Published in final edited form as:

J Fam Issues. 2014 February 1; 35(3): 291–311. doi:10.1177/0192513X12468436.

Racial Variations of Parity Status as a Predictor of Disability Onset among Old-Old Women

Kenzie Latham, PhD¹ and Jeanne Holcomb, PhD²

¹Population Studies Center, University of Michigan, Institute for Social Research, University of Michigan, 426 Thompson Street, Room 2048 ISR, Ann Arbor, Michigan, 48106-1248, kelatham@umich.edu, (734) 763-3153

²Department of Sociology, Anthropology, and Social Work, University of Dayton, Dayton, OH, jholcomb1@udayton.edu, (937) 229-3695

Abstract

This research investigates whether the capacity of parity status to predict disability onset varies by race among older women. Data from the Health and Retirement Study (HRS) (1998–2008) was used, and a series of discrete-time event-history models were estimated using multinomial logistic regression. Disability onset was constructed from five common Activities of Daily Living (ADL) measures (i.e., difficulty dressing, bathing, eating, getting in/out of bed, and walking). The initial risk group was old-old women (i.e., born before 1925) without disability in 1998 (N=2,229). Nulliparous and high parity Black women born before 1925 were more likely to develop disability onset compared to White women with the same parity status. The results suggest that parity status among Black older women may be a salient risk factor for disability onset.

Introduction

Previous research investigating parity status as a predictor of later life health outcomes has found variations in mortality by parity (Doblhammer, 2000; Grundy & Tomassini, 2005; Hurt, Ronsmans, & Thomas, 2006). In general, the extant literature has revealed a U-shaped relationship with parity and risk of mortality, where nulliparous (childfree), single-child parity, and high parity women have a greater likelihood of mortality in later life than low parity women. Research examining other later life health outcomes such as chronic disease or functional health prevalence or onset is very limited; however, there is some evidence to suggest that nulliparous (see Teachman, 2010) and high parity (see Grundy & Tomassini, 2005; Henretta, 2007) women may experience poorer health outcomes in later life compared to low parity women. However, among early twentieth century cohorts in the United States (US), rates of childlessness, single-child parity, low parity, and high parity have varied significantly (Davis, 1982; Scheon, 2006). Moreover, stark racial variations in parity status have been observed among these early twentieth century cohorts including higher rates of childlessness and high parity among Black women (Boyd, 1989; Farley, 1970). This study contributes to existing literature by examining parity status as a predictor of later life functional health and exploring racial variations in the association between parity status and functional health. Utilizing the Health and Retirement Study (HRS), this study explores the potential long-range impact of parity and race on disability onset among a nationally representative sample of older women (i.e., born before 1925) in the US. Findings are discussed within the context of a life course perspective.

Literature Review

Parity during the Early Twentieth Century

Parity rates in the US have varied significantly by cohort in the first half of the twentieth-century (Davis, 1982; Morgan, 1991; Poston & Gotard, 1977; Poston & Kramer, 1983; Rowland, 2007; Scheon 2006; Westoff, 1952). According to Morgan (1991), childlessness among White women 45 years and older increased early in the twentieth century, peaked for women in their childbearing years during the Great Depression, and then fell precipitously for the “cohorts that produced the baby boom” (p. 781). Davis (1982) reported a similar pattern for childlessness and single-child parity with increasing childlessness and single-child parity for women born in 1891 to 1910 followed by decreasing childlessness and single-child parity among ever-married White and Black women born in 1911 to 1925; however, racial differences were observed, in which Black women were more likely to experience childlessness or single-child parity in each five-year birth cohort compared to White women. Boyd (1989) notes that prior to 1940 the proportion of childfree Black women was considerably higher compared to White women: “Approximately one-third of [B]lack women in the birth cohorts of 1900–1919 were permanently childless” (p.185), compared to approximately 20% of White women who were childless (Boyd, 1989).

In addition to trends in childlessness and single-child parity, shifts in rates of high parity have also been documented. The majority of women born in the cohorts prior to the beginning of the twentieth century experienced high parity; however, a sharp drop in high parity was observed among early twentieth century cohorts (Westoff, 1952). While a decrease in high parity was documented for both Whites and non-Whites, early twentieth century non-White cohorts had higher proportions of high parity compared to White cohorts (Cutright & Shorter, 1979; Westoff, 1952). For example, although non-White women born between 1903 and 1919 were more likely to be childfree than White women, they also had higher completed parity rates than White women (Evans, 1986). According to Westoff (1952), among women born in 1910, 49.3% of White women and 61.5% of Black women experienced high parity (i.e., 5 or more children ever born), whereas 27.0% of White women and 35.8% of non-White women born in 1940 experienced high parity (p. 553). While there was a sharp drop in the percent of women experiencing high parity, large racial differences remained.

Explanations of Racial Variations in Parity Status

Davis (1982) argues that for the cohorts who were of childbearing ages during the Great Depression, the two most salient factors shaping childlessness were prevailing ideology and economic conditions, regardless of race. Other research that focuses more on racial differences in rates of childlessness points to the significance of the health of women during childbearing years. While there is debate as to the underlying mechanisms leading to the elevated rates of childlessness among Black women during the early twentieth century, involuntary childlessness (i.e., sterility) due to malnutrition, sexually transmitted infections (STIs), other health conditions, and lack of adequate health care have been considered (Boyd, 1989; Cutright & Shorter, 1979). In general, the salient factors leading to increased childlessness among the earliest twentieth century cohorts are thought to be different for Whites and Blacks, where childlessness among Black women was indicative of poor health and childlessness among White women stemmed from changing marital patterns, age of first birth, social norms, and reproductive technology (Boyd, 1989; Cutright & Shorter, 1979; Farley, 1970; Tolnay & Guest, 1982; Wright, 1989). Cutright and Shorter (1979) also note differences in meanings of high parity among Blacks and Whites and suggest that Black women who were physically able to conceive and carry to term may not have had the resources to regulate fertility.

Parity Status as a Predictor of Later Life Health

Childbearing is linked to later life health outcomes through three main processes: biological mechanisms, social mechanisms, and variations in socioeconomic status (Grundy & Tomassini, 2005; Henretta, 2007; Wadsworth, 1997). More specifically, biological mechanisms underscore the long-term biological consequences associated with childbearing including variations in patterns of diseases and cause-specific deaths by parity status (Henretta, 2007). For example, Beral (1985) found that parous women had lower mortality rates for specific cancers such as breast, ovarian, and endometrial cancer than nulliparous women, whereas diabetes and cardiovascular mortality rates were higher for parous women compared to nulliparous women. Gillespie (2001) also observed higher rates of breast and uterine cancer among nulliparous women as well as higher levels of cervical cancer among high parity women.

Additionally, life course epidemiology describes the potential for critical periods, a sensitive and limited developmental window of time when adverse (or protective) environmental conditions could influence subsequent disease outcomes, and the accumulation of risk (i.e., chains of risk) as pathways linking earlier life circumstances such as childbearing years to later life health outcomes (Kuh, Ben-Shlomo, Lynch, Hallqvist, & Power, 2003). Although these earlier life circumstances are manifested from socially constructed and unequal environments, Ferraro, Shippee, and Schafer (2009) discuss how early life events influence later life health by demonstrating the potential for biological and psychological changes in the human body to alter the aging process throughout the life course. Childbearing may directly influence biological susceptibility for specific diseases via timing and number of births; however, childbearing, particularly childlessness, may also signify disparate socio-historical and early life conditions leading to variations in parity status.

Social relationships and integration may also be an important mechanism linking childbearing to later life health (Grundy & Tomassini, 2005; Henretta, 2007). Literature on adults in later life emphasizes that children are central in the social networks of parents. Children act in many health-promoting ways, including providing support, helping parents to access social services, monitoring health behaviors, and encouraging parents to develop new social circles (Kendig, Dykstra, Van Gaalen, & Melkas, 2007). Childfree adults may have fewer support networks (Gillespie, 2001). When compared to parents, older childless adults tend to be equally integrated with friend networks, but have lower levels of integration in close family networks. Some evidence suggests that childless persons rely more on other family members, such as siblings, nieces, and nephews, in the absence of children (Zhang & Hayward, 2001). Despite efforts to build support systems in the absence of children, networks based on friends, neighbors, and relatives are less likely to provide long-term assistance or the level of instrumental support that is often provided by children (Zhang & Hayward, 2001).

Finally, early and later life socioeconomic status (SES) may be another important pathway connecting parity and later life health outcomes. Childbearing patterns are associated with early socioeconomic status such as education (Heck, Schoendorf, Ventura, & Kiely, 1997; Retherford & Luther, 1996; Westoff, 1952). Additionally, later life socioeconomic status may also be associated with differences in childbearing, where timing and number of children may influence socioeconomic trajectories (Grundy & Tomassini, 2005; Henretta, 2007). To illustrate, prior research has suggested that early childbearing (Taniguchi, 1999) and more children (Glauber, 2007) may contribute to lower SES over the life course.

Empirical research examining parity and later life functional health outcomes is limited; however, a few notable studies have explored these links and produced equivocal results. For example, Spence (2008) did not find any association between high parity (5 or more

children versus 0–4 children) and disability among older women. Teachman (2010) examined the presence of children in the home and work-related limitations and found the presence of children has a protective effect on work related limitations among women. International data has yielded indistinct findings as well. Hank (2010) observed poorer physical health among high parity old women in East Germany, but not in West Germany, no other parity status was associated with physical health. Among a cohort of older British women (born 1923–1949), childlessness and high parity were both associated with activity limitation and poorer health (Read, Grundy, & Wolf, 2011).

Although the empirical research linking parity and later life functional health is scant, there is theoretical and empirical evidence suggesting that parity may be potential predictor of disability onset. Moreover, given the previous literature examining racial differentials in parity status among the earliest twentieth century cohorts, we anticipate racial variations in the capacity of parity status to predict disability onset. Because of the severe social discrimination that Black women from these cohorts experienced in addition to health inequalities such as malnutrition, lack of medical care, and higher rates of STIs, we suggest that parity status among Black women born before 1925 may reflect biological variations in the susceptibility of chronic conditions and disability due to poor environmental conditions that occurred before and during childbearing ages. We elected to explore Activities of Daily Living (ADL) impairment as a measure of disability because it represents a loss of independence which has important individual and societal implications such as lower quality of life and a need for more in/formal care (Field & Jette, 2007). To account for the competing social hypotheses (i.e., social mechanisms via social relationships and socioeconomic status), we include numerous controls including socio-demographic characteristics, health risk behaviors, morbidity status, and social support measures.

Methods

Data

The data for this research comes from the Health and Retirement Study (HRS) (1998–2008). More specifically, the most recent RAND HRS Data file (k) was used, which is a longitudinal data set created from original HRS data by the National Institute on Aging and the Social Security Administration (RAND, 2011). The HRS is a nationally-representative, longitudinal panel study sponsored by the National Institute of Aging (NIA) (grant number NIA U01AG009740) and conducted by the University of Michigan's Institute for Social Research. Structured face to face interviews were collected in 1992, and follow-up telephone surveys have been collected every two years. The primary focus of the HRS was to describe the health, well-being, family structure, finances, and employment history of late midlife (ages 51–61 years) Americans. A cohort of late midlife adults (born 1931–1941) was sampled using a multi-stage, clustered probability frame. Interviews were collected for age-eligible respondents and their spouses (regardless of age), and the initial sample size was approximately 12,650 people from about 7,700 households. In 1998, the HRS merged with another on-going survey, the Asset and Health Dynamics among the Oldest Old (AHEAD), which consisted of respondents born in 1923 or before. Additionally, two more cohorts were added in 1998, the War Baby (WB) cohort (born 1942–1947) and the Children of Depression (CODA) (born 1924–1930). The sample size, in 1998, including all four cohorts (and respondent spouses) was 21,384 (HRS, 2011).

Measures

Disability onset was the outcome of interest, and was operationalized as Activities of Daily Living (ADL) impairment. An ADL index was created using five standard ADL measures: 1) difficulty bathing/showering; 2) difficulty dressing; 3) difficulty eating; 4) difficulty

getting in/out of bed; and 5) difficulty walking across the room. The ADL measures were all self-reported. Respondents who had “any difficulty” for each task were assigned a value of “1” for that ADL task. Respondents who had difficulty completing all of the ADL tasks were assigned the maximum value of the index (5) and those respondents who had no difficulty completing any of the ADL tasks were assigned the lowest value of the index (0). The summary index was collapsed into dichotomous measures (no difficulty vs. any difficulty) for ADL disability. Respondents who previously reported no difficulty and then reported any difficulty in the subsequent wave experienced “onset” (see Figure 1).

The independent variable of interest was parity. A four-category variable was created from the number of children ever born, where nulliparous, single-child parity, low parity (i.e., 2–3 children) (reference), and high parity (i.e., 4+ children) were the categories. Other independent variables included in the analysis were socio-demographic characteristics, health risk behaviors, morbidity status, and social support. Socio-demographic characteristics were comprised of age, race, education, and income. With the exception of household income, all socio-demographic characteristics were treated as time-fixed. A continuous measure of age (i.e., years) at Wave 4 (baseline) was used. A dichotomous variable was created for race, where Black=1. It is important to note that other racial and ethnic categories were not used because of a lack of observations among specific parity statuses such as childlessness. A total of 39 cases were omitted from the risk group because the respondents did not identify as either Black or White. Education was measured as the number of years of formal education. Household income was treated as a time-varying covariate. Household income was measured as a continuous variable and was scaled by \$10,000.

Three health risk behaviors were included in the analysis: smoking status, alcohol consumption, and body mass index (BMI). Smoking status was measured as a trichotomous variable with never smoked (reference), former smoker, and current smoker as the categories. The smoking status measure was constructed from two questions: 1) Have you ever smoked cigarettes? and 2) Do you smoke cigarettes now? Alcohol consumption was measured at a time-varying dichotomous measure, where “ever drinks” = 1. Respondents were asked whether they drink any alcoholic beverages in the last three months. A categorical measure of BMI was constructed from self-reported weight and height measures; the categories were based on the Centers for Control and Prevention’s BMI guidelines (CDC, 2011) and included underweight (below 18.5), healthy weight (normal) (18.5–24.9), and overweight (above 25). All three health risk behavior measures were treated as time-varying. Physical activity was not included in the analysis because only information about vigorous activity was able for all observational waves. Other types of physical activity measures such as low-impact physical activity would be more appropriate for a sample of older women; therefore, physical activity was omitted from these analyses.

Morbidity status was measured using number of chronic conditions and mental status. Number of chronic conditions was measured as an index with seven individual chronic conditions: 1) arthritis; 2) cancer; 3) diabetes; 4) heart problems; 5) high blood pressure; 6) lung disease; and 7) stroke. A dichotomous measure was created for each condition, where ever having the condition=1. Additionally, a dichotomous measure of psychological problems (i.e., emotional, nervous, or psychiatric problems), where yes=1 was used to assess mental health. All of the chronic conditions and the mental health measure were based on self-reported physician diagnoses and reflected ever having the condition. Both morbidity status measures were treated as time-varying covariates and assessed at each wave.

Social support measures included information about family and friends. All social support measures were treated as time-varying covariates. Three measures of social support were

included: 1) whether the respondent was partnered or married; 2) whether family members lived in the neighborhood; and 3) whether good friends lived in neighborhood. A dichotomous measure of partnered/married was created, where being currently partnered/married=1. Dichotomous variables were also created for both family living in the neighborhood or good friends living in the neighborhood, where yes=1.

Analytic Strategy

A subset (N=3,367) of Black and White women ages 74 years (i.e., born before 1925) or older at Wave 4 (1998) was created from the original sample. Approximately, one-third of the subset had already experienced ADL impairment. The remaining two-thirds of the subset encompassed the risk group (i.e., women without ADL impairment) (N=2,229). The analysis occurred in two steps. First, bivariate associations of race and parity status were tested to verify racial variations in parity status among the old-old. Second, discrete-time event history modeling with multiple competing (non-repeating) events was estimated using multinomial logistic regression. The risk group at the beginning of the interval was respondents without ADL disability and possible outcomes included: no difficulty (stable), any difficulty (onset), died, and attrited (see Figure 1). The initial risk group was followed in subsequent intervals, and initial disability onset was estimated. Although attrition was explicitly modeled, the results for attrited are not presented, but are available upon request from the authors. Odds ratio estimates were then ascertained for each variable. The analyses were unweighted because the attributes that the weights are calculated for (e.g., gender, race, and marital status) were included in the analysis, and prior research has suggested that the unweighted HRS data are less biased than the weighted data (Botoseneanu & Liang, 2010; Winship & Radbill, 1994). Robust standard errors were employed to adjust for within persons clustering from repeated observations.

This research examines whether risk of disability onset varies by race among the old-old, net of socio-demographic characteristics, health risk behaviors, morbidity status, and social support; therefore, a series of three models were created. In Model 1, parity, age, and race were included. Model 2 introduced an interaction term for parity status and race. Finally, Model 3 included socio-demographic characteristics, health risk behaviors, morbidity status, and social support measures as controls. This modeling strategy was utilized so that parity and race was underscored and to allow for an examination of unadjusted and adjusted hazard coefficients.

Results

Table 1 displays the bivariate associations between race and parity status among the initial risk group (N=2,229). A χ -square value of 64.24 (degrees of freedom=3) was estimated using cross-tabulation. Column percentage values are presented and significant at a p-value of less than 0.001. Approximately one quarter of Black respondents were nulliparous compared to only 13.8% of White respondents. A greater proportion of Black respondents (19.6%) had experienced single-child parity relative to White respondents (14.2%). Nearly half (47.1%) of White respondents had experienced low parity, while less than a quarter (22.5%) of Black respondents had experienced low parity. Approximately one-third (32.8%) of Black respondents had experienced high parity, whereas approximately a quarter (24.9%) of White respondents had experienced high parity. These results echo previous findings—Black women were more likely to be nulliparous or experience high parity compared to White women in the sample.

Table 2 displays the risk group descriptive statistics by parity status. Nulliparous women were slightly older than high parity women with the average age of 80.7 and 79.0 years, respectively. A higher proportion of Black respondents were nulliparous (20.1%) compared

to single-child parity (16.1%), low parity (6.2%), or high parity (15.4%) with the proportion of Black respondents being exceptionally small among low parity group. On average, high parity respondents had about one year fewer of education compared to the other parity statuses, while a smaller proportion of nulliparous (26.4%) respondents were currently married or partnered.

Table 3 presents odds ratio estimates of disability onset and death versus no disability. Approximately 14% to 19% of respondents experienced disability onset per interval (depending on the interval), while approximately 6% to 12% of respondents experienced death per interval. About 3% to 4% of respondents attrited per interval with the highest proportion attriting in Interval 1. For all models, the intercept and Likelihood Ratio were statistically significant at an alpha level of less than 0.001.

ADL Disability Onset vs. No ADL Disability

Model 1 indicated the main effect of parity status, net of age, race, and time. In Model 1, parity status was not significantly associated with increased risk of disability onset; however, older ages were linked to increased risk of disability. For every additional year of age, respondents were approximately 11% more likely to experience disability onset. Compared to White respondents, Black respondents (OR=1.39) were more likely to experience disability onset. Model 2 included a parity*race interaction term. The main effect of parity status was not significantly associated with disability onset, which indicated that White respondents (referent group) were not more or less likely to experience disability. Two of the three race*parity interactions were significant at an alpha-level of less than 0.05. Nulliparous Black women (OR=2.88) were much more likely to experience disability onset compared to White nulliparous women. Similarly, high parity Black women (OR=2.04) were more likely to experience disability onset. The magnitude of these associations was particularly striking. The risk of disability onset did not differ among low parity White and Black women; however, for nulliparous and high parity respondents the effect of parity status as a predictor of disability onset was moderated by race.

Coefficients from Model 2 were used to generate predicted probabilities. Figure 2 presents the age-adjusted predicted probabilities of ADL disability onset by race and parity status. Nulliparous Black women had a predicted probability of 0.54, whereas nulliparous White women had a predicted probability of 0.34. Similarly, a high parity Black woman had a higher predicted probability of experiencing disability onset during the study duration than high parity White women. Although not significantly different, low parity Black women had a slightly lower predicted probability of 0.28 relative to low parity White women with a predicted probability of 0.33. Nulliparous and high parity Black women were the most likely to experience disability onset with predicted probabilities exceeding or near 50%, net of age.

In Model 3, socio-demographic characteristics (i.e., education and household income), health risk behaviors, morbidity status, and social support measures were introduced as controls. From Model 2 to Model 3, the parity*race coefficients remained relatively stable across the models which suggests that the control variables did not mediate the parity*race associations. Current smokers (OR= 1.69) as well as underweight respondents (OR=1.43) were more likely to experience disability onset. Respondents who reported consuming any alcoholic beverage in the past three months (i.e., ever drinks) (OR=0.60) were less likely to experience disability onset. Both morbidity measures were significantly associated with disability risk with more chronic conditions (OR=1.27) or having a psychological condition (OR=1.75) being associated with increased risk of disability onset. The observational intervals did not express a general trend of risk of disability over the study duration; however, Interval 4 was associated with greater risk of disability onset than Interval 1 in all three models.

Died vs. No ADL Disability

In Model 1, nulliparous women (OR=1.71) and single-parity (OR=1.48) women were more likely to experience death compared to respondents without ADL disability, net of age, race, and time. Age (OR=1.17) was also associated with death, where older ages were linked to greater risk of death. Model 2 introduced a parity*race interaction term and there were no significant associations. With the inclusion of sociodemographic characteristics, health risk behaviors, morbidity status, and social support measures (Model 3), childlessness (OR=1.79) and single-child parity (OR=1.53) continued to be associated with a greater risk of death. Similarly, age (OR=1.16) also persisted in Model 3. The parity status*race interaction term continued to be non-significant. No other sociodemographic characteristics were significantly associated with risk of death. All of the health risk behaviors were linked to risk of death. Former smokers (OR=1.43), current smokers (OR=1.99), and underweight (OR=1.94) respondents had a higher likelihood of death, while respondents who ever drank (OR=0.66) and overweight respondents (OR=0.64) had a lower likelihood. More chronic conditions (OR=1.48) and having a psychological condition (OR=1.85) were associated with greater risk of death. Having good friends living in the neighborhood (OR=0.64) was associated with a decreased risk of death.

The observational intervals showed a general trend of higher risk of death over the duration of the study. Compared to Interval 1, Interval 2, Interval 4, and Interval 5 were all associated with increased likelihood of death. As the respondents moved through the study waves, the risk of death increased over time with the exception of Interval 3.

Discussion

These findings demonstrate a clear Black-White difference in the predicative capacity of parity status for disability onset. Nulliparous and high parity Black women, among the old-old, were more likely to experience disability onset compared to White women with the same parity status. With the inclusion of the control variables, the parity*race interaction terms remained relatively stable across the models. This suggests that the moderating effect of race on parity status was not appreciably influenced by socio-demographic characteristics, health risk behaviors, morbidity status, or social support. In relation to nulliparous parity status, we may be viewing the long-reaching implications of poor health during childbearing years, which influenced fecundity, due to severe social segregation and discrimination among Black women born before 1925. These women may represent a particularly vulnerable group for ADL disability onset, and coupled with the lack of instrumental social support from adult living children, may be in need of targeted functional health intervention or formal care. Additionally, high parity Black women were more likely to experience disability onset. Although these women were able to conceive and carry to term, the social conditions which led to high parity may reflect a lack of resources to regulate fertility. Furthermore, this research demonstrated higher mortality rates among nulliparous and single-parity women, which did not vary by race and were relatively stable across models. High parity women were not at an increased risk of mortality relative to women without disability.

Limitations

These results must be viewed in light of several limitations. One of the main limitations of this research is the inability to distinguish between voluntary and involuntary childlessness. It is possible that involuntary childlessness among White women is predictive of disability onset and also reflects important social and biological disparities during childbearing years. Another limitation of this research is the two-year interval. It cannot be established at what point during the two-year interval respondents developed disability and whether respondents

recovered from disability onset. Although annual or semi-annual data would have led to more precise estimates of disability onset, using the HRS had many benefits including many years of data, functional health measures, and a nationally representative group of older women. Selection effects due to mortality are also a key limitation. This research investigates parity as a predictor of health outcomes among the old-old. Two forms of mortality bias are of particular importance for this research: maternal mortality (especially for Black women with reduced access to healthcare during childbearing years) and premature mortality in later life (e.g., midlife), which would have occurred before the women entered the survey. Because this cohort represents the old-old, the women in the sample may be the most resilient women, which may be particularly true of Black women who survived acute discrimination and harsh social conditions. Mortality was modeled as a competing event; however, due to the modeling strategy, mortality following disability onset was not assessed. Attrition (i.e., lost to follow-up) is another limitation; however, attrition was accounted for by being modeled as a competing event. Attrition disproportionately occurred among women with limited resources (i.e., lower education attainment) and poorer health, so it is very likely that disability onset is underestimated. Further limitations stem from the operationalization of central measures. Disability measures were self-reported and severity of disability was not assessed. Although there are several key limitations of this research, there is also strong evidence to suggest that there are racial variations in the capacity of parity status to predict disability onset.

Conclusion

Using a life course perspective, this research alludes to the long-ranging implications of parity status within a socio-historical context and predictions of later life health outcomes. Although we cannot explicitly test biological variations and predispositions to disability onset due to earlier life conditions, these findings point toward a critical period (see Ferraro & Kelley-Moore, 2003; Ferraro, Shippee, & Schafer, 2009; Kuh et al., 2003). This is a particularly intriguing notion when considering that other social variables such as socio-demographic characteristics (i.e., education and income) and social support measures did not substantially influence the parity and race interactions. The socio-historical conditions which led to racial variations in parity status may also represent disparities in susceptibility for later life functional health. Parity status among Black women in the old-old cohorts may be a salient proxy of social conditions including intense social discrimination and segregation. Prior research underscores malnutrition, lack of access to healthcare, increased rates of STIs, and general poorer health during childbearing years as determinants of higher rates of childlessness and single-child parity among Black women for the earliest twentieth century cohorts. Many of these same determinants such as such as lack of access to healthcare may have shaped rates of high parity. Although the underlying mechanisms that led to greater proportions of childlessness and high parity among the old-old Black women is contested, most scholars acknowledge that contraception was used by these women to some extent (see Masnick & McFalls, 1976). It is possible that Black women who were able to regulate their fertility did not experience similar scarring from negative social conditions during childbearing years.

It is also important to note that this research did not reveal any racial variations in regard to mortality without disability. It is possible that types of diseases which cause mortality without disability do not vary by race and parity. Using disability onset as a measure may have uncovered later life health outcome disparities by race and parity that would go unnoticed if only mortality were examined. However, we did not examine risk of death among respondents with disability, which is an important limitation. In the future, we intend to explore whether the predictive capacity of parity status varies by race for mortality for women with and without disability. Additionally, research exploring cause-specific

mortality may elucidate the relationship between disability and mortality in relation to parity status and race. Finally, we recommend future research investigating other functional health and later life health outcomes. While the social conditions which helped to create racial variations in parity status among the old-old may no longer be as severe, they still persist as evidenced by the poorer health outcomes of Black infants (e.g., low birth weight and higher infant mortality) (see Geronimus, 1992; 1996; Mathews & MacDorman, 2011). Identifying racial and ethnic variations and potential determinants of parity status among more contemporary cohorts may have important consequences for later life health disparities.

Acknowledgments

This research was supported in part by an NIA training grant to the Population Studies Center at the University of Michigan (T32 AG000221). Additionally, Latham would like to gratefully acknowledge use of the services and facilities of the Population Studies Center at the University of Michigan, funded by NICHD Center Grant (R24 HD041028).

References

- Botosaneanu A, Liang J. Social stratification of body weight trajectory in middle-age and older Americans: Results from a 14-Year Longitudinal Study. *Journal of Aging and Health*. 2011; 23(3): 454–480. [PubMed: 21068396]
- Boyd RL. Racial differences in childlessness: A centennial review. *Sociological Perspectives*. 1989:183–199.
- Centers for Disease Control and Prevention (CDC). About BMI for Adults. Atlanta, GA: Centers for Disease Control and Prevention; 2011. Retrieved August 10th, 2011. (http://www.cdc.gov/healthyweight/assessing/bmi/adult_bmi/index.html)
- Cutright P, Shorter E. The Effects of Health of the Completed Fertility of Nonwhite and White US Women born Between 1867 and 1935. *Journal of Social History*. 1979; 13(2):191–217. [PubMed: 11632376]
- Davis NJ. Childless and single-childed women in early twentieth-century America. *Journal of Family Issues*. 1982; 3(4):431–458. [PubMed: 12264676]
- Doblhammer G. Reproductive history and mortality later in life: a comparative study of England and Wales and Austria. *Population Studies*. 2000; 54(2):169–176. [PubMed: 11624633]
- Farley, R. Growth of the Black population: A study of demographic trends. Markham Pub. Co; 1970.
- Evans M. American fertility patterns: A comparison of white and nonwhite cohorts born 1903–56. *Population and development review*. 1986:267–293.
- Ferraro KF, Kelley-Moore JA. Cumulative Disadvantage and Health: Long-Term Consequences of Obesity? *American Sociological Review*. 2003:707–729. [PubMed: 22581979]
- Ferraro KF, Shippee TP, Schafer MH. Cumulative inequality theory for research on aging and the life course. *Handbook of theories of aging*. 2009:413–433.
- Field, MJ.; Jette, AM. The future of disability in America. Natl Academy Pr; 2007.
- Geronimus AT. The weathering hypothesis and the health of African-American women and infants: evidence and speculations. *Ethnicity & disease*. 1992; 2(3):207. [PubMed: 1467758]
- Geronimus AT. Black/white differences in the relationship of maternal age to birthweight: a population-based test of the weathering hypothesis. *Social Science & Medicine*. 1996; 42(4):589–597. [PubMed: 8643983]
- Gillespie R. Contextualizing voluntary childlessness within a postmodern model of reproduction: implications for health and social needs. *Critical Social Policy*. 2001; 21:2, 139–159.
- Glauber R. Marriage and the motherhood wage penalty among African Americans, Hispanics, and Whites. *Journal of Marriage and Family*. 2007; 69:951–961.
- Grundy E, Tomassini C. Fertility history and health in later life: a record linkage study in England and Wales. *Social Science & Medicine*. 2005; 61(1):217–228. [PubMed: 15847974]
- Hank K. Childbearing history, later-life health, and mortality in Germany. *Population Studies*. 2010; 64(3):275–291. [PubMed: 20845224]

- Health and Retirement Study (HRS), public use dataset. Produced and distributed by the University of Michigan with funding from the National Institute on Aging (grant number NIA U01AG009740). Ann Arbor, MI: 2011.
- Heck KE, Schoendorf KC, Ventura SJ, Kiely JL. Delayed childbearing by education level in the United States: 1969–1994. *Maternal and child health journal*. 1997; 1(2):81–88. [PubMed: 10728230]
- Henretta JC. Early childbearing, marital status, and women's health and mortality after age 50. *Journal of Health and Social Behavior*. 2007; 48(3):254–266. [PubMed: 17982867]
- Hurt LS, Ronsmans C, Thomas SL. The effect of number of births on women's mortality: Systematic review of the evidence for women who have completed their childbearing. *Population Studies*. 2006; 60:55–71. [PubMed: 16464775]
- Kendig H, Dykstra PA, Van Gaalen RI, Melkas T. Health of aging parents and childless individuals. *Journal of Family Issues*. 2007; 28:11, 1457–1486.
- Kuh D, Ben-Shlomo Y, Lynch J, Hallqvist J, Power C. Life course epidemiology. *Journal of Epidemiology and Community Health*. 2003; 57(10):778. [PubMed: 14573579]
- Masnick GS, McFalls JA Jr. A New Perspective on the Twentieth-Century American Fertility Swing. *Journal of Family History*. 1976
- Mathews, TJ.; MacDorman, MF. National vital statistics reports. Hyattsville, MD: National Center for Health Statistics; 2011. Infant mortality statistics from the 2007 period linked birth/infant death data set; p. 59-56.
- Morgan SP. Late nineteenth-and early twentieth-century childlessness. *American Journal of Sociology*. 1991:779–807.
- Poston DL Jr, Gotard E. Trends in childlessness in the United States: 1910–1975. *Biodemography and Social Biology*. 1977; 24(3):212–224.
- Poston DL Jr, Kramer KB. Voluntary and involuntary childlessness in the United States: 1955–1973. *Biodemography and Social Biology*. 1983; 30(3):290–306.
- RAND HRS Data, Version K. Produced by the RAND Center for the Study of Aging, with funding from the National Institute on Aging and the Social Security Administration. Santa Monica, CA: 2011.
- Read S, Grundy E, Wolf DA. Fertility history, health, and health changes in later life: A panel study of British women and men born 1923-49. *Population Studies*. 2011; 65(2):201–215. [PubMed: 21614727]
- Retherford RD, Luther NY. Are fertility differentials by education converging in the United States? *Genus*. 1996:13–37. [PubMed: 12320815]
- Rowland DT. Historical trends in childlessness. *Journal of Family Issues*. 2007; 28(10):1311–1337.
- Schoen R. Insights from parity status life tables for the 20th century US. *Social Science Research*. 2006; 35(1):29–39.
- Spence NJ. The long-term consequences of childbearing. *Research on Aging*. 2008; 30(6):722–751. [PubMed: 19122886]
- Taniguchi H. The timing of childbearing and women's wages. *Journal of Marriage and the Family*. 1999:1008–1019.
- Teachman J. Family life course statuses and transitions: Relationships with health limitations. *Sociological Perspectives*. 2010; 53:2, 201–220.
- Tolnay SE, Guest AM. Childlessness in a transitional population: The United States at the turn of the century. *Journal of Family History*. 1982; 7(2):200–219. [PubMed: 11611792]
- Wadsworth M. Health inequalities in the life course perspective. *Social Science & Medicine*. 1997; 44(6):859–869. [PubMed: 9080567]
- Westoff CF. Differential fertility in the United States: 1900 to 1952. *American Sociological Review*. 1954; 19(5):549–561.
- Winship C, Radbill L. Sampling weights and regression analysis. *Sociological Methods & Research*. 1994; 23(2):230–257.
- Wright P. An examination of factors influencing black fertility decline in the Mississippi Delta 1880–1930. *Biodemography and Social Biology*. 1989; 36(3–4):213–239.

Zhang Z, Hayward MD. Childlessness and the psychological well-being of older persons. *Journal of Gerontology, Social Sciences*. 2001; 56B:5, S331–S320.

Figure 1.
Illustration of risk group and potential outcomes for ADL disability

Figure 2.
Age-Adjusted Predicated Probabilities of ADL Disability Onset by Race and Parity Status

Table 1

Bivariate Association of Race and Parity Status among the Old-Old Women without ADL Disability
(N=2,229)

	White	Black	Total
Parity Status			
Nulliparous	13.8% (n=270)	25.1% (n=68)	15.2% (n=338)
Single-Child Parity	14.2% (n=277)	19.6% (n=53)	14.8% (n=330)
Low Parity	47.1% (n=923)	22.5% (n=61)	44.1% (n=984)
High Parity	24.9% (n=488)	32.8% (n=89)	25.9% (n=577)
Total	100.0% (n=1,958)	100.0% (n=271)	100.0% (n=2,229)

Source: *The Health and Retirement Study (HRS) (1998)*

Notes: Chi-square=63.24 (df=3); p-value <0.001

Table 2

Descriptive Statistics of Risk Group by Parity Status (N=2,229)

	Nulliparous (n=338)	Single- Child Parity (n=330)	Low Parity (n=984)	High Parity (n=577)	Total Sample (n=2,229)
Socio-demographic Characteristics:					
Age	80.7 (5.2)	80.2 (4.7)	79.9 (4.9)	79.0 (4.5)	79.9 (4.8)
Race:					
White	79.9%	83.9%	93.8%	84.6%	87.8%
Black	20.1%	16.1%	6.2%	15.4%	12.2%
Education (yrs.)	11.7 (3.5)	11.6 (2.9)	11.8 (2.9)	10.4 (3.6)	11.4 (3.3)
Household Income (\$10,000)	2.8 (3.7)	2.7 (3.8)	2.9 (3.1)	2.6 (5.7)	2.8 (4.1)
Health Risk Behaviors:					
Smoking Status:					
Never Smoked	59.1%	56.7%	63.4%	62.0%	61.4%
Former Smoker	31.4%	38.5%	30.5%	31.1%	32.0%
Current Smoker	9.5%	4.8%	6.1%	6.9%	6.6%
Ever Drinks (yes=1)	40.5%	37.0%	35.1%	29.1%	34.6%
Body Mass Index:					
Underweight	10.4%	7.4%	6.1%	5.1%	6.7%
Health Weight	53.9%	55.3%	52.9%	46.2%	51.6%
Overweight	35.7%	37.3%	41.0%	48.7%	41.7%
Morbidity Status:					
Num. of Chronic Conditions	1.6 (1.1)	1.7 (1.1)	1.7 (1.2)	1.8 (1.3)	1.7 (1.2)
Psychological Condition (yes=1)	6.8%	8.5%	8.5%	9.9%	8.6%
Social Support Measures:					
Partnered/Married (yes=1)	26.4%	35.5%	35.6%	32.6%	33.4%
Relatives Living Nearby (yes=1)	33.1%	24.5%	31.8%	38.3%	32.6%
Friends Living Nearby (yes=1)	79.9%	74.8%	75.5%	73.1%	75.4%

Source: *The Health and Retirement Study (HRS) (1998)*

Notes: Percentage distributions are shown for categorical variables; means and (standard deviations) are shown for continuous variables.

Table 3

Odds Ratios of Disability Onset and Death, by Parity, Socio-demographic Characteristics, Health Risk Behaviors, Morbidity Status, Social Support Measures, Interactions, and Time (Initial Risk Group: N=2,229)^a

	Onset of Disability ^b			Died ^b		
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
Parity:						
Nulliparous	1.19	1.04	1.12	1.71***	1.70**	1.79**
Single-child Parity	0.94	0.92	0.91	1.48**	1.54**	1.53**
Low Parity (2-3 children) (ref.)						
High Parity (4+ children)	1.16	1.09	1.01	1.24	1.22	1.18
Interactions:						
Nulliparous*Black		2.88**	2.75**		1.08	1.10
Single-child Parity*Black		1.58	1.53		0.75	0.56
High Parity*Black		2.04*	2.07*		1.16	1.09
Socio-demographic Characteristics:						
Age	1.11***	1.11***	1.11***	1.17***	1.17***	1.16***
Race:						
White (ref.)						
Black	1.39**	0.80	0.71	1.18	1.19	1.25
Education (yrs.)			0.99			0.99
Household Income (\$10,000)			1.00			0.99
Health Risk Behaviors:						
Smoking Status:						
Never Smoked (ref.)						
Former Smoker			1.03			1.43**
Current Smoker			1.69***			1.99***
Ever Drinks (yes=1)			0.60***			0.66***
Body Mass Index:						
Underweight			1.43**			1.94***

	Onset of Disability ^b			Died ^b		
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
Health Weight (ref.)						
Overweight		1.07				0.64***
Morbidity Status:						
Chronic Conditions		1.27***				1.48***
Psychological Condition (yes=1)		1.75***				1.85***
Social Support Measures:						
Partnered/Married (yes=1)		1.03				0.98
Relatives Living Nearby		1.10				0.93
Friends Living Nearby		0.88				0.64***
Time:						
Interval 1 (ref.)						
Interval 2	1.18	1.18	1.16	1.34*	1.34*	1.30*
Interval 3	0.98	0.99	0.95	1.02	1.02	0.91
Interval 4	1.52***	1.54***	1.43**	1.70***	1.70***	1.39*
Interval 5	1.29	1.31*	1.21	2.47***	2.47***	2.00***
Intercept	-9.93***	-9.86***	-10.02***	-14.97***	-14.93***	-14.58***
Likelihood Ratio	418.80***	434.56***	803.22***	418.80***	434.56***	803.22***
Degrees of Freedom	9	12	24	9	12	24

Source: RAND Health and Retirement Study (HRS) Data (1998–2008)

Notes:

^a N=6,260 person-intervals;

^b Compared to women without disability;

* 0.05 p < 0.01;

** 0.01 p < 0.001;

*** p 0.001