerning gender and SRH may be the result of differences in covariates included, survey years used in analyses, non-representative samples, or different age compositions of the samples. Indeed, findings of worse health among women persist partly because little research examines gender differences in health at different ages of adulthood (Read & Gorman, 2006). In fact, age has been argued as “central to our understanding of gender differences in health” and yet is often overlooked (Read & Gorman, 2006, p. 97). Age differences in health have also typically been indistinguishable from cohort differences (McCullough & Laurenceau, 2004; Read & Gorman, 2006; Rohlfen & Kronenfeld, 2014). Thus, inconsistencies in existing findings may also be explained by one key factor that has largely been ignored in discussions of gender differences in self-rated health: the confounding of age and cohort effects.

#### 2.1 Age v. cohort

Age effects refer to physiological or social changes that occur as individuals get older (Zheng, Yang, & Land, 2011). That is, as individuals move from childhood to old age, their bodies, roles, statuses, and beliefs change. Cohort effects, however, occur as groups

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<sup>1</sup> **Etherington, N.** Forthcoming. Re-examining gender differences in self-rated health: The importance of cohort. *Journal of Women & Aging*, 29(6).



of individuals who are born in the same time interval age together and experience similar events at similar times in their lives (Ryder, 1965). This means that cohorts differ from each other through their unique historical origins and experiences over the life course (Ryder, 1965). In other words, age effects reflect developmental changes that occur internally in the individual, while cohort effects capture social change through external social-environmental phenomena or contextual changes (Yang, 2007, 2008). This renders the distinction between age and cohort effects critical for “attributions of etiology or social causation” (Yang, 2007, p. 17). Further, distinguishing between age and cohort has implications for the generalizability of findings. While cohort differences suggest cohort-specific effects of exposure to social risk factors, changes with age can be applicable across cohorts (George, 2004). Since cohort effects capture the effects of historical and social forces throughout the life course, cohort differences are telling of cohort-specific factors that affect changes in SRH. Cohort is also a source of structural inequality with analytical utility analogous to social class (Ryder, 1965), as both categories represent the common experiences of individuals, which serve as important sources of social variation in health and other life course outcomes. Individuals’ life chances can be impacted by the historical time in which they were born. For example, a woman born in the 1990s would have more opportunities for educational attainment than a woman born in the 1920s by virtue of the time period in itself. Incorporating cohort into analyses of gender differences in health can therefore advance our understanding of heterogeneous experiences of SRH over the life course for both men and women and address whether gender differences in SRH are the result of social or individual-level factors. Stratifying models by cohort can identify whether gender differences in SRH are unique to the historical and social forces of a particular time period or occur across time periods, indicative of individual differences between women and men.

From a life course perspective, the inadequate consideration of cohort differences in the study of gender and health is not only a conceptual problem but also methodological, resulting from an overreliance on cross-sectional research designs (e.g., Schnittker, 2008; Zheng et al., 2011). In general, much of what is known about gender and SRH stems from cross-sectional studies (e.g., Cherepanov, Palta, Fryback, & Robert, 2010; Cummings & Jackson, 2008; Denton & Walters, 1999; Duetz, Abel, & Niemann,

2003; Erving, 2011; Lahelma, Arber, Martikainen, Rahkonen, & Silventoinen, 2001; Orfila et al., 2006; Prus & Gee, 2003). Yet cross-sectional data are not indicative of the “true life course changes of individuals” (Yang, 2007, p. 16). Additionally, the cross-sectional relationship among gender, SRH, and age should not be assumed to approximate actual long-term trajectories of men’s and women’s SRH because age and cohort differences are confounded at any one fixed point in time (Yang, 2007). Cross-sectional designs prevent the empirical disentanglement of these effects (Miyazaki & Raudenbush, 2000). Accordingly, the use of cross-sectional data has involved the construction of “pseudo cohorts” and the assumption that the average experience of a particular cohort is represented by the experience of respondents in a specific survey year (Suen, 2011). The ideal cohort study, however, would follow the same individuals throughout their life course (Glenn, 2003). Ignoring cohort patterns also leads to model misspecification, resulting in biased estimations of the relationship between gender and health (Lynch, 2003). Thus, research employing panel data is necessary to test the robustness of cross-sectional findings and to conclusively answer whether gender differences in SRH exist across all cohorts or are specific to certain cohorts.

Although limited by these considerations, extant research on gender and health that incorporates cohort finds a decline in the gender difference in SRH across cohorts and time or a widened then narrowed SRH gap between women and men (Suen, 2011; Zheng et al., 2011). This is not surprising given the substantial changes in women’s work and family lives over the last several decades. Women have surpassed men in terms of university participation and graduation rates (Schnittker, 2008), and women’s rate of labor force participation has greatly increased, along with their annual income. The reduction in the gender gap in self-rated health has been attributed to these factors as well as a decline in men’s self-rated health (Schnittker, 2008). It has also been speculated that as women’s social status becomes more similar to men’s, as is the case in younger cohorts, the gender gap in SRH will diminish (Read & Gorman, 2006; Schnittker, 2008; Ross & Mirowsky, 2010). The social and historical context of gender differences in health, as represented by cohort, should therefore be taken into account (Macintyre et al., 1996).

## 2.2 Socioeconomic status, gender, and SRH

While gender differences in SRH have often been attributed to disparities in resources, the context-dependent nature of the relationship among socioeconomic resources, gender, and health has been under-investigated. A popular explanation for gender differences in health certainly has been the differential allocation of resources to women and men stemming from systems of stratification (for a review, see Read & Gorman, 2010). Some research supports this hypothesis, finding that the gender gap can be explained by differences in socioeconomic and other resources (Denton, Prus, & Walters, 2004; Read & Gorman, 2006), while other research suggests these variables do not explain the gap (Chen, Chang, & Yang, 2008). Mixed results also have emerged concerning the importance of social structural factors in predicting SRH for men and women, particularly education and income, two important protective resources. For example, some studies indicate that education matters more for women's SRH, and nearly all of the improvement in SRH over the last several decades for women is due to their increased educational attainment (Luo & Waite, 2005; Schnittker, 2008). Others find education to be a stronger predictor of men's SRH, while income is reported as a stronger predictor of SRH for women (Prus & Gee, 2003). Specifically, belonging to the highest income category appears to be a more important predictor of SRH for women than men (Denton & Walters, 1999), although other research suggests income is actually more important for men (Luo & Waite, 2005). Finally, Duetz et al. (2003) report that socioeconomic status is significantly associated with SRH for women only. These inconsistent findings may be partially explained by cohort, as gendered access to these resources has changed across time. The impact of education and income on gender differences in health may vary from older to more recent cohorts. This has yet to be examined but is an important part of understanding how specific cohorts may experience health differently and whether social status characteristics have as strong of an effect on health inequality for different cohorts.

Research does suggest that the health benefits of education and income accumulate over time and that this occurs less so for earlier cohorts (Willson, Shuey & Elder, 2007). Cohort differences in returns to education have resulted from homogeneity in educational attainment among more recent cohorts as education levels have increased

(Levy & Murnane, 1992). The relationship between education and health varies across cohorts, with effects increasing among more recent cohorts (Lynch, 2003). These effects may, however, be indirect through income, suggesting that the direct effect of education on health has actually decreased across cohorts (Lynch, 2006). Research also finds that educational disparities in health have widened among more recent cohorts of women in particular (Cutler, Lange, Meara, Richards-Shubik, & Ruhme, 2011; Liu & Hummer, 2008; Montez & Zajacova, 2013; Montez, Hummer, Hayward, Woo, & Rogers, 2011). According to the theory of resource substitution, education is more beneficial to women's health than men's in that it can substitute for other resources of which women have fewer (e.g., power, earnings) (Ross & Mirowsky, 2010). Conversely, the reinforcement of advantage hypothesis postulates education as more beneficial to men's health because men are already advantaged in terms of resources, and this advantage becomes multiplied with higher educational attainment. While some studies support the theory of resource substitution (Ross & Mirowsky, 2010), it is unclear whether women reap greater benefits of education than men across cohorts.

It is also the case that women are more likely than men to be economically disadvantaged, despite slightly higher high school and postsecondary graduation rates (Read & Gorman, 2010). Indeed, women earn 20% less than men in comparable jobs with the same qualifications across all workers age 16 and older (Hegewisch, Liepmann, Hayes, & Hartmann, 2010). This gap has decreased over time, which may have important implications for SRH. For instance, Schnittker (2008) suggests that if women reported the same income as men, they would also report better health, on average. Whether different patterns of educational and income differences in health occur for women and men across cohorts remains to be examined. Time and context are therefore key elements to consider in specifying the relationship among gender, resources, and SRH.

Currently, there is limited understanding of the role of birth cohort as a factor contributing to gender differences in SRH over the life course. As new methodologies and longitudinal data become available, it is important to reexamine gender differences in health to obtain a better understanding of their complexity (Macintyre et al., 1996) as well as an accurate portrayal of how men's and women's trajectories of SRH converge

and diverge over time. The present study distinguishes between age and cohort and assesses the extent to which gender differences in SRH and in returns to education and income vary by cohort. The following research questions are posed:

(1) (a) How do gender differences in trajectories of self-rated health vary by age and cohort? (b) Is this relationship explained by education and income?

(2) (a) Does education or income improve health more for one gender than the other? (b) Does this relationship depend on cohort?

## 2.3 Methods

### 2.3.1 Data

This study uses data drawn from the 1999–2011 waves of the U.S. Panel Study of Income Dynamics (PSID). The PSID began in 1968 with a nationally representative sample of 4,802 families who were interviewed each year until 1997, when interviews became biennial (Panel Study of Income Dynamics, 2013). As the world’s longest-running nationally representative household survey, the PSID is ideal for assessing cohort effects and trajectories of health over time. Wave-to-wave response rates for the PSID are between 95% and 98%. When children of PSID respondents leave the family home, they become PSID sample members as well, and this “split-off” sample has been found to be representative. The seven observation points selected are years in which information on self-rated health and detailed data on other important covariates such as lifestyle factors and health conditions were collected. Since growth curve models allow for the use of unbalanced panels, it was not necessary for a respondent to be interviewed in every wave. The sample was, however, limited to respondents who were 26 to 75 years old in 1999. In addition, respondents had to be a “head” or a “wife” in 1999, as these are the individuals for which information on all covariates is collected in each survey year. These restrictions resulted in a sample of 6,782 respondents.

Longitudinal data analysis faces the challenge of missing data. Individuals remaining in the PSID sample tend to be healthier, White, female, and of higher socioeconomic status, with annual attrition rates between 2.5% and 3% (Fitzgerald et al.,

1998). Logistic regressions not shown here find that respondents with lower education and income, poor health, and men were less likely to have an observed health observation in 2011. Nevertheless, analyses of the PSID suggest that the PSID has generally maintained its representativeness over time (Fitzgerald, 2011; Halliday, Kimmitt, & Kimmitt, 2008; Meer, Miller, & Rosen, 2003). Still, the results of this analysis are likely conservative in nature.

### 2.3.2 Analytic strategy

Trajectories of SRH from 1999 to 2011 were estimated using growth curve analysis. Growth curve models use longitudinal data to assess between-person differences in within-person change over time (Singer & Willett, 2003). This strategy allows the estimation of gender and cohort differences in initial SRH (i.e., intercepts) and rate of growth over time (i.e., slopes). Two level growth curve models were estimated. The level-1 equation describes within-individual changes in SRH ( $i$ ) with age ( $t$ ):

$$y_{it} = \alpha_i + \beta_i t + y_i w_{it} + \varepsilon_{it}$$

Individuals' SRH ( $y_{it}$ ) is characterized by a unique intercept ( $\alpha_i$ ) and a time-dependent linear slope ( $\beta_i$ ). The effects of time-varying covariates on SRH is captured by  $y_i w_{it}$ , which represents the effect of time-varying covariates  $w_{it}$  on SRH at time  $t$  for each  $i^{th}$  individual.

The level-2 equation describes variation in SRH between women and men or across individual effects. The level-2 equations can be represented as follows:

$$\alpha_i = \alpha_0 + \alpha_1 x_{i1} + \alpha_2 x_{i2} \dots \alpha_k x_{ik} + u_i$$

$$\beta_i = \beta_0 + \beta_1 x_{i1} + \beta_2 x_{i2} \dots \beta_k x_{ik} + v_{it}$$

These equations specify that the random intercepts ( $\alpha_i$ ) and slopes ( $\beta_i$ ) are a function of time invariant covariates ( $x_{ik}$ ) and error terms ( $u_i, v_{it}$ ).

I first examine the effect of gender on SRH in addition to the role of education and income without assessing cohort. Next, I present these effects across cohorts.

Differences between cohorts are assessed using the  $\chi^2$  difference test. All analyses were executed using Stata 13. Analyses were weighted using the PSID 2011 longitudinal weights.

## 2.4 Measures

### 2.4.1 Dependent variable

SRH is measured based on respondents' answers to the question "Would you say your health in general is excellent, very good, fair, or poor?" Similar to other research, SRH is treated as a continuous measure (e.g., Chen et al., 2008; Denton & Walters, 1999; Gorman & Read, 2006; McCullough & Laurenceau, 2004; Prus & Gee, 2003; Willson et al., 2007). Values were recorded so that 1 refers to excellent health and 5 refers to poor health. SRH is measured at each time point included in the analysis. SRH is highly correlated with chronic health problems, functional limitations, and mortality (Idler & Benyamini, 1997; McDonough & Amick, 2001; Benyamini et al., 2003). It is also a useful measure to capture differences in health across a wide range of ages (Deaton & Paxton, 1998), as other measures of health such as chronic disease are more relevant at later stages of life.

### 2.4.2 Independent variable

In order to understand long-term health outcomes as a result of being born in a certain cohort, meaningful categories are necessary. Cohort was included as a categorical variable, as a continuous variable prevents the identification of particular social factors operating at certain time points, which may influence health. Respondents were divided into five cohorts based on their age at the beginning of the observation period (1999), starting with the earliest year of birth in the sample. The cohorts reflect 10-year groupings and correspond to the following years of birth: 1924–1933, 1934–1943, 1944–1953, 1954–1963, and 1964–1973. These groupings reflect important periods of time, where individuals who were born or were young children in the Great Depression (1924–1933), prior to/after World War II (1934–1943, 1944–1953), and early (1954–1963) and

late baby boomers (1964–1973) are identifiable. Age is assessed in years at the time of each survey and is centered at the grand mean of the sample (56 years) to aid with interpretation. Research suggests that experiences of aging vary by cohort (Kasen, Cohen., Chen, & Castillee, 2003; O’Rand & Henretta, 1999), making an interaction of age and cohort effects on SRH necessary. Such an interaction hypothesizes that cohorts differ in initial health and in rates of change in health over time (Yang, 2007). An *age X cohort* interaction term is therefore included.

Gender was coded 0 for men and 1 for women. Education was measured as a continuous variable reflecting number of years of school completed (Rohlfesen & Jacobs Kronenfeld, 2014; Ross & Mirowsky, 2010; Walters, McDonough, & Strohschein, 2002). For each year of observation, total household income was lagged by one year, adjusted for inflation, and logged to adjust for skew.

### 2.4.3 Control variables

Important gender differences exist in patterns of smoking, drinking, and physical activity, with implications for health (Barbeau, Krieger, & Soobader, 2004; Lipowicz, Koziel, Hulanicka, & Kowalisko, 2007; Trost, Owen, Bauman, Sallis, & Brown, 2002; York, Welte, & Hirsch, 2003). Specifically, these health behaviours can impact how individuals assess their health in addition to objective physical health conditions, which also impact how individuals feel about their health. Accordingly, variables were included from each survey year to account for these lifestyle factors. Drinking and smoking were dichotomized, with current smoker equal to 1 and one or more drinks per day equal to 1 (Kagotho, 2009). Frequency of heavy physical activity was also dichotomized, with 1 equal to never engages in physical activity (Hamil-Luker & O’Rand, 2007; Kagotho, 2009).

Since men and women also differ in the types of health conditions they experience, with men generally having more life-threatening diseases and women having more chronic debilitating conditions (Bird & Rieker, 2008), several physical health conditions were included, similar to Rohlfesen and Kronenfeld (2014). For each condition, 1 indicates the presence of that condition. The following conditions were included:



diabetes, high blood pressure, cancer, heart attack, stroke, arthritis, heart disease, and lung disease (excluding asthma). Mental health can also affect ratings of physical health, and depressive symptoms are more common among women (Bird & Rieker, 2008). In addition, stress-related factors appear to matter more for women's ratings of SRH. While the PSID does not include a measure of depression, it does include a measure of nonspecific psychological distress using the K6 scale. The K6 scale consists of responses to six questions concerning how often the respondent felt nervous, restless or fidgety, hopeless, sad, worthless, or that everything was a struggle in the past 30 days. Scores for each question range from 0 to 4 and are summed for a total score between 0 and 24, with higher scores indicating greater distress. The K6 scale is a reliable estimator of mental illness and has strong psychometric properties (Kessler et al., 2002).

In models not shown here, all of the health conditions described above were excluded, and findings generally remained the same. It can be important to understand SRH net of existing objective health conditions given research indicating that individuals' perceptions about their health is related to accessing services, health behaviours, and so forth – all of which impact health, and ultimately, mortality (Schnittker & Bacak, 2014; Schootman et al., 2012; DeSalvo et al., 2005). Further, quantitative analyses of SRH complement existing qualitative analyses of what people perceive as important to their health through taking into account factors related to social location at a large, representative scale (Rohlfen & Kronenfeld, 2014). Finally, how individuals feel about their health can offer a more complete account given individual ratings tend to reflect a more inclusive set of factors that can typically be gathered via survey instruments or even medical examinations (Welch, Schwartz & Woloshin, 2011). For example, individuals might consider not only chronic conditions or functional limitations, but also cognitive abilities, psychological well-being, health behaviours, health history, medication use, and social participation, among other factors (Feng et al., 2014).

Given its association with SRH as well as with socioeconomic status (Cummings & Jackson, 2008; Erving, 2011; Shuey & Willson, 2008), race/ethnicity was also included in the analysis and coded as non-Hispanic White (0) and non-Hispanic Black

(1). Finally, employment and marital status, two important predictors of SRH, were measured as 1 = employed and 1 = married.

## 2.5 Results

### 2.5.1 Descriptive statistics

Table 2.1 presents weighted proportions and means for the key variables involved in the study. Across cohorts, there is a slightly higher proportion of women than men. A larger proportion of individuals in cohort 5 report fair (0.21) and poor (0.08) health compared to other cohorts. Cohorts 1 and 2 have the highest proportion of individuals reporting very good (0.41, 0.40) and excellent (0.24, 0.19) health. Mean years of education is approximately 14 for cohorts 1 through 3. Cohorts 4 and 5 have an average of 13 years of education. Logged income is similar across cohorts. The mean age in years for each cohort is roughly 38, 48, 57, 67, and 77 respectively.

**Table 2.1 Weighted proportions and means for key variables, PSID 1999-2011**

Variable	Cohort 1 (1965- 1973)	Cohort 2 (1954- 1963)	Cohort 3 (1944- 1953)	Cohort 4 (1934- 1943)	Cohort 5 (1924- 1933)
<i>Gender</i>					
Female	0.53	0.51	0.52	0.56	0.59
Male	0.47	0.49	0.48	0.44	0.41
<i>Self-Rated Health</i>					
Excellent	0.24	0.19	0.17	0.14	0.10
Very Good	0.41	0.40	0.36	0.31	0.28
Good	0.26	0.29	0.31	0.35	0.33
Fair	0.07	0.09	0.12	0.15	0.21
Poor	0.02	0.03	0.04	0.06	0.08
<i>Education (Years)</i>					
	13.74 (0.04)	13.62 (0.03)	13.94 (0.04)	13.24 (0.05)	12.83 (0.07)
<i>Income (Logged)</i>					
	11.17 (0.02)	11.27 (0.02)	11.23 (0.02)	10.90 (0.02)	10.63 (0.02)
<i>Age (Years)</i>					
	37.89 (0.08)	47.83 (0.07)	57.39 (0.07)	67.21 (0.11)	77.00 (0.12)

Notes. Proportions and means represent average values across all survey years.

### 2.5.2 Multivariate analyses

Growth curve models of self-rated health prior to the inclusion of cohort are presented in Table 2.2. In model 1, women have slightly worse self-reported health than men ( $\beta =$

0.04,  $p = .02$ ) but experience a slower rate of decline over time ( $\beta = -0.003$ ,  $p = .01$ ). This gender difference in health remains significant when education and income are introduced in model 2. An interaction between gender and education is tested in model 3 but is not significant, indicating that education has the same health-protective effect for both men and women. In contrast, in model 4, there is a significant interaction between gender and income ( $\beta = 0.14$ ,  $p = .02$ ), indicating that higher income does not offer the same protection for women as men. Across all models, education and income are beneficial for health but do not explain the gender difference in SRH. Increasing age is also associated with worse health over time, while women remain advantaged in terms of rate of decline in SRH (prior to interaction terms introduced in model 4).

Tables 2.3 through 2.6 stratify models by cohort. Model 1 (Table 2.3) reveals no gender difference in health in cohorts 1, 2, 3, or 4. A difference does exist, however, in cohort 5 (the earliest cohort), with women of this cohort reporting significantly worse health than men ( $\beta = 0.48$ ,  $p = .017$ ). Predicted means for women and men in each cohort with all controls are displayed in Figure 2.1. Here it can be seen that women and men have similar SRH in all cohorts but cohort 5, where a significant difference is evident. Age is associated with worse SRH in all cohorts, while women experience a slower health decline than men.

**Table 2.2 Growth curve models of self-rated health, PSID 1999-2011 (N=6782)**

	Model 1		Model 2		Model 3		Model 4	
	$\beta$ (SE)	p- value	$\beta$ (SE)	p- value	$\beta$ (SE)	p- value	$\beta$ (SE)	p- value
Female	0.04 (0.02)	0.02	0.04 (0.02)	0.02	-0.12 (0.10)	0.22	-1.15 (0.71)	0.10
Age (Centred)	0.01 (0.001)	0.000	0.01 (0.001)	0.000	0.01 (0.001)	0.000	0.02 (0.01)	0.001
Female X Age (Centred)	-0.003 (0.001)	0.01	-0.003 (0.001)	0.01	-0.002 (0.001)	0.02	-0.01 (0.01)	0.18
Education			-0.06 (0.0003)	0.000	-0.07 (0.01)	0.000	-0.05 (0.01)	0.000
Income			-0.04 (0.01)	0.000	-0.04 (0.01)	0.000	-0.19 (0.05)	0.000
Female X Education					0.01 (0.01)	0.09		
Female X Income							0.14 (0.06)	0.02
Intercept	3.35 (0.08)	0.000	3.35 (0.08)	0.000	3.44 (0.09)	0.000	4.30 (0.58)	0.000

Notes. All models control for marital status, employment status, smoking, drinking, physical activity, psychological distress, high blood pressure, arthritis, diabetes, heart disease, lung disease, cancer, stroke, and race/ethnicity. 5=poor health.

Variance components not shown to conserve space.

**Table 2.3 Growth curve models of self-rated health, PSID 1999-2011 (N=6782): Model 1**

	Cohort 1 <sup>a</sup>		Cohort 2 <sup>b</sup>		Cohort 3 <sup>c</sup>		Cohort 4 <sup>d</sup>		Cohort 5 <sup>e</sup>	
	$\beta$ (SE)	p-value	$\beta$	p-value	$\beta$	p-value	$\beta$	p-value	$\beta$	p-value
Female	-0.03 (0.08)	0.72	-0.000 (0.04)	0.99	0.02 (0.03)	0.49	0.03 (0.09)	0.72	0.48*** (0.20)	0.017
Age (Centred)	0.03 (0.003)	0.000	0.02 (0.002)	0.000	0.01 (0.003)	0.008	0.01 (0.01)	0.02	0.03 (0.01)	0.000
Female X Age (Centred)	-0.01 (0.004)	0.10	-0.01 (0.003)	0.02	-0.002 (0.004)	0.47	0.003 (0.01)	0.63	-0.02 (0.01)	0.08
Intercept	2.32 (0.08)	0.000	2.27 (0.05)	0.000	2.15 (0.05)	0.000	2.04 (0.10)	0.000	1.54 (0.17)	0.000

Notes. \*\*\* $\chi^2=11.26$ ,  $p=0.0008$ . All models control for marital status, employment status, smoking, drinking, physical activity, psychological distress, high blood pressure, arthritis, diabetes, heart disease, lung disease, cancer, stroke, and race/ethnicity. Variance components not shown to conserve space. 5=poor health.

<sup>a</sup>N(obs)=7663. <sup>b</sup>N(obs)=10426. <sup>c</sup>N(obs)=7441. <sup>d</sup>N(obs)=2924. <sup>e</sup>N(obs)=2148.

**Table 2.4 Growth curve models of self-rated health, PSID 1999-2011 (N=6782): Model 2**

	Cohort 1 <sup>a</sup>		Cohort 2 <sup>b</sup>		Cohort 3 <sup>c</sup>		Cohort 4 <sup>d</sup>		Cohort 5 <sup>e</sup>	
	$\beta$ (SE)	p-value	$\beta$	p-value	$\beta$	p-value	$\beta$	p-value	$\beta$	p-value
Female	-0.01 (0.08)	0.88	-0.00 (0.04)	0.991	-0.00 (0.03)	0.907	-0.02 (0.09)	0.86	0.45*** (0.20)	0.024
Age (Centred)	0.03 (0.003)	0.000	0.02 (0.002)	0.000	0.01 (0.003)	0.01	0.01 (0.01)	0.02	0.03 (0.01)	0.000
Female X Age (Centred)	-0.01 (0.003)	0.126	-0.01 (0.003)	0.023	-0.003 (0.004)	0.407	0.00 (0.01)	0.61	-0.02 (0.01)	0.07
Education	-0.06 (0.01)	0.000	-0.07 (0.01)	0.000	-0.07 (0.01)	0.000	-0.07 (0.01)	0.000	-0.05 (0.01)	0.000
Income	-0.04 (0.01)	0.000	-0.04 (0.01)	0.000	-0.04 (0.01)	0.000	0.00 (0.02)	0.945	-0.10 (0.03)	0.003
Intercept	3.57 (0.17)	0.000	3.63 (0.14)	0.000	3.56 (0.15)	0.000	3.05 (0.26)	0.000	3.28 (0.39)	0.000

Notes. \*\*\* $\chi^2=10.08$ ,  $p=0.0015$ . All models control for marital status, employment status, smoking, drinking, physical activity, psychological distress, high blood pressure, arthritis, diabetes, heart disease, lung disease, cancer, stroke, and race/ethnicity. Variance components not shown to conserve space. 5=poor health.

<sup>a</sup>N(obs)=7663. <sup>b</sup>N(obs)=10426. <sup>c</sup>N(obs)=7441. <sup>d</sup>N(obs)=2924. <sup>e</sup>N(obs)=2148.

**Table 2.5 Growth curve models of self-rated health, PSID 1999-2011 (N=6782): Model 3**

	Cohort 1 <sup>a</sup>		Cohort 2 <sup>b</sup>		Cohort 3 <sup>c</sup>		Cohort 4 <sup>d</sup>		Cohort 5 <sup>e</sup>	
	$\beta$ (SE)	p-value	$\beta$	p-value	$\beta$	p-value	$\beta$	p-value	$\beta$	p-value
Female	-0.14 (0.22)	0.51	0.08 (0.18)	0.65	-0.21 (0.19)	0.25	-0.18 (0.30)	0.56	-0.10 (0.32)	0.76
Age (Centred)	0.03 (0.003)	0.000	0.02 (0.002)	0.000	0.01 (0.003)	0.01	0.01 (0.01)	0.02	0.03 (0.01)	0.000
Female X Age (Centred)	-0.01 (0.003)	0.13	-0.01 (0.003)	0.02	-0.003 (0.004)	0.41	0.003 (0.01)	0.60	-0.02 (0.01)	0.06
Education	-0.06 (0.01)	0.000	-0.06 (0.01)	0.000	-0.07 (0.01)	0.000	-0.08 (0.02)	0.000	-0.08 (0.02)	0.000
Education X Female	0.01 (0.01)	0.51	-0.01 (0.01)	0.64	0.02 (0.01)	0.25	0.01 (0.02)	0.58	0.05*** (0.02)	0.03
Income	-0.04 (0.01)	0.000	-0.04 (0.01)	0.64	-0.42 (0.01)	0.000	0.001 (0.02)	0.94	-0.10 (0.03)	0.003
Intercept	3.65 (0.21)	0.000	3.59 (0.17)	0.000	3.67 (0.18)	0.000	3.12 (0.29)	0.000	3.56 (0.41)	0.000

Notes. \*\*\* $\chi^2=12.06$ ,  $p=0.0005$ . All models control for marital status, employment status, smoking, drinking, physical activity, psychological distress, high blood pressure, arthritis, diabetes, heart disease, lung disease, cancer, stroke, and race/ethnicity. Variance components not shown to conserve space. 5=poor health,

<sup>a</sup>N(obs)=7663. <sup>b</sup>N(obs)=10426. <sup>c</sup>N(obs)=7441. <sup>d</sup>N(obs)=2924. <sup>e</sup>N(obs)=2148.

Education and income are introduced in model 2 (Table 2.4). The gender difference in health in cohort 5 remains significant ( $\beta = 0.45$ ,  $p = .02$ ). Once more, there is no difference in the SRH of women and men in any of the other cohorts. Education is protective for health in all cohorts, while income is protective for health in all cohorts but cohort 4. Age remains a significant predictor of worse SRH for all cohorts, and the female advantage remains for cohort 2.

An interaction between education and gender is tested in model 3 (Table 2.5). Here, women in cohort 5 report slightly worse SRH than men even with higher levels of education ( $\beta = 0.05$ ,  $p = .03$ ). This interaction is not significant in any other cohort. Higher education remains beneficial to SRH in all cohorts. Findings related to age maintain their significance.

Model 4 (Table 2.6) introduces an interaction between gender and income. In cohort 5, women with higher levels of income remain at a health disadvantage relative to men ( $\beta = 0.14$ ,  $p = .01$ ). Income is beneficial to SRH in all cohorts but cohort 4. Once again, SRH worsens with age, and women in cohort 2 experience slower decline than men.



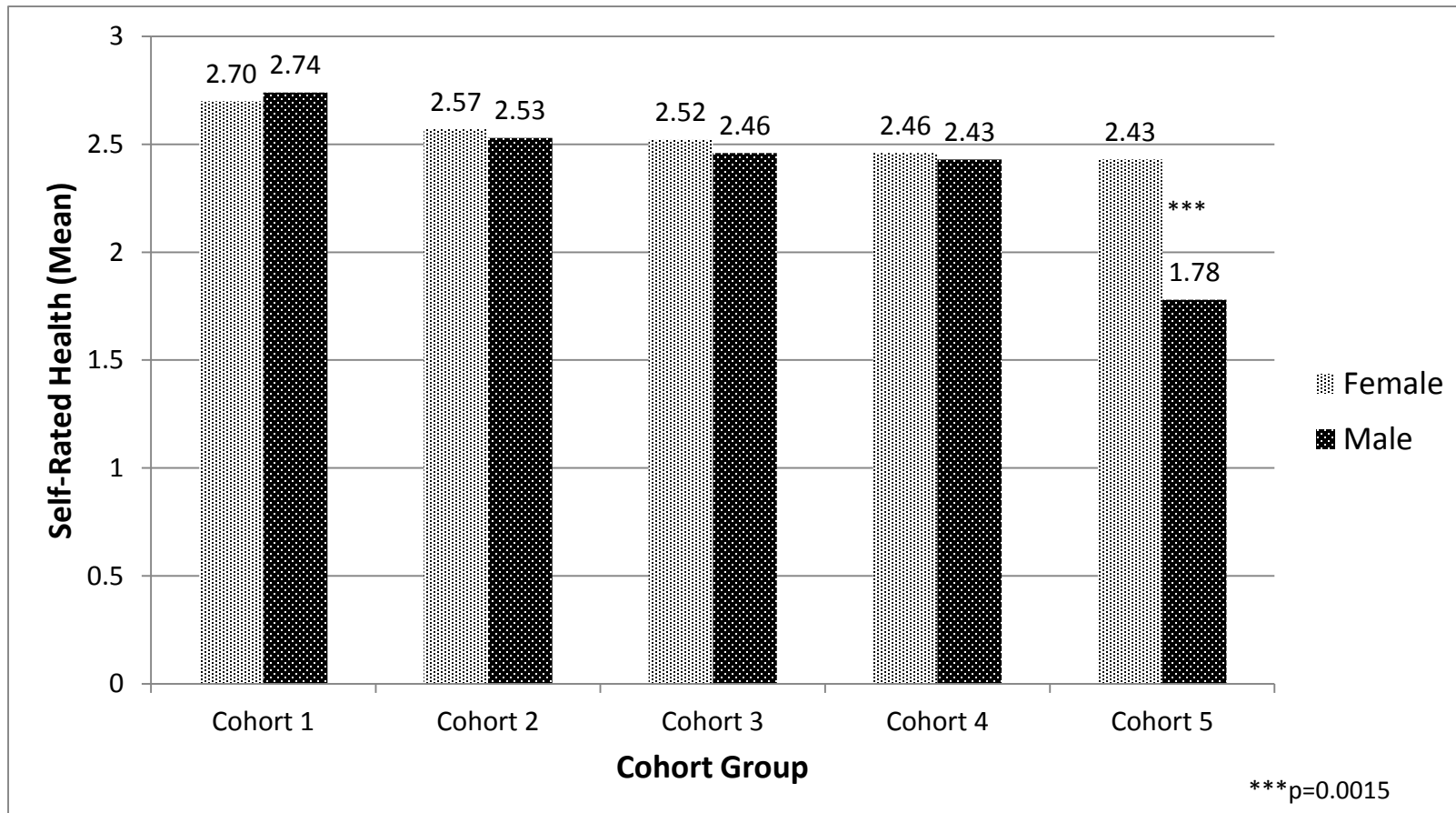
**Table 2.6 Growth curve models of self-rated health, PSID 1999-2011 (N=6782): Model 4**

	Cohort 1 <sup>a</sup>		Cohort 2 <sup>b</sup>		Cohort 3 <sup>c</sup>		Cohort 4 <sup>d</sup>		Cohort 5 <sup>e</sup>	
	B (SE)	p-value	β	p-value	β	p-value	β	p-value	β	p-value
Female	0.27 (0.26)	0.30	-0.23 (0.20)	0.27	-0.07 (0.23)	0.77	-0.44 (0.40)	0.27	-1.15 (0.71)	0.10
Age (Centred)	0.03 (0.003)	0.000	0.02 (0.002)	0.000	0.01 (0.003)	0.01	0.01 (0.01)	0.03	0.02 (0.01)	0.001
Female X Age (Centred)	-0.01 (0.003)	0.15	-0.01 (0.003)	0.02	-0.003 (0.004)	0.43	0.004 (0.01)	0.51	-0.01 (0.01)	0.18
Education	-0.06 (0.01)	0.000	-0.07 (0.01)	0.000	-0.07 (0.01)	0.000	-0.07 (0.01)	0.000	-0.05 (0.01)	0.000
Income	-0.03 (0.02)	0.12	-0.05 (0.01)	0.000	-0.04 (0.01)	0.003	-0.02 (0.03)	0.47	-0.19 (0.05)	0.000
Income X Female	-0.03 (0.02)	0.26	0.02 (0.02)	0.26	0.01 (0.02)	0.78	0.04 (0.03)	0.28	0.14*** (0.06)	0.01
Intercept	3.40 (0.23)	0.000	3.76 (0.18)	0.000	3.59 (0.19)	0.000	3.27 (0.33)	0.000	4.30	0.000

Notes. \*\*\* $\chi^2=8.13$ ,  $p=0.0044$ . All models control for marital status, employment status, smoking, drinking, physical activity, psychological distress, high blood pressure, arthritis, diabetes, heart disease, lung disease, cancer, stroke, and race/ethnicity. Variance components not shown to conserve space. 5=poor health.

<sup>a</sup>N(obs)=7663. <sup>b</sup>N(obs)=10426. <sup>c</sup>N(obs)=7441. <sup>d</sup>N(obs)=2924. <sup>e</sup>N(obs)=2148.

Figure 2.1 Predicted means for self-rated health by gender and cohort, PSID 1999-2011 (N=6782)



## 2.6 Discussion

Research examining gender differences in SRH has typically not used panel data, inhibiting the distinction of age and cohort-related changes in the health of men and women over time. Previous studies have produced mixed findings concerning gender and SRH as well as the role of important socioeconomic resources such as income and education. Results from this study indicate that purported gender differences in SRH may actually be an artifact of cohort. Prior to examining health across cohorts, there appeared to be a gender difference in health not explained by education or income, with women reporting slightly worse health than men. Income also appeared to be more protective to men's health. With the introduction of cohort to the models, no gender difference was found except in the earliest cohort. This difference remained when education and income were taken into account. It is important to note that these findings are net of differences in physical health conditions and mental health, which differ for men and women and influence reports of SRH. Additional analyses not shown here were conducted without these control variables and still found that gender differences in SRH existed only in the earliest cohort. Thus, men and women's reports of health differ only among those individuals in the early cohort, regardless of controls for other health conditions. Still, these controls were included in order to rule out differing physical and mental health conditions as explanations for gender differences in SRH (see Rohlfsen & Jacobs Kronenfeld, 2014), and to capture individuals' perceptions of their own health (Feng et al., 2014).

Education and income were found to benefit health across cohorts, but this beneficial effect was not equal for men and women in the earliest cohort. Men in the earliest cohort receive higher rates of return to both education and income across the life course, leaving women at a health disadvantage relative to men despite the presence of these resources. Thus, the theory of resource substitution, which has found support in other research (Luo & Waite, 2005; Schnittker, 2008; Mirowsky & Ross, 2010), may not apply for all cohorts. This study also challenges research finding income to be more beneficial to women's SRH (Denton & Walters, 1999) and provides a more nuanced account of this relationship through identifying its cohort-specific nature. Overall, then,

disadvantages in resources and health found among women may actually reflect those found in the earliest cohort and not a generalizable phenomenon irrespective of birth year.

A lack of gender differences in the rate of change in SRH over time also suggests it is not necessarily gender that is associated with worse SRH with age but rather that birth cohorts experience different patterns of gender differences in SRH. One exception with regard to change in health over time was that the advantage in health was found among women born in 1954–1963 only. These individuals represent the “late” or “trailing edge” boomers. Accordingly, a female advantage in SRH with time may be understood in the context of greater average life expectancy, increasing numbers of women completing higher education, and more women in managerial and professional jobs found among this cohort (Marrion, 2013; MetLife, 2009).

Repeated cross-sectional research also finds a smaller gap in SRH between men and women of more recent cohorts and a larger gap in earlier cohorts (Zheng et al., 2011). This raises important questions concerning the causes of cohort differences in gendered health inequalities. It may be that women who were young children or born in challenging historical periods, such as the Great Depression, were more adversely affected by these events. Certainly, research suggests that childhood disadvantage may matter more for women’s health outcomes than men’s (Hamil-Luker & O’Rand, 2007). Women in the earliest cohort in this study would have been in infancy and early childhood during the Great Depression. Elder’s (1999) study of children of the Great Depression found that younger children were more negatively affected by the economic collapse than individuals in adolescence. Further, economic losses appeared less consequential for the boys in the study. Future research should continue to explore how negative social events and economic disadvantage may impact men’s and women’s health differently over the life course. The less-protective capacity of income and education for women in the earliest cohort compared to men lends support to the idea that women are more adversely affected by childhood deprivation and that this is not compensated for by adult resources over time. Still, gender differences in the impact of particular resources may be a result of cross-sectional designs that fail to capture the

relationship between health and its predictors over time and across cohorts.

This study has several limitations. First, sample attrition is unavoidable in longitudinal research. Sensitivity analyses indicate that respondents in poor health in the first wave of data collection were less likely to remain in the sample until 2011. Accordingly, SRH may be overestimated and its relationship with SES underestimated. Men were also less likely to remain in the sample, meaning any gender differences in health may be underestimated. Second, reverse causality is a possibility, although several steps were taken to limit this problem, such as incorporating measures of the prior year's income as well as multiple measures of SES.

Overall, results from this study provide support to cross-sectional findings that challenge findings of women reporting worse SRH than men (Suen, 2011; Zheng et al., 2011) and highlight the importance of historical timing in the relationship among gender, health, and aging. Importantly, the health disadvantage faced by women appears to exist only for the earliest cohort. This study therefore challenges findings that suggest that the gender gap disappears with age (e.g., McCullough & Laurenceau, 2004) and confirms findings of a decline in gender differences in SRH in recent years to nonexistent (e.g., Cummings & Jackson, 2008; Prus & Gee, 2003). Cohort-specific effects of income and education for women were also demonstrated, meriting the reexamination of protective resources, gender, and health.

Given that the gender gap in SRH is specific to a particular cohort, there are two promising avenues for future research. First, research should focus on further understanding what contributes to the gap between men and women in the earliest cohort and how to best address their unique health needs. Second, research should turn to examining processes of heterogeneity among women and men rather than emphasizing differences between them (Evans-Campbell et al., 2010). That is, women and men are not homogenous groups, and the intersection of gender with race/ethnicity, age, ability, sexual orientation, and other aspects of identity warrants further investigation. Certainly, further incorporation of life course principles such as timing should take precedence as we continue to improve our understanding of gender and health because these principles

can challenge traditional assumptions based on models with limited to no consideration of historical context. The health of men and women from different birth cohorts unfolds in different sociohistorical contexts, and we should not expect the magnitude of gender differences in health to be uniform across cohorts.

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## Chapter 3

### 3 Heterogeneity in chronic disease outcomes among women and men in midlife: Examining the role of stability and change in childhood economic hardship<sup>2</sup>

Improving gender equity in health has been recognised by the World Health Organization as “one of the most direct and potent ways to reduce health inequities overall and ensure effective use of health resources” (Sen & Ostlin 2007: viii.). Gender differences in health are well-documented in the United States (US) (see Read & Gorman, 2010 for a review), with women experiencing greater morbidity than men despite living longer. Yet attempts to explain and reduce disparities in health faced by women and men have typically focused on the relationship between health and resources in adulthood, with limited attention to the early origins of disease and health over the life course.

Through its emphasis on human development and aging as lifelong processes (Elder, Johnson, & Crosnoe, 2003), the life course perspective has directed attention to the ‘long arm’ of childhood disadvantage, or how early life conditions impact health and other outcomes in adulthood. Research increasingly has recognised the importance of childhood origins in shaping health disparities (Murray et al., 2011; Diprete & Eirich, 2006), yet little work has examined how childhood context may differentially affect men and women. Existing research treats gender as a control variable rather than a focal point (e.g. Bowen, 2010), often attempting to “explain away” gender differences rather than examining how social factors may operate differently for women and men and lead to divergent health trajectories and heterogeneity within groups (Evans-Campbell, Lincoln & Takeuchi, 2010). Further, childhood disadvantage has been treated as static, rather than as a dynamic process involving stability and change over time (e.g. Pudrovska &

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<sup>2</sup> **Etherington, N.**, Willson, A.E., & Shuey, K. (2016). Childhood economic hardship and chronic disease onset in midlife: A gendered process of cumulative disadvantage? *Longitudinal and Life Course Studies*, 7(1), 3-24.

Anikputa, 2013; Walsemann, Ailshire, Bell & Frongillo, 2012; Lemelin et al., 2009), and its effects rarely compared across health outcomes.

The current study advances research on childhood disadvantage, gender, and health by conceptualizing and measuring childhood economic context as a dynamic process that may affect disease onset in midlife differently for women and men. Specifically, we take into consideration stability and change in the experience of childhood poverty and its impact on health in midlife. Using the US Panel Study of Income Dynamics (PSID), these relationships are examined across four chronic disease outcomes that are among the most prominent causes of morbidity and mortality in the United States.

### 3.1 Background

Diabetes, hypertension, arthritis, stroke, heart attack and heart disease are among the most common causes of morbidity and mortality in the US (Heron 2007; Gluckman & Hanson, 2005; Centers for Disease Control and Prevention, 2014). In general, men tend to experience more life-threatening chronic diseases at younger ages, while women have higher rates of chronic debilitating conditions (Bird & Rieker, 2008). Across all age groups, heart disease is more prevalent among men than women, although it remains the leading cause of death for both genders (National Center for Health Statistics, 2009). Partly due to heart attack occurring at later ages for women, nearly half of all fatal heart attacks each year in the US occur in women. For men up to age 75, the incidence of stroke is higher than in women, but this trend reverses in adults 85 years and older (Petrea et al., 2009). Women also have a higher lifetime risk of stroke (Petrea et al., 2009). Gender differences do not appear with regard to the overall prevalence of hypertension (33.6% of men and 32.3% of women), but prevalence is higher for men under 25 (Domas, Papademetriou, Faselis & Kokkinos, 2013). Finally, women experience higher rates of arthritis than men (26% vs. 19%) while a slightly higher percentage of men have diabetes than women (14% vs. 11%) (Centers for Disease Control and Prevention, 2014).

Social explanations of differences in men and women's health outcomes have centred on differential access to protective resources, including income and education, as well as exposure to factors that negatively affect health, such as behavioral risk factors, in adulthood (Bird & Rieker, 2008). Life course research in both the United States and many European countries, however, has consistently linked each of these chronic disease outcomes to childhood socioeconomic circumstances (Hamil-Luker & O'Rand, 2007; Danese, Pariante, Caspi, Taylor & Poulton, 2007; Luo & Waite, 2005; Blackwell, Hayward, & Crimmins, 2001; Kivimaki et al., 2006; Maty, Lynch, Raghunathan & Kaplan, 2008; Mensah & Hobcraft, 2008; Johnson & Schoeni, 2011; Drakopoulos, Lakioti, & Theodossiou, 2011; McKenzie, Carter, Blakely & Ivoer, 2011). Cumulative dis/advantage is a key framework used to conceptualise this link, referring to a process through which initial disadvantage or advantage is compounded or amplified over time to produce heterogeneity in life course outcomes, such as health (O'Rand, 1996). In other words, the relationship between socioeconomic resources and health begins in early life and is magnified over time. Widening health disparities between advantaged and disadvantaged groups with age suggest that processes of cumulative dis/advantage operate across the life course (Lynch, 2003; Dupre, 2007; Willson et al., 2007; Shuey & Willson, 2014; Shuey & Willson, 2008; Brown, O'Rand, & Adkins, 2012).

Early life inequalities in socioeconomic environment are thought to initiate processes of cumulative advantage and disadvantage which lead to divergent trajectories of health across the life course (Dannefer, 2003). Research also indicates that the timing, duration, and sequencing of childhood exposure to economic hardship are critical for many adulthood outcomes, including health (Wagmiller, Lennon, Kuang, Alberti & Aber, 2006; Shuey & Willson, 2014). Existing models of cumulative dis/advantage, however, differentially emphasize the importance of each temporal complexity (see Shuey & Willson, 2014, for a review). Such approaches also do not take into account heterogeneity in childhood circumstances, ignoring the way in which socioeconomic circumstances can improve or deteriorate throughout childhood, as well as issues of timing related to the onset of disadvantage. Instability in resources in childhood and throughout the life course often occurs, challenging notions of disadvantage that view poverty as a long-term and irreversible state (McDonough & Berglund, 2003; McDonough, Sacker, & Wiggins,

2005; Western, Bloome, Sosnaud & Tach, 2012). Little attention has been given to patterns of change in childhood circumstances. Existing research has also relied heavily on retrospective data and static measures of childhood socioeconomic status (SES). Measures of childhood SES used in previous studies have included: parents' education (e.g. Walsemann et al., 2012; Lemelin et al., 2009; Bowen, 2010), parents' occupation (e.g. Pudrovska & Anikputa, 2013; Gustafsson & Hammarstrom, 2012; Hallqvist, Lynch, Bartley, Lang & Blane, 2004; Maty et al., 2008; Lidfelt, Li, Hu, Manson, & Kawachi, 2007., 2006), family income at a single point in childhood (e.g. Fothergill, Ensminger, Green, Robertson & Juon, 2009), or some combination of factors, such as receipt of welfare, parental divorce, and father's education (e.g. Schafer, Markus & Ferraro, 2012; Montez & Hayward, 2014).

While these studies have made key contributions to our understanding of life course processes of health, they are not able to address the effects of dynamic and differing experiences of economic hardship. For example, long-term exposure to childhood disadvantage appears to have the strongest negative effect on adult achievement outcomes and is harmful to health in adulthood (e.g., Shuey & Willson, 2014; Wagmiller et al., 2006). However, research also suggests that transitions into or out of sustained poverty in childhood have distinct effects on health. For example, deteriorating health in mid-life is more likely among those who transition into sustained economic hardship in childhood, while those whose families move out of poverty during childhood have health trajectories similar to those who never faced economic hardship (Shuey & Willson, 2014). Accordingly, the timing and duration of experiences of disadvantage in childhood are important to understanding life course trajectories of health. Yet studies tend to draw conclusions about long-term processes based on single snapshots in time (e.g. Pudrovska & Anikputa, 2013).

Although research demonstrates processes of cumulative advantage and disadvantage begin early in life, it should not be assumed that they operate similarly across groups given disparities in risks and opportunities associated the occupation of various social statuses (George, 2005). Little empirical attention has been given to whether cumulative processes of inequality that begin in childhood may differ for men



and women. Such differences are likely given gender differences in biological disease processes, responses to stressors and social conditions, and access to resources (Taylor et al. 2000; Zunzunegui, Alvarado, Béland & Vissandjee, 2008). For example, women earn less than men even after controlling for education, work experience, and marital status (Hogan & Perrucci, 2007), dominate temporary and part-time jobs (Prokos, Padavic, & Schmidt, 2009; Fuller & Vosko, 2008), and are more likely to experience discontinuity in their employment histories due to their role as primary caregiver (Moen, Robison, & Fields, 1994). Research that has incorporated gender into the study of childhood disadvantage and adult health suggests that childhood socioeconomic disadvantage predicts psychological distress, depressive symptoms, body mass index (BMI), cardiovascular disease, metabolic syndrome, diabetes and risk of heart attack for women significantly more than for men (Gilman, Kawachi, Fitzmaurice & Buka, 2002; Pudrovska & Anishkin, 2013; Walsemann et al., 2012; Lemelin et al., 2009; Lipowicz. Kozieł, Hulanicka & Kowalisko, 2007; Gustafsson & Hammarstrom, 2012; Maty et al., 2008; Hamil-Luker & O’Rand 2007). Pudrovska and Anikputa (2013) find evidence of an indirect relationship between early life SES and health through the operation of health behaviours for women only, though it should be noted that early life conditions are only measured at a single point in time. It is likely that cumulative processes differ by gender; however, research has not adequately problematised heterogeneity among women and men to understand how the timing and duration of childhood economic hardship generate health inequality within these groups.

Finally, in examining these processes, it is important to consider multiple measures of health rather than single or monolithic measures for two reasons. First, “different health conditions vary in their etiologies” (Brown et al., 2012, p. 360). Therefore, combining multiple health concerns into an all-encompassing measure risks overlooking the differential accumulation of risk factors that lead to different conditions. Second, the direction and magnitude of gender differences in health vary depending on the condition examined (Denton, Prus, & Walters, 2004). It is therefore useful to examine multiple health conditions in order to understand similarities and differences in the processes leading to each and to capture important variations by gender. Whereas past studies on cumulative disadvantage, gender and health have considered a single or limited number

of health outcomes, the present analysis examines multiple chronic diseases and compares how dynamic experiences of childhood economic hardship are related to each for men and women.

## 3.2 Research questions

Based on the above considerations, we ask whether trajectories of childhood economic hardship are associated with chronic disease outcomes in midlife for both women and men. We examine patterns of stability and change in childhood circumstances and their association with processes of cumulative health disadvantage for both women and men. Previous literature suggests childhood disadvantage may be more detrimental for women's health outcomes (e.g. Hamil-Luker & O'Rand, 2007), so it is possible that the relationship between trajectories of childhood economic hardship and some health outcomes will differ for men and women. We therefore investigate whether the observed relationships between childhood economic hardship and disease onset vary by the health outcome under investigation.

## 3.3 Methods

### 3.3.1 Data

This study uses the US Panel Study of Income Dynamics (PSID), an ongoing survey that began in 1968 with a nationally representative sample of 4,802 families (Panel Study of Income Dynamics, 2013). Information was collected on all household members, primarily from the household head, annually until 1997 when interviewing became biennial. In married families, the 'head' is the husband unless he is incapable of being interviewed and 'wife' is the female in a married or cohabitating couple (McGonagle, Schoeni, Sastry & Freedman, 2013). The 'head' can also refer to a single female. Annual response rates have ranged from 95 to 98 percent (McGonagle et al., 2013). Interviews are conducted via telephone. The latest wave of available data used in this analysis was collected in 2011. Children of PSID families who leave their parents' homes also become PSID family units, and sample representativeness has been maintained (McGonagle et al., 2013). The multi-generational design of data collection enables adult children to be linked to their parents. Data on childhood socioeconomic environment provided

prospectively by parents at the time the child was in the parental home avoids recall bias in childhood conditions. An oversample of families from low-income neighborhoods was included in the original sample design of the PSID, which enables the differentiation of various experiences of childhood economic hardship. Finally, the PSID contains rich information on various health outcomes, health behaviours, and other important covariates such as income, employment, and marital status. The PSID is one of the few large survey data sets worldwide that has followed multiple generations of families for such a long period of their lives, and as such provides a unique opportunity to prospectively examine the long-term health effects of childhood economic hardship.

### 3.3.2 Analytic sample

This study focuses on individuals who were newborn to eight years old in 1968. This age range is particularly useful for this analysis as, during the observation period, these individuals enter a stage of the life course in which many health problems begin to emerge. Latent classes of childhood economic hardship experience were estimated for the full sample of these respondents (N=4,167) using data collected from PSID families from 1968-1977 (see Shuey & Willson, 2014). The sample used in multivariate analyses includes the subsample of individuals who remained in the study in adulthood and were a PSID ‘head’ or a ‘wife’ at the start of the observation period in 1999 as these are the household members that the PSID collects detailed information on in each survey year (Number of individuals=1,229; 697 women, 532 men).

Missing data is a challenge in any longitudinal study. This paper uses survival analysis, which allows the use of unbalanced panels, meaning individuals who attrited from the PSID after the initial observation year (1999) are still included in the analysis.

Additionally, one advantage of the PSID is that, unlike retrospective studies, which do not begin studying individuals until much older ages, many selection processes are observable. Multiple studies have extensively examined the effects of the attrition of this cohort of children from the PSID sample on intergenerational models (e.g., those using family income during the respondent’s childhood) with covariates that predict adult health outcomes and demonstrate that the PSID maintains its representativeness over time without strong evidence of attrition bias, with the exception that the effect of higher

education on sample attrition is stronger than that of health and that female subsamples demonstrate weaker effects of attrition than males (Fitzgerald, 2011; Halliday, Kimmitt, & Kimmitt, 2012; Meer, Miller, & Rosen, 2003). Previous research also has found that individuals who experienced childhood poverty are less likely to have remained in the PSID to have an observed health outcome in 1999 when health data began to be collected (see Shuey & Willson, 2014). Any selective attrition with respect to health will likely lead to an underestimate of the impact of childhood economic hardship. Taken together, this indicates that, while not significantly biased, results from this study are likely conservative estimates of the association of childhood economic hardship and adult health (Shuey & Willson, 2014).

### 3.3.3 Measures

#### 3.3.3.1 Disease outcomes

Four disease outcomes are assessed in this study: high blood pressure, diabetes, arthritis, and a measure consisting of heart attack, heart disease, and stroke. Stroke, heart attack, and heart disease were grouped together due to relatively low prevalence levels in middle age in addition to all affecting the heart and circulatory system (Johnson & Schoeni 2011). The conditions are measured by responses to the question: “Has a doctor or health professional ever told you that you have had–?” Respondents were asked this question in each survey wave from 1999 to 2011. It is possible for individuals to have comorbidities, but each condition was examined separately and each measure included all those individuals who reported having been diagnosed with that particular health condition. It should be acknowledged that these measures are somewhat non-specific and the measure of arthritis does not distinguish between types of arthritis, which are experienced at different rates by men and women and have differing etiologies. Variation in the experience of arthritis could contribute to gender differences in association with childhood economic hardship, however we believe this is minimal given the similar rates of arthritis among the men and women in the sample. We further discuss the potential implications in the discussion section.

### 3.3.3.2 Childhood economic hardship

Histories of economic hardship in childhood were analyzed over a 10-year period, from 1968 (when the children were 0-8 years old) to 1977. A child was considered to be living in poverty in a given year if the family's total annual income fell below 125% of the official U.S. poverty threshold.<sup>3</sup> These indicators and repeated measures of latent class analysis were used to identify subgroups of individuals with similarities in their experience of economic hardship in childhood (see Shuey & Willson, 2014, for a detailed discussion). Based on fit statistics from the latent class models, and the previous literature (Wagmiller et al., 2006), it was determined that there were four groups into which respondents could be classified: non-poor, moving into poverty, moving out of poverty, and long-term poverty. Those who moved into poverty began with a relatively low risk of experiencing poverty, which increased as they reached and transitioned into adolescence, while those who moved out of poverty had a relatively high risk of poverty in early childhood that dropped steadily as they approached late childhood. The long-term poor had a very high probability of exposure to poverty during the entire period of observation (1968-1977), and the non-poor had a very low probability of experiencing poverty during this period.

### 3.3.3.3 Other covariates

Both adult resources and health behaviours are associated with childhood disadvantage and adult health (e.g. Hayward & Gorman, 2004; Pudrovska & Anishkin, 2013). All covariates belonging to these categories were included as time-varying, with the exception of education (coded as less than high school, high school, some post-secondary, and post-secondary in 1999). Employment status and marital status were dichotomised (1=employed; 1=married). Total household income was lagged by one year, adjusted for inflation, and logged for each year of observation. Based on considerations from previous literature (Kagotho, 2009; Hamil-Luker & O'Rand, 2007), frequency of

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<sup>3</sup> Consistent with previous literature using the PSID, 125% of the U.S. poverty threshold was used because the PSID consistently finds higher reported incomes than the Census Bureau (Wagmiller et al., 2006).

heavy physical activity was coded as 1=never engages in physical activity. Drinking and smoking were also dichotomised, with 1=drinks one or more drinks per day and 1=current smoker (see Kagotho, 2009). Race/ethnicity, which is strongly associated with both childhood poverty and adult health (Lynch, 2008; National Poverty Center, 2013), was also included as a covariate and coded as non-Hispanic black (1) and non-Hispanic white (0). Other racial/ethnic groups were not included due to an inadequate number of observations.

### 3.3.4 Analytic strategy

Survival analysis was used to determine how experiences of economic hardship in childhood affect the risk of disease onset in midlife. Multivariate analyses were clustered by person ID. The unit of analysis for all analyses was person-years ( $N=15,624$ ). Analyses were weighted using the PSID longitudinal weight to adjust for oversampling of low-income families as well as for attrition (McGonagle et al., 2013). The population at risk for each disease outcome was defined as those individuals who had not experienced disease onset for the particular condition under investigation before age 40. Thus, the models predict the likelihood that an individual would develop a condition by the end of the observation period assuming they did not have it by age 40. This restriction was imposed for two reasons: to address left-censoring, and also, because the focus of this study was to examine disease onset in mid-life.

Multivariate discrete-time hazard models were estimated using logistic regression. These models were appropriate given the fairly large intervals at which the presence of each disease was measured (years) as well as the censoring of some data. Women and men were analysed as separate groups. Such an approach provides greater ease with which to assess the significance of covariate effects within each group (Phillips & Sweeney, 2005). In addition, it allows us to assess how childhood economic hardship produces heterogeneous health outcomes within women and men, similar to Hamil-Luker and O’Rand (2007). This is particularly important considering our limited understanding of the unique health experiences of men and women who face economic hardship in childhood.

In analyses of temporal dependence, the risk of experiencing the onset of a chronic condition was found to change over time for both women and men; therefore, age was included in the multivariate models as a categorical variable: 40-45 (0) and 46-52 (1). These categories were chosen as they are reflective of a division between early and late middle-age. The proportional hazards assumption was also evaluated for women and men. This assumption implies that predictor variables have uniform effects across time, or that there are no interactions between predictors and time (see Allison, 2010; Borucka, 2013). The proportional hazards assumption was violated for women with regard to the effect of poverty class on health, indicating that an interaction term between age and poverty class was necessary for models of women's health to allow for non-proportional hazards (Allison, 2010; Borucka, 2013). The assumption was not violated for men; therefore, models predicting men's outcomes did not include the interaction terms. In other words, the effect of childhood economic hardship on disease onset changes over time for women but not men. This prevents testing for significant differences across the two groups as the model specification for women and men is different; however, the focus of this paper is to examine whether and how childhood economic hardship produces heterogeneous health outcomes among women and among men. Results will demonstrate how the timing and duration of childhood economic hardship impacts the health of women and men.

## 3.4 Results

### 3.4.1 Descriptive results

Weighted proportions and means by gender are presented in table 3.1. On average, over the observation period, the same percentage of women and men reported having diabetes (5%) and stroke, heart disease, or heart attack (3%). In any given year, about 8% of men reported arthritis compared to 12% of women. Over the observation period, men and women experienced similar rates of high blood pressure, at 17% and 16%, respectively. Rates of childhood economic hardship were roughly similar across women and men. High school graduates make up the largest proportion of education categories for both men and women (39% and 34%, respectively). More men (43%) than women (25%) reported drinking one or more drinks per day and more women (33%) than men (25%)

reported never exercising. Smoking rates were similar at 25% for women and 23% for men.



**Table 3.1 Descriptive statistics by gender (weighted), 1999-2011 PSID**

Variable	Women	Men
<i>Diabetes</i>		
Yes	0.05	0.05
No	0.95	0.95
<i>High blood pressure</i>		
Yes	0.16	0.17
No	0.84	0.83
<i>Arthritis</i>		
Yes	0.12	0.08
No	0.88	0.82
<i>Stroke, heart disease, heart attack</i>		
Yes	0.03	0.03
No	0.97	0.97
<i>Childhood poverty status</i>		
Non-poor	0.75	0.75
Move into poverty	0.05	0.04
Long-term poor	0.10	0.08
Move out of poverty	0.09	0.13
<i>Race/Ethnicity</i>		
Non-Hispanic Black	0.27	0.20
Non-Hispanic White	0.73	0.80
<i>Age</i>	41.37	41.27
<i>Adult education</i>		
<High school	0.10	0.07
High school	0.34	0.39
Some post-secondary	0.29	0.22
Post-secondary	0.27	0.32
<i>Employment status</i>		
Employed	0.22	0.90
Not employed	0.78	0.10
<i>Marital status</i>		
Married	0.66	0.76
Not married	0.34	0.24
<i>Income (median)</i>	60851	78087
<i>Smoking status</i>		
Yes	0.25	0.23
<i>Drinking frequency</i>		
1+/Day	0.25	0.43
<i>Physical activity</i>		
Never engages	0.33	0.25
<i>N</i>	697	532
<i>N(person-years)</i>	8848	6776

Notes: Proportions for disease outcomes refer to the average proportion in each category over the observation period. Data were converted into person-year format required for survival analysis.

### 3.4.2 Multivariate analyses

In all models for women, the reference group is women in early midlife (aged 40-45) who did not experience economic hardship in childhood. The comparison group is women in late midlife (aged 46-52) who have experienced some form of poverty.

#### 3.4.2.1 Diabetes (table 3.2)

In model 1, women in late midlife who experienced long-term poverty in childhood were approximately eight times as likely to experience the onset of diabetes by the end of the observation period compared to their younger counterparts who did not experience poverty ( $p=0.006$ ). In addition, older women who moved out of poverty in late childhood were still 16 times as likely to experience the onset of diabetes than younger women who did not experience poverty ( $p=0.022$ ). Conversely, younger women were less likely to experience diabetes even if they experienced long-term poverty as children ( $OR=0.20$ ;  $p=0.026$ ). With the introduction of adult resources, health behaviours and other covariates, women belonging to both of these poverty classes remained more likely to experience the onset of diabetes by the end of the observation period relative to younger women who did not experience poverty ( $OR=9.01$ ,  $p=0.007$ ;  $OR=12.97$ ,  $p=0.032$ ). Stated as probability, women who experienced long-term poverty had a 90% chance of developing diabetes by the end of the observation period and women who moved out of poverty had a 93% chance. For women, then, the effects of childhood economic hardship on diabetes onset in midlife vary by age, with the impact of long-term poverty and poverty in early childhood manifesting in late middle age. Conversely, childhood economic hardship was not a significant predictor of diabetes onset for men.

**Table 3.2 Discrete-time logistic regression estimated effects of childhood economic hardship on the risk of onset of diabetes within 12 years, by gender: 1999-2011 PSID**

Independent variable	Model 1		Model 2 <sup>a</sup>		Model 3 <sup>b</sup>		Model 4 <sup>c</sup>									
	Women	Men	Women	Men	Women	Men	Women	Men								
	Odds ratio	p-value	Odds ratio	p-value	Odds ratio	p-value	Odds ratio	p-value								
<i>Childhood economic hardship (non-poor)</i>																
Move into poverty	0.92	0.889	2.15	0.285	1.17	0.819	1.75	0.371	0.94	0.929	1.83	0.350	0.70	0.652	1.54	0.433
Long-term poverty	0.20	0.026	0.76	0.289	0.17	0.019	0.73	0.422	0.15	0.011	0.72	0.401	0.10	0.007	0.49	0.168
Move out of poverty	0.15	0.081	2.21	0.279	0.15	0.126	1.78	0.429	0.13	0.091	1.63	0.518	0.10	0.051	1.29	0.718
<i>Age (40-45)</i>																
46-52	2.04	0.096	7.42	0.000	2.44	0.045	8.90	0.000	2.51	0.076	9.01	0.000	2.57	0.062	9.82	0.000
<i>Childhood economic hardship X age</i>																
Move into poverty X 46-52	1.06	0.921			0.76	0.688			1.02	0.982			0.90	0.887		
Long-term poverty X 46-52	8.17	0.006			8.43	0.006			9.07	0.008			9.01	0.007		
Move out of poverty X 46-52	15.67	0.022			12.37	0.034			11.88	0.038			12.97	0.032		
Constant	0.29	0.002	0.12	0.000	19.75	0.190	0.00	0.002	6.27	0.465	0.00	0.002	6.77	0.437	0.00	0.002

Notes:

Number of observations (women) = 810. Number of observations (men) = 594.

<sup>a</sup> Model 2 controls for adult resources: education, income, employment status, and marital status.

<sup>b</sup> Model 3 controls for the variables specified in Model 2 and adds adult health behaviours: smoking, drinking, and physical activity.

<sup>c</sup> Model 4 controls for the variables specified in Models 2 and 3 and adds race/ethnicity.

**Table 3.3 Discrete-time logistic regression estimated effects of childhood economic hardship on the risk of onset of high blood pressure within 12 years, by gender: 1999-2011 PSID**

Independent variable	Model 1		Model 2 <sup>a</sup>		Model 3 <sup>b</sup>		Model 4 <sup>c</sup>									
	Women	Men	Women	Men	Women	Men	Women	Men								
	Odds ratio	p-value	Odds ratio	p-value	Odds ratio	p-value	Odds ratio	p-value								
<i>Childhood economic hardship (Non-poor)</i>																
Move into poverty	1.13	0.758	1.52	0.280	1.22	0.635	1.65	0.200	1.40	0.412	1.63	0.235	1.50	0.321	1.66	0.228
Long-term poverty	0.65	0.108	0.97	0.861	0.72	0.259	0.98	0.937	0.78	0.396	1.01	0.953	0.85	0.589	0.95	0.838
Move out of poverty	0.59	0.192	0.95	0.808	0.68	0.340	1.03	0.873	0.70	0.392	1.03	0.891	0.76	0.504	0.96	0.897
Age (40-45)																
46-52	5.90	0.000	7.69	0.000	6.85	0.000	8.30	0.000	6.65	0.000	8.17	0.000	6.70	0.000	8.17	0.000
<i>Childhood economic hardship X age</i>																
Move into poverty X 46-52	0.62	0.289			0.53	0.170			0.55	0.191			0.55	0.194		
Long-term Poverty X 46-52	1.51	0.191			1.31	0.394			1.34	0.366			1.33	0.379		
Move out of poverty X 46-52	1.81	0.202			1.52	0.380			1.65	0.294			1.62	0.310		
Constant	0.17	0.000	0.12	0.000	0.20	0.082	0.17	0.049	0.26	0.147	0.17	0.062	0.30	0.225	0.15	0.055

Notes:

Number of observations (women) = 2592. Number of observations (men) = 2020.

<sup>a</sup> Model 2 controls for adult resources: education, income, employment status, and marital status.

<sup>b</sup> Model 3 controls for the variables specified in Model 2 and adds adult health behaviours: smoking, drinking, and physical activity.

<sup>c</sup> Model 4 controls for the variables specified in Models 2 and 3 and adds race/ethnicity.

### 3.4.3 High blood pressure (table 3.3)

Childhood economic hardship was not a significant predictor of the midlife onset of high blood pressure for women or men in any of the four models.

### 3.4.4 Arthritis (table 3.4)

In model 1, women in late middle-age who moved out of poverty were about six times as likely to experience arthritis compared to their younger peers who did not experience childhood poverty ( $p=0.006$ ). This relationship persisted when adult resources, health behaviours, and race/ethnicity were taken into account ( $OR=7.75$ ,  $p=0.011$  in Model 4). These women had an 89% chance of developing arthritis. For men, however, childhood economic hardship was not a significant predictor of arthritis onset in midlife.

### 3.4.5 Stroke, heart disease, heart attack (table 3.5)

In model 1, women in late midlife who lived in long-term poverty as children were about six times as likely to experience stroke, heart disease, or a heart attack compared to their younger peers who did not experience childhood poverty ( $p=0.001$ ). This relationship remained the same controlling for adult resources, health behaviours, and race/ethnicity ( $OR=6.02$ ,  $p=0.002$ ; Model 4). That is, these women had an 86% probability of developing stroke, heart disease, or heart attack. For men, having moved into poverty in childhood was associated with being three times more likely to experience stroke, heart disease, or a heart attack by the end of the observation period ( $p=0.004$ ; Model 1). With introduction of adult resources in model 2, this increased to four times as likely ( $p=0.001$ ). When health behaviours were added in model 3, men who moved into poverty as children were nearly six times as likely to experience stroke, heart disease, or a heart attack compared to their non-poor peers ( $p=0.001$ ). This remained true in model 4.

**Table 3.4 Discrete-time logistic regression estimated effects of childhood economic hardship on the risk of onset of arthritis within 12 years, by gender: 1999-2011 PSID**

Independent variable	Model 1		Model 2 <sup>a</sup>		Model 3 <sup>b</sup>		Model 4 <sup>c</sup>									
	Women	Men	Women	Men	Women	Men	Women	Men								
	Odds ratio	p-value	Odds ratio	p-value	Odds ratio	p-value	Odds ratio	p-value								
<i>Childhood economic hardship (Non-poor)</i>																
Move into poverty	0.35	0.085	0.82	0.535	0.30	0.043	0.64	0.233	0.29	0.034	0.67	0.340	0.31	0.053	0.66	0.297
Long-term poverty	0.55	0.066	0.77	0.268	0.49	0.033	0.65	0.193	0.48	0.030	0.65	0.219	0.54	0.105	0.47	0.108
Move out of poverty	0.19	0.005	1.62	0.203	0.18	0.008	1.39	0.435	0.17	0.008	1.47	0.357	0.19	0.011	1.07	0.901
Age (40-45)																
46-52	4.03	0.000	6.07	0.000	4.38	0.000	6.40	0.000	4.60	0.000	6.43	0.000	4.57	0.000	6.62	0.000
<i>Childhood economic hardship X age</i>																
Move into poverty X 46-52	2.56	0.167			2.57	0.162			2.49	0.174			2.58	0.151		
Long-term poverty X 46-52	1.63	0.209			1.46	0.329			1.45	0.333			1.48	0.307		
Move out of poverty X 46-52	6.18	0.006			5.55	0.011			5.53	0.012			5.75	0.011		
Constant	0.27	0.000	0.15	0.000	1.11	0.931	0.30	0.481	1.03	0.981	0.18	0.330	1.39	0.804	0.18	0.330

Notes:

Number of observations (women) = 1944. Number of observations (men) = 950

<sup>a</sup> Model 2 controls for adult resources: education, income, employment status, and marital status.

<sup>b</sup> Model 3 controls for the variables specified in Model 2 and adds adult health behaviours: smoking, drinking, and physical activity.

<sup>c</sup> Model 4 controls for the variables specified in Models 2 and 3 and adds race/ethnicity.

**Table 3.5 Discrete-time logistic regression estimated effects of childhood economic hardship on the risk of onset of stroke, heart disease, heart attack within 12 years, by gender: 1999-2011 PSID**

Independent variable	Model 1		Model 2 <sup>a</sup>		Model 3 <sup>b</sup>		Model 4 <sup>c</sup>									
	Women	Men	Women	Men	Women	Men	Women	Men								
	Odds ratio	p-value	Odds ratio	p-value	Odds ratio	p-value	Odds ratio	p-value								
<i>Childhood economic hardship (Non-poor)</i>																
Move into poverty	1.35	0.534	2.92	0.004	1.43	0.513	4.03	0.001	1.99	0.215	5.83	0.001	2.03	0.217	5.99	0.000
Long-term poverty	0.33	0.022	0.73	0.399	0.32	0.053	0.43	0.146	0.39	0.110	0.44	0.158	0.40	0.173	0.14	0.000
Move out of poverty	0.71	0.421	1.06	0.915	0.76	0.540	1.09	0.864	0.76	0.542	1.05	0.928	0.78	0.646	0.44	0.18
Age (40-45) 46-52	2.13	0.015	4.52	0.000	2.35	0.008	6.21	0.000	2.42	0.012	5.95	0.000	2.40	0.013	8.91	0.000
<i>Childhood economic hardship X age</i>																
Move into poverty X 46-52	1.02	0.986			1.02	0.985			0.78	0.793			0.79	0.808		
Long-term poverty X 46-52	6.07	0.001			5.80	0.002			5.98	0.002			6.02	0.002		
Move out of poverty X 46-52	2.71	0.093			2.62	0.143			3.24	0.102			3.24	1.00		
Constant	0.34	0.000	0.23	0.000	3.08	0.532	1.43	0.883	5.92	0.279	25.08	0.285	6.13	0.302	15.91	0.353

Notes:

Number of observations (women) = 486. Number of observations (men) = 356.

<sup>a</sup> Model 2 controls for adult resources: education, income, employment status, and marital status.

<sup>b</sup> Model 3 controls for the variables specified in Model 2 and adds adult health behaviours: smoking, drinking, and physical activity.

<sup>c</sup> Model 4 controls for the variables specified in Models 2 and 3 and adds race/ethnicity.

### 3.5 Discussion

While studies of gender and health have typically focused on ‘explaining away’ the gender difference, we take a different approach to this commonly investigated phenomenon through examining how patterns of change and stability in childhood economic hardship initiate processes of disadvantage in health among women and men rather than between them. Although men and women in this study experienced chronic disease at similar rates, the cumulative processes leading to heterogeneity within each group were quite different. Consistent with previous US and European literature, in this analysis childhood economic hardship differentiated between women at low and high risk of chronic disease in midlife but not men (e.g. Walsemann et al., 2012; Lemelin et al., 2009; Lipowicz et al., 2007; Gustafsson & Hammarstrom, 2012). One exception was stroke, heart disease, and heart attack, where childhood economic hardship increased risk of onset for both men and women. This inconsistency with previous research could be the result of a more nuanced and prospective measure of childhood economic hardship than was used in previous studies (e.g. Hamil-Luker & O’Rand, 2007).

Overall, our findings indicate that it is not just long-term poverty that matters for women’s health outcomes, but poverty may have lasting effects even after leaving it. For example, long-term poverty in childhood significantly predicted women’s risk of onset for diabetes and stroke, heart disease, and heart attack. In addition, women who began life in poverty but moved out of poverty in childhood also were more likely than those who were never in poverty to experience diabetes onset in late midlife, as well as arthritis. These findings reveal the importance of measuring childhood poverty as dynamic rather than capturing it at a single point in time or as a retrospective global measure. Research on the impact of SES in early life on later life health that relies on one measure of parents’ SES (e.g. Beebe-Dimmer et al., 2004), and retrospective accounts of childhood SES, which increase the likelihood of recall bias may underestimate the effects of childhood context (e.g. Galobardes et al., 2004). Thus, while research in many Western countries finds disparities in adult health outcomes to be linked to childhood circumstances (e.g. Kivimaki et al., 2006; Mensah & Hobcraft, 2008; Johnson & Schoeni, 2011; Drakopoulos, Lakioti, & Theodossiou, 2011; McKenzie et al., 2011; Gustafsson &



Hammarström, 2012), our study provides support for a more nuanced and dynamic conceptualisation and measurement of childhood poverty with implications for how we understand processes of status and health.

These analyses demonstrate that women's health in later midlife is more sensitive to the timing and duration of childhood economic hardship than men's. It should be noted that findings remained significant and odds ratios remained about the same even after adjustment for covariates. This may be indicative of a direct effect of childhood poverty, as hypothesised by other research (e.g. Diprete & Elrich, 2006). Nevertheless, future research should continue to examine the mechanisms through which childhood poverty affects adult health as this was beyond the scope of this paper. Potential pathways through which childhood disadvantage affects later life health include negative changes in physiology and metabolism in utero (DeBoo & Harding, 2006), disruptions to biological functioning (Miller, Chen & Parker, 2011), increased stress levels (Miech & Shanahan, 2000), and continuous exposure to health-compromising circumstances as a result of disadvantage (Willson, Shuey & Elder, 2007).

Socioeconomic conditions in childhood may be more detrimental to women's health outcomes, in particular, because they are less likely to experience social mobility over the life course than men (Walsemann et al., 2012). In other words, the impact of childhood disadvantage on health is eventually less important for men because of their greater resource attainment in adulthood. Childhood adversity is also associated with reduced accumulation of life course capital, and this relationship is stronger for women (Hamil-Luker & O'Rand, 2007). Additionally, qualitative research suggests a greater accumulation of adversity over the life course for women than men (deVries & Watt, 1996). Disadvantage experienced in childhood may therefore continue to accumulate for women over the life course based on the structuring of opportunities and life chances by gender (Hunt & Annandale, 1999). For example, through disadvantages in paid and unpaid labour, discriminatory experiences, stress, and caregiving burdens, the impact of childhood economic hardship on health may be aggravated (Turner, Wheaton & Lloyd, 1995; Turner & Avison, 2003; Lundberg & Parr, 2000; Lundberg, 1996; Coltrane, 2000). While gender differences in health are consistent across many European countries and the

US (Crimmins, Kim, & Sole-Auro, 2010), the relationship between socioeconomic resources, gender and health can vary by welfare regime (Bambra et al., 2009). Due to the unique nature of the social welfare and health care systems in the US, it is possible that the association between childhood economic hardship and adult health found in this analysis may be more pronounced than would be found in countries where SES and health are not as strongly linked. Future research should continue to assess pathways through which heterogeneous trajectories of childhood economic hardship are associated with health among women.

As expected, the observed relationships between childhood economic hardship and health for both men and women also depended on the health outcome examined. For example, women who began childhood with a high risk of exposure to poverty but moved out of poverty as they reached adolescence were more likely to have arthritis in late midlife; in contrast, long-term poverty was most consequential for women's heart disease outcomes. For men, a move in to poverty in childhood predicted stroke, heart disease, and heart attack in midlife, but childhood economic hardship was not a significant predictor of men's other health outcomes. These findings are not surprising given that different health outcomes often have different etiologies (Brown et al., 2012), and also provide support for the dynamic measurement of childhood economic hardship in future research given the nuanced effects on health that emerge (Shuey & Willson, 2014).

This study has several limitations. First, as in any longitudinal analysis that covers a large span of time, there is the potential for unobserved heterogeneity resulting from panel attrition; although there is comprehensive evidence that suggests that the observed relationships were not seriously biased due to attrition, they were potentially weakened. Second, childhood disadvantage was conceptualized and measured as economic hardship based on household income. Future research may also consider such experiences as change in family structure. Based on available disease measures, we were also unable to account for different types of particular diseases, which is most relevant for the measure of arthritis (e.g. osteo vs. rheumatoid arthritis); however, analyses were stratified by gender and disease given known gender differences in patterns of disease. Research demonstrates that women more often experience acute and chronic conditions while men

experience more life-threatening disease (Bird & Rieker, 2008). Not surprisingly, we find childhood disadvantage to be associated with diabetes and arthritis for women, and stroke, heart disease, and heart attack for men. Further, men and women in the sample actually reported similar rates of these diseases. The disease measures used were also self-reports of doctor diagnoses. Research demonstrates that lower income individuals are less likely to regularly seek care or visit a doctor (Dubay & Lebrun, 2012), meaning results may be conservative. Finally, this study considers the onset of four physical disease outcomes. Future research should also examine other physical and mental health outcomes to determine whether the observed relationships hold.

Despite these limitations, this study is the first to examine whether the timing and duration of childhood exposure to economic hardship generates a process of cumulative disadvantage in health for both men and women. It has demonstrated the importance of measuring poverty as dynamic rather than static in that long-term and an initial high risk of childhood poverty appeared to be more consequential than other experiences of childhood economic hardship for women. Indeed, when childhood economic hardship is measured as dynamic, nuances emerge that have not been captured by other studies. Although this study is unable to concretely determine why different experiences of economic hardship in childhood matter for different disease outcomes, it clearly demonstrates that change and stability in childhood socioeconomic circumstances matter. Further, the link between childhood economic hardship and long-term negative health consequences may be more relevant to women's health over the life course.

Rather than focusing on how socioeconomic circumstances in adulthood explain differences in health between men and women, we focus on how childhood adversity differentiates risk of onset within each group. Instead of simply controlling for gender, we explore the unique patterns of cumulative disadvantage among women and men. In so doing, we not only find childhood economic hardship produces heterogeneity in women's chronic disease outcomes (Hamil-Luker & O'Rand, 2007), but also, that the impact of childhood poverty varies by age for women. Little empirical research has examined whether the process of cumulative disadvantage is the same across different sub-groups of the population over time or when the effects of childhood economic hardship may

emerge for particular groups. This study suggests that cumulative disadvantage may be a gendered process, with age-dependent effects and heterogeneous health outcomes generally emerging for women, but not for men.

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## Chapter 4

### 4 Race, gender, and the resources that matter: An investigation of intersectionality and health<sup>4</sup>

Both psychosocial and socioeconomic resources have been found to promote physical and mental health (Pearlin 1989; Thoits 2010; Phelan, Link, and Tehranifar 2010). Although it is well-established that racial differences exist in access to and levels of these resources, limited empirical investigation has examined their role in racial disparities in health among women. Indeed, research has long demonstrated the health disadvantage faced by African Americans (see Lynch 2008), but has only recently begun to consider the implications of occupying multiple disadvantaged statuses to health (e.g. Ajrouch et al. 2010). Attempts to capture the effect of occupying multiple disadvantaged statuses on health have also failed to examine racial differences among women adequately. Instead, race and gender have just been “controlled for” or “explained away” (Evans-Campbell et al. 2010: 177).

This study aimed to move beyond the use of intersectionality as a “theoretical buzzword” (Perry, Harp and Oser 2013: 25) and implement it methodologically in the study of health. This was accomplished through examining racial differences in health among women and then through subsequent analysis of health within groups of white women and black women. The role of both psychosocial and material resources was examined across self-rated health and chronic conditions, two frequently used measures in social science research. Factors related to black and white women’s psychosocial resources were first examined to detect possible differences in their operation as mechanisms related to health disparities. Ultimately, this research aims to advance our understanding of racial disparities in health as they intersect with gender through a comprehensive examination of the resources that matter.

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<sup>4</sup> **Etherington, N.** (2015). Race, gender, and the resources that matter: An investigation of intersectionality and health. *Women & Health*, 55(7), 754-777.

## 4.1 Race, gender, and health

While interest in race and health certainly has been a focus of social science and epidemiological research, the relationship between race and health for women is not as well understood. In addition, mechanisms that produce health disparities between white women and black women have been under-researched. Extant research on race, gender, and health has demonstrated that women who belong to disadvantaged racial and ethnic groups are more likely to experience poor health (Committee on Health Care 2005). Black women, in particular, appear to be at the greatest health disadvantage. Indeed, relative to their white counterparts, black women have experienced a gap in life expectancy that begins at birth and persists throughout adulthood (U.S. Department of Health 2006; Adams et al. 2007), with white women's life expectancy at birth exceeding that of black women by 5.2 years (Bharmal et al. 2011). Across all health indicators, black women are disadvantaged relative to whites. For example, black women are more likely to be in an advanced stage of cancer at the time of diagnosis than whites (Williams 2002) and have a higher breast cancer mortality rate than white women despite lower breast cancer incidence (Committee on Health Care 2005). Hypertension and heart disease mortality rates have also been found to be highest for black women (National Center for Health Statistics 2000; Committee on Health Care 2005). In addition, rates of stress-related health problems, such as cardiovascular disease, adverse birth outcomes, and cerebrovascular disease are disproportionately high among African American women (Office on Women's Health 2006).

Although it is clear that African American women in particular experience disproportionately higher rates of morbidity and mortality than individuals who are not racial/ethnic minorities, few studies have examined whether resources protective to health have different associations for white and black women. Further, what is known about black women generally comes from studies of differences in health between men and women rather than studies reflecting the diversity between different groups of women (e.g. Fitzpatrick and Van Tran 1997; Erving 2011) and/or from studies based on discrimination-related stress rather than physical health outcomes (e.g. Greer 2011; Perry, Harp, and Oser 2013). Existing knowledge is also limited by the consideration of only

one health outcome, such as mental health (e.g. Spence, Adkins, and Dupre 2011), as well as the failure to consider psychosocial resources in addition to material resources as key sources of health inequality (e.g. Grote et al. 2007). Because disparities among white and black women could be due to different levels of resources or different returns to resources, it is important to expand upon the way in which these relationships have been previously investigated.

## 4.2 Psychosocial resources and health

While personal resources such as self-efficacy, social support, and self-esteem have been deemed essential mediators in the relationship between social characteristics and health outcomes (Turner 2010), research is limited as to how these mechanisms function across women of different races. Self-efficacy is defined as “the perception of oneself as a causal agent in one’s environment, as having some control over one’s circumstances, and being capable of carrying out actions to produce intended effects” (Gekas 2003: 370). Research has shown that self-efficacy, or mastery, is strongly related to overall physical health (Caputo 2003; Kosteniuk and Dickinson 2003; Pudrovska et al. 2005). It is also significantly related to cardiovascular health and inversely related to blood pressure (Russek et al. 1990). Self-efficacy may affect health through its association with a healthy lifestyle (Mirowsky and Ross 2003). People who feel in control of their own lives are more likely to seek and practice a lifestyle that promotes health. Greater self-efficacy is also postulated to increase individuals’ capacity to solve health-related problems. Finally, self-efficacy is thought to protect physical and mental health against economic hardship, amongst other adversities (Pudrovska et al. 2005).

While self-efficacy captures one’s sense of control over life circumstances, self-esteem refers to one’s overall evaluation of one’s worth (Blascovich and Tomaka 1991). Research has also demonstrated a consistent relationship between self-esteem and physical health (Gidron et al. 2006). Individuals with high self-esteem may experience less stress, demonstrate adaptive coping behaviors, and to seek and obtain more social support, all of which enhance health (Orth, Robins, and Widaman 2012). Social support itself is considered a very important resource that influences health outcomes as it is associated with positive psychological well-being (Ajrouch et al. 2010) and physical

health (Mirowsky and Ross 2003; Uchino 2009). Social relationships can also promote psychological resources and enhance individuals' coping mechanisms, which influence health directly and indirectly (Uchino 2009; Thoits 2010). While personal resources such as self-efficacy, social support, and self-esteem have been deemed essential mediators in the relationship between social characteristics and health outcomes (Turner 2010), little research exists on how these mechanisms function for women of different races.

The association between psychosocial resources and health may vary by individual characteristics, such as race and gender (House 2002), but limited empirical investigation is available in this regard, especially concerning the intersection of these characteristics. Research has suggested that disadvantaged individuals have lower levels of self-efficacy, self-esteem, and social support, placing them at even greater risk of developing physical and mental health problems (Thoits 2010). For example, women generally experience lower levels of self-esteem and self-efficacy than men (Thoits 1995). Interestingly, blacks have relatively high self-esteem, either equal to or greater than that of whites, but have lower levels of self-efficacy (Mirowsky and Ross 1990; Schieman 2001; Erol and Orth 2011). Also, self-efficacy may be of particular importance in women's health outcomes (Longest and Thoits 2012), and black men have higher levels of self-efficacy than black women (Erving 2011). It is less clear how these resources may vary for women by race. Social support is also absent in this research, particularly concerning physical health outcomes, but may be especially important for black women (Lin, Thompson, and Kaslow 2009; Spence, Adkins, and Dupre 2011).

Given the importance of psychosocial resources for health, and that they may be especially relevant to women who are racial minorities, it is critical to examine these relationships. It is also necessary to identify factors that may promote these protective resources among white and black women, such as higher education, which is associated with greater self-efficacy, self-esteem, and social support (Mirowsky and Ross 2003). In fact, research has postulated that it is education's promotion of psychosocial well-being that accounts for its robust relationship with health, above and beyond its association with other resources, such as income and employment (Mirowsky and Ross 2003). Research also suggests, however, that in addition to education, employment may be particularly



important for women's mastery and self-esteem (Schieman 2002). The positive effects of autonomy and non-routinized work have been more beneficial to women's self-concept than men's. It is yet to be determined whether education and employment equally benefit the psychosocial resources of white and black women. For this reason, the relations of both employment status and education to women's psychosocial outcomes were examined.

### 4.3 Socioeconomic status and health

Education in particular is an important component of socioeconomic status (SES) and its relationship with health. According to the theory of resource substitution, those with the fewest alternative resources benefit the most from the acquisition of education (Ross and Mirowsky 2006), as socioeconomic disadvantage makes these individuals more dependent on education to attain well-being. However, blacks do not experience the same level of health benefits from higher education as whites (Shuey and Willson 2008). It remains an open question whether black women experience the same psychosocial or health gains of higher education as white women.

The association between SES and health is certainly complicated by the intersection of race and gender. The debate has been longstanding as to whether racial disparities in health are a reflection of socioeconomic inequalities (Evans-Campbell et al. 2007). While the evidence is mixed, it does seem to be the case that SES differences in health are larger within racial groups than between them, and racial differences persist within socioeconomic strata (Williams 2002). Similarly, research on gender and health often attributes gender differences to differential access to resources but simultaneously finds that these differences persist within SES groups (see Bird and Rieker 2008). In each case, it is clear that SES does not entirely explain health disparities within these groups. This necessitates investigations of health that also take into account other types of resources, and further, investigation of how SES shapes health outcomes within groups as well as between them.

In sum, gender, race, and SES may all work in concert to shape health outcomes, and this is a process that can be better understood through examining the interaction of race and gender. The following hypotheses are examined:

H1: (a) Higher education and employment are positively associated with women's psychosocial resources; (b) Black women have lower levels of psychosocial resources than white women; (c) Black women do not experience the same psychosocial benefits to higher socioeconomic resources as white women.

H2: (a) Higher education and psychosocial resources are associated with good health and decreased likelihood of chronic conditions for women, after adjustment for covariates; (b) White women have both better self-rated health and fewer chronic conditions than black women, adjusting for confounding variables; (c) Psychosocial and material resources are not associated with better self-reported health in black women as much as in white women.

#### 4.4 Data and methods

This study used data from the 2007 wave of the U.S. Panel Study of Income Dynamics (PSID), Child Development Supplement (CDS) (PSID, 2013). Data collection for the PSID began in 1968 by the Survey Research Center, Institute for Social Research, at the University of Michigan, with a nationally representative probability sample of 18,000 individuals in 5,000 households. The initial PSID sample consisted of two independent samples: a national sample of the civilian noninstitutional population of the U.S. population and an oversample of low-income families drawn from the Survey of Economic Opportunity. Sample members were followed each year until 1997 when interviews became biennial. The survey was administered using computer-assisted telephone interviews by trained interviews at the University of Michigan. In 1997, the PSID supplemented main data collection with additional data on parents and their children. All sample members who had 0-12 year old children were eligible for this supplemental data collection. The CDS contains some information on primary caregivers not found in the main PSID, such as measures of self-efficacy, self-esteem, and social support. Approximately 95% of the primary caregivers interviewed were the biological

mothers of the children in the CDS (Institute for Social Research 2012). These women were selected for the analytic sample for the present study as they could be matched with the demographic and socioeconomic information contained in the main PSID. Thus, this study is generalizable to women with children. The response rate was quite high at 96 percent. A total of 1,111 biological mothers, aged 26 to 68 years, were included in the 2007 wave of the survey. Respondents who were missing data on sociodemographic and health (n=155) were excluded from the analysis. Because the focus of this study was on non-Hispanic whites and non-Hispanic blacks, those of other race/ethnicities were also dropped (n=87). Thus, the analytic sample for this study consisted of 869 women.

#### 4.4.1 Dependent variables

##### 4.4.1.1 Psychosocial variables

To test Hypothesis 1, self-efficacy, self-esteem, and social support were the outcomes of interest. Self-efficacy was measured using Pearlin et al.'s (1981) mastery scale. The score was calculated based on respondents' answers to questions of whether they feel in control, helpless, pushed around, or cannot solve problems. For each question, respondents indicated how strongly they agreed or disagreed on a scale of one to four. The average of these responses was then calculated to provide the self-efficacy score, which also ranges from one to four. Self-esteem was assessed using the Rosenberg (1986) Self-Esteem Scale. Respondents answered 10 questions concerning how much they agree with statements of self-worth, such as "On the whole, I am satisfied with myself" and "I certainly feel useless at times." Responses were averaged to form a score of one to four. An index of social support was generated from respondents' answers regarding how satisfied they felt with the practical and emotional support they receive from their friends and from their family. A higher score indicates a higher sense of social support through greater satisfaction with the help received. Given that social support can be instrumental or emotional (Ajrouch et al. 2010), it is essential to include both dimensions as well as different sources from which such help can arise. Spousal support was not included as not all women in the sample were married or in a relationship, however, marital status is included in the analyses as a control variable, coded as married (0), single (1), or divorced/separated (2).

#### 4.4.1.2 Health variables

In the second set of analyses, health was the outcome of interest, and two health variables were used: self-rated health and chronic illness. Self-rated health was based on responses to the question, “Would you say your health in general is excellent, very good, good, fair, or poor?” Responses were recoded into four ordered categories, with 0 being the lowest category (fair/poor) and 3 being the highest category (excellent). Fair and poor responses were combined to increase statistical power. Despite its subjective nature, self-rated health has been found to be a highly reliable measure of health status (see Idler and Benyamini 1997 for a review). Chronic illness was measured by report of the presence of a chronic condition. Respondents in the survey were asked whether they had hypertension, cancer, heart disease, diabetes, arthritis, or another chronic condition. These responses were combined into one measure of whether a chronic condition was present.

#### 4.4.2 Key explanatory variables

In models assessing psychosocial variables, key explanatory variables were race, respondent’s education, and employment status. Respondents’ race was a dummy variable for which 1 indicated non-Hispanic Black, and 0 indicated non-Hispanic white. Education was included as a dummy variable with 0 = less than high school, 1 = high school, 2 = some post-secondary, and 3 = post-secondary. Finally, employment status was included as a dummy variable with 0 = employed, 1 = unemployed, and 2 = homemaker. Key variables included in models of health were the psychosocial variables of self-efficacy, self-esteem, and social support.

#### 4.4.3 Control variables

All models controlled for marital status, personal annual income, age and mother’s education. Personal annual income was measured through how much money a woman earned in the year prior, coded as a dummy variable where 0 = \$0-19 999, 1 = \$20 000 – 39 999, 2 = \$40 000 – 59 999, and 3 = \$60 000 or greater. Personal income was used rather than household income as studies have shown that a woman’s own resources can be most beneficial to her own well-being and control over her life (e.g. Kan 2008;

Udansky and Parker 2011). Mother's education, an indicator of family background, has been shown to be critical in children's attainments, including own education (e.g. Duncan and Brooks-Gunn 1997). Mother's education was included as a dummy variable with 0 = less than high school, 1 = high school, 2 = some post-secondary or greater, and 3 = "don't know/missing". Logistic regressions not shown here indicated that those who did not know their mother's education level or who were missing on this variable were among the most disadvantaged.

#### 4.4.4 Analytic strategy

OLS regression models were used to estimate models for self-efficacy, self-esteem, and social support. Self-rated health was estimated using ordered logistic regression. The likelihood of exhibiting a chronic condition was estimated using binary logistic regression. In addition to full models which pool white and black women, split-models were run for white and black women separately. These models are equivalent to testing interaction terms in a full model of pooled groups and provide greater ease with which to assess the statistical significance of independent variables within each group (Phillips and Sweeney, 2005). This approach is particularly important given the limited understanding of how psychosocial and material resources relate to health within black and white women. Chi square tests were conducted to determine any significant differences between these groups. The control variables identified in the section above were included as they were identified as variables which could affect the dependent variables of interest. These variables were retained in multivariate models, whether significant or not, because of their theoretical relevance to the outcomes under study. Model fit was assessed for OLS regressions using R-square values. For logistic regression models, Hosmer and Lemeshow's goodness-of-fit test in addition to the log likelihood chi-square and pseudo R-square. All analyses were conducted using STATA 11 software. The PSID 2007 cross-sectional weights were applied to univariate and bivariate analyses. Due to the complex sampling design of the PSID, which includes an oversample of some groups, these weights compensate for unequal selection probabilities and permit population characteristics to be estimated from sample persons (see Heeringa et al., 2011).

## 4.5 Results

### 4.5.1 Descriptive analyses

White women constituted 84% of the sample, while African-American women comprised 16% (Table 4.1). The mean age of the sample was 42 years. The proportions of women who had completed only high school, had some post-secondary education, or had finished their post-secondary degree were approximately equal at 0.30, 0.31, and 0.30, respectively. About half of the women reported annual earnings of less than \$20 000, although the majority were employed (76%). Most women reported being in good (28%), very good (36%), or excellent (26%) health. Approximately 38% indicated that they had a chronic condition. Self-efficacy, self-esteem, and social support were generally high, with average scores of 3.13 (range 1-4), 3.48 (range 1.8-4), and 29.48 (range 4-44), respectively. A greater proportion of black women than white women reported fair/poor health and the presence of a chronic condition (Table 2). Black women also averaged lower social support than white women (24.91 vs. 30.35); however, levels of self-esteem and self-efficacy were similar.

**Table 4.1 Sample percentages (weighted) by race, health status, material and psychosocial resources, and demographic controls, CDS-PSID, 2007 (N=869)**

Variable	Percentages	Standard Error
<i>Race</i>		
White	83.8	
Black	16.2	
<i>Self-Rated Health</i>		
Excellent	26.0	
Very good	36.0	
Good	28.4	
Fair/Poor	9.6	
<i>Chronic Condition</i>		
No	62.5	
Yes	37.5	
<i>Education Level</i>		
Less than high school	8.9	
High school	30.3	
Some post-secondary	31.1	
Post-secondary	29.7	
<i>Income Level</i>		
\$0-19,999	49.6	
\$20,000-39,999	29.2	
\$40,000-59,999	11.8	
\$60,000+	9.3	
<i>Employment Status</i>		
Employed	0.76.3	
Unemployed	0.08.5	
Homemaker	0.15.2	
<i>Marital Status</i>		
Married	74.7	
Single	10.1	
Divorced/Separated	15.2	
<i>Mother's Education</i>		
Less than high school	17.9	
High school	46.9	
>High school	29.7	
Don't know/Missing	5.5	
	<u>Means</u>	
Self-efficacy (range 1-4)	3.13	0.024
Self-esteem (range 1.8-4)	3.476	0.017
Social Support (range 4-44)	29.476	0.29
Age (years)	42.359	0.276

PSID cross-sectional weight is applied.

Self-efficacy and self-esteem are measured on a scale of 1 (lowest) to 4 (highest).

Social support is measured on a scale of 1-44.

**Table 4.2 Weighted percentages and means self-rated health, chronic conditions, and psychosocial resources by race, CDS-PSID, 2007 (N=869)**

	White	Black
	Percentages	
<i>Self-Rated Health</i>		
Excellent	28.4***	13.4***
Very Good	37.5***	28.9***
Good	26.0***	40.6***
Fair/Poor	8.1***	17.1***
<i>Chronic Condition</i>		
Yes	35.4*	48.1*
No	64.6*	51.9*
	Means	
<i>Self-efficacy</i>	3.134 (0.026)	3.107 (0.062)
<i>Self-esteem</i>	3.480 (0.019)	3.451 (0.037)
<i>Social Support</i>	30.357** (0.304)	24.906** (0.640)

PSID cross-sectional weight is applied.

Self-efficacy and self-esteem are measured on a scale of 1 (lowest) to 4 (highest).

Social support is measured on a scale of 1-44.

Test for significance was Chi<sup>2</sup>.

\*p<0.05; \*\*p<0.01; \*\*\*p<0.001



## 4.6 Multivariate analyses

### 4.6.1 Psychosocial outcomes

#### 4.6.1.1 Self-efficacy

Model 1 included race, education, and employment in relation to self-efficacy for the entire group of women, in addition to specified control variables (Table 4.3). Supporting hypothesis 1a, education, and employment had modest relations to self-efficacy. Post-secondary education was associated with an increase in self-efficacy ( $p < 0.05$ ). Relative to women who were employed, unemployed women had lower self-efficacy ( $p < 0.001$ ). Black women had slightly higher self-efficacy than whites ( $p < 0.05$ ), contrary to hypothesis 1b.

To test hypothesis 1c, Model 2 compared variables related to white and black women's self-efficacy separately. Education was not significantly associated with self-efficacy for white women. Black women who obtained a post-secondary education had significantly higher self-efficacy than those black women with less than a high school degree. Contradicting this hypothesis, in Model 2, black women with post-secondary education had a self-efficacy score nearly 30% higher than those with less than a high school education ( $p < 0.05$ ). Education coefficients for blacks and whites, however, were not statistically different. Unemployment was negatively associated with self-efficacy for both black and white women, with a slightly stronger relation for white women ( $p < 0.05$ ).

**Table 4.3 Estimated coefficients from a series of OLS models of women's self-efficacy (N=869)**

	Model 1	Model 2	
	(SE)	White	Black
Black	0.121* (0.048)		
Education (<H.S.)			
High School	0.014 (0.067)	-0.150 (0.104)	0.094 (0.091)
Some Post-Secondary	0.134 (0.070)	-0.021 (0.105)	0.193 (0.101)
Post-Secondary	0.192* (0.083)	0.038 (0.116)	0.289* (0.141)
Employment Status <sup>a</sup> (Employed)			
Unemployed	- 0.287*** (0.066)	- 0.310** (0.096)	- 0.256** (0.094)
Homemaker	-0.020 (0.063)	-0.020 (0.075)	-0.035 (0.116)
R <sup>2</sup>	0.075	0.076	0.119

Models also control for personal annual income, marital status, age, and mother's education.

N(white women)=487; N(black women)=382.

Self-efficacy is measured on a scale of 1(lowest) to 4 (highest).

<sup>a</sup>Coefficients for blacks and whites are statistically significant at  $\chi^2=5.41$ ,  $p<0.05$ .

\* $p<0.05$  \*\* $p<0.01$  \*\*\* $p<0.001$ .

#### 4.6.1.2 Self-esteem

Similar to self-efficacy, black women had slightly higher self-esteem than white women, and higher education increased and unemployment lowered women's self-esteem (Table 4.4). When self-esteem was evaluated separately for white and black women in Model 2, again contrary to hypothesis 1c, higher education was significantly associated with higher self-esteem for black women only ( $p < 0.01$ ). Unemployment was significantly related to self-esteem for white women but not for black women ( $p < 0.01$ ).

#### 4.6.1.3 Social support

Contrary to hypothesis 1, no significant racial differences were observed in social support, and education and employment were not significantly associated with social support (Table 5). Though not shown here, models introducing control variables in stages revealed a significant association between marital status and social support, such that being single or divorced/separated was related to less social support for women. Once again, results were similar when factors related to social support were evaluated for white and black women separately (see Model 2).

Overall, consistent with the expectations of hypothesis 1, socioeconomic resources were related to higher levels of psychosocial resources (self-efficacy and self-esteem) among women. However, contrary to expectations, black women had modestly higher levels of these psychosocial resources than white women, and it appeared that black women experienced the benefits of higher education for psychosocial resources to a greater extent than white women.

**Table 4.4 Estimated coefficients from a series of OLS models of women's self-esteem (N=869)**

	Model 1 (SE)	Model 2 (SE)	
		White	Black
Black	0.089* (0.034)		
Education (<H.S.) <sup>a</sup>			
High School	0.067 (0.047)	-0.034 (0.076)	0.093 (0.063)
Some Post- Secondary	0.170** (0.050)	0.052 (0.072)	0.196** (0.070)
Post- Secondary	0.235*** (0.059)	0.106 (0.085)	0.304** (0.098)
Employment Status <sup>b</sup> (Employed)			
Unemployed	-0.133** (0.047)	- (0.041)	-0.068 (0.065)
Homemaker	0.006 (0.045)	0.041 (0.055)	-0.061 (0.080)
R <sup>2</sup>	0.097	0.085	0.162

Models also control for personal annual income, marital status, age, and mother's education.

N(white women)=487; N(black women)=382.

Self-esteem is measured on a scale of 1(lowest) to 4 (highest).

<sup>a</sup>Coefficients for blacks and whites are statistically significant at 6.62,  $p < 0.01$ .

<sup>b</sup>Coefficients for blacks and whites are statistically significant at  $\chi^2 = 8.91$ ,  $p < 0.01$ .

\* $p < 0.05$  \*\* $p < 0.01$  \*\*\* $p < 0.001$ .

**Table 4.5 Estimated coefficients from a series of OLS models of women's social support (N=869)**

	Model 1 (SE)	Model 2 (SE)	
		White	Black
Black	-0.023 (0.525)		
Education (<H.S.)			
High School	-0.531 (0.724)	-1.307 (1.128)	-0.208 (0.995)
Some Post-Secondary	-1.359 (0.762)	-1.187 (1.146)	-1.765 (1.109)
Post-Secondary	-1.582 (0.899)	-1.804 (1.261)	-2.132 (1.547)
Employment Status (Employed)			
Unemployed	-0.564 (0.716)	-0.225 (1.048)	-0.908 (1.032)
Homemaker	-0.363 (0.691)	-0.034 (0.822)	-1.549 (1.268)
R <sup>2</sup>	0.327	0.232	0.302

Models also control for personal annual income, marital status, age, and mother's education.

N(white women)=487; N(black women)=382.

Self-efficacy and self-esteem are measured on a scale of 1 (lowest) to 4 (highest).

Social support is measured on a scale of 1-44.

\*p<0.05 \*\*p<0.01 \*\*\*p<0.001.

## 4.6.2 Health Outcomes

### 4.6.2.1 Self-rated health

Model 1 included race and psychosocial variables only (Table 4.6). As expected, black women were nearly 60 percent less likely to be in good health compared to their white counterparts ( $p<0.001$ ). Women with high self-efficacy were nearly 1.5 times as likely to report better self-rated health as women with lower scores ( $p<0.01$ ). Those with high self-esteem were about twice as likely to report good, very good, or excellent health ( $p<0.001$ ). Social support did not appear to be significantly related to women's self-rated health. Self-efficacy and self-esteem remained positively associated with self-rated health in subsequent models. With the introduction of education and employment status

in Model 2, black women were about half as likely to be in better health than white women ( $p < 0.001$ ). Post-secondary education and employment were not significantly related to self-rated health for women in the full model, contrary to hypothesis 1a.

Models 3 and 4 compared factors related to self-rated health for white and black women separately. Split models revealed that psychosocial variables operated somewhat differently for white and black women. In Model 3, black women with high self-esteem were almost three times as likely to report better self-rated health as black women with lower self-esteem scores ( $p < 0.001$ ), although the coefficient was not significant for white women. The association between self-esteem and self-rated health was also significantly different for whites and blacks ( $p < 0.001$ ). Conversely, white women with high self-efficacy scores were about twice as likely to be in higher categories of self-rated health than white women with lower scores ( $p < 0.01$ ). Again, this association was significantly different for white and black women ( $p < 0.001$ ).

Education was not significantly associated with self-rated health for black women, although odds ratios did indicate that even black women with a post-secondary education were less likely to be in high self-rated health. Education did benefit the health of white women, who were about 3.4 times as likely to report good, very good, or excellent health with a post-secondary education compared to whites who did not complete high school ( $p < 0.01$ ). Educational disparities in health for white and black women were significant. White women who were unemployed were approximately half as likely to report better self-rated health as white women who were employed ( $p < 0.05$ ), but employment status was not significantly associated with health in black women. Like education, employment's association with health differed by race ( $p < 0.001$ ).

**Table 4.6 Estimated odds ratios from a series of ordered logistic regressions of women's self-rated health (N=869)**

	Model 1 (SE)	Model 2 (SE)	Model 3 (SE)		Model 4 (SE)	
			White	Black	White	Black
Black	0.430*** (0.057)	0.478*** (0.074)				
Self-efficacy <sup>a</sup>	1.459** (0.197)	1.365* (0.188)	1.987** (0.396)	1.128 (0.208)	1.848** (0.378)	1.051 (0.200)
Self-esteem <sup>b</sup>	2.097*** (0.394)	1.726** (0.333)	1.603 (0.421)	2.738*** (0.739)	1.244 (0.337)	2.440** (0.692)
Social support	1.003 (0.008)	1.004 (0.010)	0.991 (0.013)	1.011 (0.012)	0.989 (0.015)	1.016 (0.014)
Education (<H.S.) <sup>c</sup>						
High school		0.722 (0.161)			1.128 (0.419)	0.532* (0.151)
Some Post-secondary		0.973 (0.230)			1.982 (0.764)	0.507* (0.160)
Post-secondary+		1.639 (0.452)			3.435** (1.451)	0.637 (0.272)
Employment Status (Employed) <sup>d</sup>						
Unemployed		0.695 (0.148)			0.495* (0.165)	0.809 (0.234)
Homemaker		0.784 (0.162)			0.977 (0.255)	0.500 (0.184)
Log likelihood	- 1106.289	- 1081.400	- 610.480	-492.719	- 586.511	-493.118

Models 2 and 4 also control for personal annual income, marital status, age, and mother's education. In models not shown here, self-efficacy, self-esteem, and social support were included in separate models to isolate their effects. Results were similar to those of the combined model, which is included here.

N(white women)=487; N(black women)=382.

Self-efficacy and self-esteem are measured on a scale of 1(lowest) to 4 (highest). Social support is measured on a scale of 1-44.

<sup>a</sup>Coefficients for blacks and whites are statistically significant at  $\chi^2=46.61$ ,  $p<0.001$ .

<sup>b</sup>Coefficients for blacks and whites are statistically significant at  $\chi^2=42.38$ ,  $p<0.001$ .

<sup>c</sup>Coefficients for blacks and whites are statistically significant at  $\chi^2=30.86$ ,  $p<0.001$ .

<sup>d</sup> Coefficients for blacks and whites are statistically significant at  $\chi^2=23.66$ ,  $p<0.001$ .

\* $p<0.05$  \*\* $p<0.01$  \*\*\* $p<0.001$ .

#### 4.6.2.2 Chronic illness

Black women were about 1.7 times as likely as white women to experience a chronic disease ( $p < 0.001$ ) (see Table 4.7, Models 1 and 2). While self-esteem and social support were not significantly associated with chronic conditions, women with higher self-efficacy scores were about 30 percent less likely to report a chronic condition ( $p < 0.05$ ). Education was not significantly associated with women's likelihood of reporting a chronic condition. Women who were unemployed were nearly twice as likely to report a chronic condition ( $p < 0.01$ ).

In Models 3 and 4, separate odds ratios for white and black women were compared. White women with high self-efficacy were about 40 percent less likely to exhibit a chronic condition ( $p < 0.05$ ), while neither self-esteem or self-efficacy were significantly related to chronic conditions reported by black women. These psychosocial resources were differentially associated in white and black women ( $p < 0.001$ ). As hypothesized, racial differences were observed in the relation of education to the likelihood of reporting a chronic condition ( $p < 0.001$ ). White women with a post-secondary education were about 65 percent less likely to report a chronic condition than whites with less than high school ( $p < 0.05$ ), while education was not significantly related to chronic illness for black women. White women who were unemployed were almost 3 times as likely to experience chronic illness as those who were employed ( $p < 0.01$ ) while unemployment was not significant for black women.

Contrary to hypothesis 2, higher education was not associated with increased reporting of good health or decreased reporting of chronic illness for women. Consistent with this hypothesis, greater psychosocial resources generally were associated with better health. Self-efficacy and self-esteem were associated with better self-rated health, while only self-efficacy was significantly associated with chronic illness. As expected, black women reported worse self-rated health than white women and were more likely to report a chronic condition. Hypothesis 2c was also supported as black women did not report better health associated with both psychosocial and material resources as did white women.



**Table 4.7 Estimated odds ratios from a series of logistic regressions of women's chronic conditions (N=869)**

	Model 1	Model 2	Model 3		Model 4	
	(SE)	(SE)	White	Black	White	Black
Black	1.716*** (0.253)	1.675** (0.295)				
Self-efficacy <sup>a</sup>	0.706* (0.106)	0.717* (0.111)	0.608* (0.134)	0.807 (0.168)	0.588* (0.135)	0.786 (0.176)
Self-esteem <sup>b</sup>	0.945 (0.195)	1.035 (0.223)	1.194 (0.352)	0.750 (0.219)	1.362 (0.420)	0.972 (0.313)
Social support	1.006 (0.009)	1.005 (0.011)	1.011 (0.015)	1.003 (0.013)	1.008 (0.018)	1.004 (0.016)
Education (<H.S.) <sup>c</sup>						
High school		0.676 (0.164)			0.309** (0.128)	1.149 (0.361)
Some Post-secondary		0.860 (0.220)			0.471 (0.196)	1.414 (0.499)
Post-secondary+		0.612 (0.187)			0.344* (0.160)	1.188 (0.581)
Employment Status (Employed) <sup>d</sup>						
Unemployed		1.884** (0.452)			2.757** (1.052)	1.309 (0.426)
Homemaker		1.203 (0.277)			0.924 (0.285)	1.661 (0.674)
Log likelihood	-578.219	-	-	-	-	-
		562.118	315.694	261.755	302.744	261.784

Models 2 and 4 also control for personal annual income, marital status, age, and mother's education. In models not shown here, self-efficacy, self-esteem, and social support were included in separate models to isolate their effects. Results were similar to those of the combined model, which is included here.

N(white women)=487; N(black women)=382.

Self-efficacy and self-esteem are measured on a scale of 1(lowest) to 4 (highest). Social support is measured on a scale of 1-44.

<sup>a</sup> Coefficients for blacks and whites are statistically significant at  $\chi^2=13.14$ ,  $p<0.001$ .

<sup>b</sup> Coefficients for blacks and whites are statistically significant at  $\chi^2=11.79$ ,  $p<0.001$ .

<sup>c</sup> Coefficients for blacks and whites are statistically significant at  $\chi^2=8.02$ ,  $p<0.01$

<sup>d</sup> Coefficients for blacks and whites are statistically significant at  $\chi^2=13.28$ ,  $p<0.001$ .

\* $p<0.05$  \*\* $p<0.01$  \*\*\* $p<0.001$ .

## 4.7 Discussion and conclusion

This study has focused on racial disparities in health among women, and, in particular, on the various aspects of social status in relation to both the development of psychosocial resources and good health. The findings demonstrate that research should not treat women as a homogenous group, assuming that mechanisms affecting health operate the same for women, regardless of their race. Through investigating intersectionality and health, the processes leading to health disparities can be better understood as both race and gender are strongly linked to health outcomes. This has been accomplished through evaluating psychosocial resources and their relations to women's health outcomes, as well as examining these relationships for black and white women separately. It is clear that these important resources are not equally beneficial to the health of black and white women.

Similar to previous research (Mirowsky and Ross 1990; Schieman 2001; Erol and Orth 2011), the findings here demonstrated that black women had higher self-esteem than their white counterparts, albeit this difference was of little practical significance. Contrary to previous research, however, black women had higher self-efficacy than whites, although this difference was once again small. Nevertheless, positive self-esteem and self-efficacy may be further enhanced through socioeconomic advancement. Consistent with the theory of resource substitution (Ross and Mirowsky 2006), black women experienced the greatest psychosocial gains from the acquisition of a post-secondary education. Material resources also explained more of the variation in black women's self-efficacy, and in particular, self-esteem, than white women's. Structural circumstance may therefore be more important to the psychosocial development of black women.

For women as a whole, education appeared to have a slightly stronger association with self-esteem, while unemployment was central to women's self-efficacy. Unemployment was also central to white women's psychosocial outcomes, but less so or not at all for black women. This highlights the importance of employment to women's psychosocial outcomes (Schieman 2002), but also demonstrates that it may be more specific to white women. Meanwhile, education appears to matter more for black women.

Given that blacks may be less likely to convert education into other resources, such as employment (Shuey and Willson 2008), it could be that these women are more dependent on education for their psychosocial outcomes, as postulated by resource substitution theory (Ross and Mirowsky 2006).

With regard to health outcomes, higher education was associated with better self-reported health for white women only. This adds to findings such as Shuey and Willson (2008) that African Americans do not obtain the same level of health benefits of education as whites. Furthermore, black women reported increases in both self-esteem and self-efficacy associated with higher education, two psychosocial resources that have been postulated as essential for health and one reason why education has such a robust relationship with health (Mirowsky and Ross 2003). Yet, while black women may develop these intrapersonal rewards through structural advantage, such as higher education, they certainly do not experience the same degree of health improvement as white women. Instead, highly educated black women, though equipped with self-enhancing psychosocial resources, remained at a persistent health disadvantage relative to whites. That being said, black women with higher self-esteem were more likely to report being in better health than black women with lower levels of these resources. Thus, this study has provided a more nuanced account of previous findings on race and health by showing that resources may improve health within a disadvantaged group while still not bringing them up to the level of health experienced by their advantaged counterparts.

Similar to findings related to self-rated health, black women's disadvantage persisted with regard to chronic illness. Once again, education did not appear to protect black women against chronic conditions, to the same extent that it did for white women. Like self-rated health, within groups models revealed that self-efficacy was associated with less reporting of chronic illness for white women only. Accordingly, disparities in the health benefits of self-efficacy exist between whites and blacks, posing challenges to assumptions that this resource is equally beneficial (Mirowsky and Ross 2003). Self-esteem was not significantly associated with chronic illness for whites or blacks. Thus, even though black women's self-rated health was associated with higher levels of self-

esteem, the same was not true for chronic illness. Accordingly, the health benefits of particular psychosocial resources may be specific to the outcome under study.

Ultimately, it is clear that black women experience a health disadvantage relative to white women across both measures of health used in this study, and that this is not explained by differences in psychosocial or material resources. Furthermore, a discrepancy was observed between the psychosocial enhancement experienced by black women who obtain a post-secondary education and their persistent health disadvantage. While resource substitution appeared to operate for black women's psychosocial outcomes, the same cannot be said with regard to health. The most disadvantaged group – in this case, black women – did not report better health with increased educational attainment. Thus, while previous research has argued that resources, such as self-efficacy and self-esteem, are critical to good health (Mirowsky and Ross 2003), it appears that these resources, in addition to socioeconomic ones such as education, are not enough to bring black women up to the same health standard as whites. Research must therefore continue to investigate the mechanisms through which black women experience, on average, poorer health than their white counterparts. An interesting avenue to pursue is the role of early childhood poverty, as black children are more likely than their white peers to live in poverty (National Poverty Center 2013). Given the association between childhood poverty and poor adult health (see Seabrook and Avison 2012 for a review), it may be that adult resources acquired by black women may not be enough to compensate for exposure to poverty in childhood. Future research, both qualitative and quantitative, should design questions that relate specifically to identity and its impact on the lived health experiences of individuals.

In general, action must be taken in both research and policy address how forms of oppression (e.g. gender and race) intersect to produce health inequalities. This information can not only shift how researchers conceptualize health disparities (Hankivsky, 2012), but also, can be used within the health care system to effectively monitor health disparities, identify problematic areas of care, more precisely determine causes, and develop interventions (Geiger, 2006). Continued study in this area can also generate improved understanding of “the social, cultural, and political processes that

produce disparities in health” (Schulz, Freudenberg, and Daniels, 2006: 371). Further, health care professionals and services need to consider the identities of their patients, and the unique way in which these identities impact health, as demonstrated by the present study. Continued incorporation of intersectionality in empirical research is therefore critical.

This study, of course, was not without its limitations. The data used were cross-sectional, so it was not possible to address temporal sequence of the associations or change over time; however, it is one of the few data sources available to examine the research questions of interest. The women in the sample used were also generally middle-aged and were mothers of young children. Currently, no studies compare differences in psychosocial resources and health between women with and without children. However, 80% of women in this age group in the United States have children (U.S. Census Bureau, 2014), and the PSID is one of the few datasets that contains these important variables. Accordingly, the research is valuable in demonstrating how the health benefits of psychosocial and material resources may be shaped by the intersection of race and gender. Future research might use a sample that also includes women without children as well as older women.

A complex relationship clearly exists among race, gender and health that may result in a unique form of health disadvantage. Capturing the extent of this disadvantage and determining all that explains it remains a methodological challenge in sociology, as well as a promising area for continued investigation. Once again, important intervention opportunities can be identified through examining the combined effects of race and gender. Through examining white and black women as separate groups, this study has revealed some of the issues unique to each group, such as differential associations between resources and health. Moving beyond the inclusion of gender and race as variables to be controlled in our models to understanding the mechanisms that increase and diminish health within these groups is necessary if we are to truly understand their effects.

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## Chapter 5

### 5 Conclusion

#### 5.1 Contributions

Disparities in health between women and men have often been assumed to be both constant and inevitable. With an emphasis on differences between women and men, much research has overlooked the importance of temporal, contextual, and intersectional factors that influence health disparities. While the health trajectories of men and women may differ from each other, these trajectories can also differ within each group. Further, underlying mechanisms of health inequality (e.g. socioeconomic status), may be patterned differently for women and men, rendering comparisons between them difficult. Accordingly, this dissertation contributes to the emerging body of literature that seeks to challenge traditional approaches to the conceptualization and measurement of gender and health (e.g. Pudrovska, 2014; Brown & Hargrove, 2013). The complementary theoretical frameworks of the life course and intersectionality are employed in this dissertation to conceptualize and measure health as a process and to examine how gender, as a determinant of health, does not operate in isolation. Rather, differences between and among men and women can be understood as a result of how gender works within a broader network of social forces to impact health.

This dissertation also demonstrates that there is much to be gained from examining how the aforementioned factors create inequalities not only between women and men, but also, among them. Understanding processes of heterogeneity among these groups may be key to promoting population health through ensuring specific groups of individuals have what they need to enjoy healthy lives. Some (not all) differences in health between women and men may be unavoidable for a variety of complex reasons (e.g. biology/genetics), as existing literature demonstrates (e.g. Crimmins et al., 2002). Differences in health among women and among men, however, actually may be preventable with attention to how gender works in conjunction with other factors in unique ways for women and men. For example, while socioeconomic status and race

affect health, the ways in which they affect health, and the particular outcomes they affect, can be different for women than for men. Beyond this, in some instances, health inequality between women and men may appear to have been achieved, but this should not obscure inequalities existing among them. Research that provides insight into which factors are relevant to health among groups and how these factors impact health is critical for this aim. Therefore, recognizing diversity and inequality among women and men is an important contribution of the research presented here, in addition to contextualizing the nature of health disparities between the two.

## 5.2 Summary of and Links Between Findings

Beginning in Paper 1 (Chapter 2), the relationship between gender and health is situated within time and place, based on the life course theoretical tenet that individual pathways are influenced by social and historical forces (Elder, Johnson, & Crosnoe, 2003). Through the introduction of birth cohort to analyses of gender and self-rated health, this chapter reveals that differences in these trajectories between women and men may in fact be less about gender in itself, and more about its relationship to other social institutions of a particular historical period, which change over time. Specifically, from this analysis, gender differences in health appeared to be specific to cohort – or the year in which a particular group of individuals were born – rather than generalizable across all women and men. Additionally, men and women of more recent cohorts, who have come of age in a vastly different social and historical context than their early birth year counterparts, may be much more similar in health status than initial literature has suggested. Thus, contrary to prior literature which finds that women report worse self-rated health than men (e.g. Idler, 2003) or that no differences exist at all (e.g. Rohlfen & Kronenfeld, 2014), this paper indicates that the extent to which there are gender differences in reported health can vary by cohort. From a methodological standpoint, Paper 1 also reveals that women and men's experiences of health in a particular survey year cannot be assumed to represent the average experience of a cohort across time. Thus, panel data are of utmost utility in understanding the relationship between gender and health.

The importance of examining mechanisms of health inequality across multiple health outcomes is demonstrated in Paper 2 (Chapter 3). Here, the impact of dynamic experiences of childhood poverty in shaping trajectories of health among women and men was examined, with results varying by disease outcome. In using separate models to reveal how processes of disadvantage impact health, this paper found that initial and long-term childhood poverty may be more consequential for women's health. Consequently, this chapter builds on recent research which incorporates the timing and duration of childhood economic hardship (e.g. Shuey & Willson, 2014) through considering how dynamic processes of disadvantage and health may vary by gender as well as by health outcome. While much life course research has demonstrated a link between early life conditions and later life health outcomes (e.g. Luo & Waite, 2005; Johnson & Schoeni, 2011) less has investigated how the "long-arm of childhood" (see Hayward & Gorman, 2004) may be contingent on gender. In fact, economic hardship may affect disease onset differently for women and men, as demonstrated by Paper 2, indicating that processes leading to adverse health outcomes are not always the same across gender – and potentially, other groups (e.g. blacks vs. whites). Had women and men been examined in the same model with childhood economic hardship examined as a key explanatory variable, the study might have concluded that childhood conditions affect the adult health of everyone in the same way or that it in fact affects no one at all. Instead, this chapter indicates childhood economic hardship may be a primary mechanism in creating heterogeneity in the health of women and may not be as critical for men. Gender is intricately tied to life chances, as women's social roles, including their work and family lives, are structured by existing power relations, which in turn impact their well-being (see Radtke & Stam, 1994; Lorber, 2011; Zawilski, 2015). It could be the case that when women begin life in an already disadvantaged position (i.e. poverty), this serves to amplify the role of gender in shaping outcomes as women age. That is, by the nature of a patriarchal society, women are already more likely to face discrimination, increased caregiving burdens, lower pay, and so forth, all of which are explicated by existing theories of gender and health as important determinants (see Bird & Rieker, 2008). It may be more difficult to overcome these challenges from a position of childhood poverty, whereas women born into a position of advantage may be more able

to cope with or overcome gender-based challenges through increased access to resources and personal capacities such as resilience, for example. Conversely, men have a certain advantage in life by virtue of their gender, which may increase their chances of social mobility and its health benefits. This is not to say that childhood circumstances are inconsequential for men's health: Paper 2 found it to significantly impact men's cardiovascular outcomes. Interestingly, previous work using static measures of childhood disadvantage found it to be insignificant for men's heart health outcomes (Hamil-Luker & O'Rand, 2007). Consequently, this measure, which accounted for the timing and duration of childhood poverty, may be more useful in teasing out the effects of hardship on adult health.

Paper 2 provides further nuance to prior studies indicating a stronger association between childhood and adult health for women (e.g. Hamil-Luker & O'Rand, 2007; Pudrovska & Anikputa, 2013) through attention to the timing and duration of these experiences and multiple health outcomes. Specifically, it showed that for some health outcomes, the timing of childhood poverty is important (e.g. early on in childhood -- arthritis) while for others, duration is what matters (e.g. long-term poverty -- heart attack/heart disease/stroke). Hence, not only does childhood economic hardship impact women and men in potentially different ways, different characteristics of poverty experiences are associated with different health outcomes. The significance of varying experiences of poverty on health speaks to the contention of life course theory that timing is critical in the unfolding of individual trajectories (Elder, Johnson, & Crosnoe, 2003). It also exemplifies processes of cumulative disadvantage theory in the growth of inequality over time, which stemming from initial disparities between individuals (O'Rand, 2006). These findings add to existing research on cumulative disadvantage and health, through utilizing a dynamic measure of disadvantage that advances approaches which focus on a indicators at one point in time, such as education and/or occupation of the household head (e.g. Ferraro, Schafer, & Wilkinson, 2015). Given the relevance of timing and duration to experiences of advantage and disadvantage, future studies might consider these concepts as integral to their theoretical framework.

Given the importance of childhood economic hardship to women's health outcomes, this paper supports suggestions in Paper 1 that disparities in health between women and men in the earliest cohort were the result of a greater impact of economic hardship on women's health. That is, Paper 2 demonstrates that women's health is potentially more adversely impacted by circumstances of early life involving hardship. In Paper 2, women born or very young during the Great Depression, a time of immense economic hardship, had worse health. This chapter examines multiple health conditions among a group of women and men in a more recent cohort (individuals 0-8 years old in 1968, or late baby-boomers). Like self-rated health in Paper 1, there appeared to be no significant differences in the health of men and women in this recent cohort, with each group experiencing similar rates of the chronic conditions examined. However, within-group models revealed heterogeneity among women and men, meaning that while there may be no apparent differences between these groups, there are important differences among them. Thus, intragroup analyses are essential to the study of health inequality.

The value of intragroup analyses is highlighted by Paper 3 (Chapter 4), which examines the intersection of gender and race with socioeconomic and psychosocial resources. This chapter adds support to the notion that women are not a homogenous group – a central contention of intersectionality theory (Crenshaw, 1989) – and that resources can impact health very differently within and between groups. In this particular investigation, it was evident that while resources such as education and self-efficacy can improve health among one group (e.g. black women), they do not necessarily bring these individuals to the same level of health experienced by their advantaged counterparts. This is an example of how health-promoting resources can contribute to improved health among groups (e.g. for black women) but not always between them (e.g. between black and white women), and demonstrate the important insights gained by within-groups analyses (see Hankivsky, 2012; McCall, 2005).



### 5.3 Limitations and Future Directions

The papers comprising this dissertation are not without their limitations. Details and implications of specific limitations are discussed in each chapter. Here, limitations with specific implications for future research will be discussed further.

Paper 1 (Chapter 2) is limited by the examination of one health outcome (self-rated health), necessitating the examination of additional measures of health, because the relationship between gender and health can be dependent upon the outcome examined (Gorman & Read, 2006). While differences in self-rated health between women and men are not evident among more recent cohorts, the same may not be true with regard to chronic conditions, for example. Self-rated health net of physical conditions represents individuals' perceptions of their own health, which can take into account a more inclusive or comprehensive set of factors than physical health indicators only (Welch, Schwartz, & Woloshin, 2011). Nevertheless, it would be of value to assess cohort related changes in gender differences in physical health conditions, given the varying etiologies of such conditions in addition to potential changes in gendered patterns of disease across historical time, such as women's increased access to resources, changes in the health behaviours of men and women, and changes in social roles. Determining whether the observed findings hold across other measures of health is key to advancing the concept of cohort as analytically useful and important for understanding the relationship between gender and health. While cohort patterns in the relationship between factors such as education and health have been well-established in the literature (e.g. Lynch, 2003; Chen, Yang, & Liu, 2010), the relationship between gender and health across cohorts has been less examined. Research using long-term panel data to assess these relationships is especially scarce.

Future research should also consider the impact of childhood economic hardship on the health of women and men across multiple cohorts, as this Paper 2 (Chapter 3) was limited to one birth cohort only. Such an investigation would speak to the relevance of childhood economic hardship across time as well as the generalizability of its effects. Beyond this, Paper 2 is limited by the examination of economic hardship as a marker of disadvantage, when other forms of disadvantage (such as family structure or childhood

trauma) can also have long-term implications for health. Accordingly, future research should investigate other measures of hardship in a dynamic way to determine cumulative impacts. This paper also did not examine potential mechanisms through which disadvantage may accumulate for women (and men), and understanding these pathways may be important to intervention strategies.

Finally, it should be noted that Paper 3 (Chapter 4) is limited by its cross-sectional design and future research should examine how trajectories of health are impacted by the intersection of race, gender, and other social locations over time. As demonstrated by Papers 1 and 2, health and some aspects of social location are dynamic. Nevertheless, this chapter highlights the multifaceted nature of gender and health through considering its intricate relationship with race. A challenge for empirical work utilizing an intersectional perspective exists in accounting for potentially endless combinations of individual identities. Thus, for practical purposes, this investigation was limited to race and gender, two well-documented and critical determinants of health. As McCall (2005) points out, empirical investigations of intersectionality can restrict the number of included dimensions with justification for their inclusion. Further, many existing quantitative datasets are not adequately equipped to investigate a high number of social statuses at once given limitations in sample size, questions asked, and so forth. Future research development might involve a sampling frame that would enable investigators to combine multiple statuses without losing power as well as methods that accommodate various intersections over time. Qualitative research may also be useful here to supplement quantitative results.

## 5.4 Concluding Thoughts

In attending to life course and intersectional principles, this dissertation demonstrates that implicit in research on gender and health must be considerations of processes that begin in early life and evolve over time, of inequalities that cross multiple lines of stratification, and of various measures of resources and health. Rather than framing the health of women and men as a primary competing inequality, this dissertation has recognized that differences among women can be just as important as differences between women and men with implications for health equality. Foremost, how we understand health equality

should be grounded in historical context. While disparities between women and men, for example, may appear large in cohorts of a particular time period, this is not necessarily the case in subsequent years. Second, while disparities in health may appear to be minimal or even non-existent between groups, they actually may be quite large among them. As demonstrated collectively by Chapters 2 and 3, the health of women and men may become more similar with cohort replacement, but significant intragroup disparities can simultaneously exist. Third, mechanisms of health inequality, such as cumulative disadvantage, do not always impact groups in the same way and should not be assumed as such. Fourth, resources can promote health within a particular group without contributing to health equality between groups. In the case of the black and white women in Chapter 4, this can certainly be the case. Moving beyond comparing women and men to understanding the processes affecting heterogeneous health outcomes among them is therefore essential to advancing population health and well-being.

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