

## Original Article

# Childhood Conditions and Multimorbidity Among Older Adults

Gregory Pavela,<sup>1</sup> and Kenzie Latham,<sup>2</sup>

<sup>1</sup>Office of Energetics, Nutrition Obesity Research Centre, University of Alabama at Birmingham. <sup>2</sup>Department of Sociology, Indiana University—Purdue University of Indianapolis.

Correspondence should be addressed to Gregory Pavela, PhD, University of Alabama at Birmingham, 407 Lister Hill Library, 1700 University Boulevard, Birmingham, AL 35226. E-mail: [gregpavela@gmail.com](mailto:gregpavela@gmail.com).

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## Abstract

**Objectives:** This research tests whether childhood conditions are associated with trajectories of chronic conditions among older adults.

**Methods:** Using data from the Health and Retirement Study (1992–2008), a series of hierarchical linear models are used to estimate number of chronic conditions at survey midpoint and the rate of increase in chronic conditions across 18 years of data.

**Results:** Results suggest that lower childhood socioeconomic status (SES) and poor childhood health are associated with increased number of chronic conditions; however, childhood SES is no longer associated with chronic conditions after adjustment for adult SES and adult health. Poor childhood health continues to be associated with total number of chronic conditions after adjustment for adult SES and health. Rate of change in chronic conditions was not associated with childhood conditions. Results from a multinomial logistic regression model further indicated that the association between childhood conditions and adult multimorbidity increased at higher levels of multimorbidity.

**Discussion:** This research adds to the evidence that early life conditions have a lasting influence on adult health, and that their influence may be independent of adult health and SES.

**Keywords:** aging—life course—life course epidemiology—multilevel models—multimorbidity—socioeconomic status—U.S

Chronic conditions affect a significant number of older Americans and are a matter of concern in the United States (Ward & Schiller, 2013). The burden of chronic disease differs by sex, race, and socioeconomic status (SES); however, another potential source of differentiation, early life conditions, has been less well characterized. The importance of early life conditions for later life health outcomes has gained prominence among health and aging scholars from a variety of disciplines as evidence of the “long arm” of childhood extends into multiple domains of health including morbidity (Blackwell, Hayward, & Crimmins, 2001), functional status (Bowen,

2009; Bowen & González, 2010; Haas, 2008), and mortality (Hayward & Gorman, 2004). This research tests whether childhood conditions are associated with trajectories of chronic conditions among adults, and whether these associations are explained by adult SES and health. The extant literature exploring risk factors of multimorbidity is limited by an overreliance of cross-sectional data (Marengoni et al., 2011). This research attempts to address these gaps by employing 16 years of longitudinal data (along with retrospective childhood data) to explore whether early life circumstances including economic hardship and poor childhood health are associated with

trajectories of chronic conditions among a late midlife American cohort.

Multimorbidity (also known as comorbidity) is the co-occurrence of two or more chronic health conditions (e.g., arthritis and diabetes) and has been characterized as an epidemic (Schneider, O'Donnell, & Dean, 2009). Among adults over the age of 65, 46.0% are estimated to have between two and three chronic conditions, and an additional 16.6% have four or more conditions (Ward, 2013). Among Medicare beneficiaries, the most prevalent condition is diabetes (24.3%), followed by heart failure (18%). Managing more than one chronic condition has important implications for individuals, healthcare providers, and policy makers. Generally, multimorbidity is linked to lower quality of life, barriers to healthcare, disability, institutionalization, and higher rates of mortality (Barile et al., 2012; Bayliss, Steiner, Fernald, Crane, & Main, 2003; Fried, Ferrucci, Darer, Williamson, & Anderson, 2004; Gijsen et al., 2001; Marengoni et al., 2011; Verbrugge, Lepkowski, & Imanaka, 1989) as well as increased medical expenditures (Anderson & Horvath, 2004; Druss et al., 2001).

Previous research has also documented the potential of early life conditions to affect the development of chronic conditions later in life, including hypertension, diabetes, and heart disease (Curhan et al., 1996; O'Rand & Hamil-Luker, 2005; Osmond & Barker, 2000).

Such research often adopts a disease-specific model, which is suited for the identification of disease-specific etiological factors; however, disease-specific models may underestimate the true association between social exposures and health. By modeling the accumulation of morbidities rather than a specific-disease, our research adopts a "generalized health impact" (GHI) approach (O'Campo & Urquia, 2012; White, O'Campo, Moineddin, & Matheson, 2013). This approach places specific social arrangements, such as childhood SES, at the theoretical center of analysis rather than a specific disease, and is appropriate when the research aim is to estimate the overall association between social factors of interest and cumulative health burden (Aneshensel, 2005). As childhood exposures to SES and health are likely to have a generalized impact on later health, a GHI approach may more accurately estimate the population level association between childhood conditions and later health. Whether the generalized impact of childhood conditions on later health operate through biological imprinting or pathway mechanisms is an active area of research that is often structured within a life course epidemiology (LCE) framework, one which we adopt in the present analysis.

LCE is a theoretical framework that links early life conditions with later health. LCE begins with the premise that early life events, beginning in the prenatal period and extending into adulthood, have long lasting effects on later health (Ben-Shlomo & Kuh, 2002). LCE offers two non-mutually exclusive explanations linking early events with

later health: the critical period model and the accumulation of risk model.

The critical period, or latency model, describes early life exposures which result in biological imprinting or scarring, increasing susceptibility to certain diseases later in life. Barker's fetal origins hypothesis that poor fetal and early postnatal nutrition work in combination to shape metabolic development, leading to increased cardiovascular risk in later life, is a prototypical example of the critical period model (Hales & Barker, 1992). If, after controlling for a full range of adult characteristics, an association between an early exposure and later health persists, the evidence supports (although does not prove) a critical period model. Evidence consistent with a critical period model of childhood conditions has been found for mortality, functional health, and chronic conditions. Haas (2008) found early life conditions to be associated with functional health among older adults—poor childhood health was associated with greater number of functional limitations at baseline as well as an increased rate of accumulation over time. These associations remained after adjustment for adult SES and health behaviors, suggesting an effect of childhood conditions consistent with a critical period model. Focusing on chronic conditions specifically, Blackwell and colleagues (2001) found a relationship between childhood health and lung conditions, arthritis, cancer, and cardiovascular disease consistent with a critical period model.

In contrast to the critical period model, the accumulation of risk model hypothesizes that individuals accumulate biological experiences, or "health capital," across the life-course. Rather than exert their influence through biological scarring, early life events are hypothesized to set individuals on divergent health trajectories. Health trajectories which comprise a series of correlated events (e.g., lower educational attainment leading to lower income) are known as pathways, life trajectories, or "chains of risk" (Kuh, Ben-Shlomo, Lynch, Hallqvist, & Power, 2003). Pollitt, Rose, and Kaufman (2005) found evidence of pathway effects between early life events and risk factors for cardiovascular disease, such as leisure time and alcohol consumption. Sociological research has underscored the potential of early life events to initiate these chains of risk, where childhood is both a sensitive (critical) period and catalyst for either chains of advantage or disadvantage (Umberson, Williams, Thomas, Liu, & Thomeer, 2014). Among sociological research, childhood circumstances, in particular childhood SES, is thought to have a lasting impact on adult health by influencing exposure to stressors (e.g., major life events and traumas) via social statuses (e.g., social class, race/ethnicity, gender, and age) as well as influencing psychosocial resources and coping/adaptation strategies (O'Rand & Hamil-Luker, 2005; Turner & Avison, 2003; Umberson et al., 2014). The burgeoning area of biomarker research has found evidence that childhood hardship is associated with reduced immune functioning and development of age-related chronic conditions in adults (Danese & McEwen, 2012; Danese et al.,

2007; Miller et al., 2009). Furthermore, a recent study by Fabbri and colleagues (2015), investigated whether inflammatory and anabolic hormonal biomarkers were associated with multimorbidity among a sample of adults over the age of 60 years; the authors documented a steeper increase in multimorbidity among respondents with high Interleukin 6 (IL-6), levels, an inflammatory biomarker measure.

Repeated exposure to a series of uncorrelated events can also affect later health outcomes. Support for this model has been found for cardiovascular disease (Pollitt et al., 2005), marital history and mortality (Henretta, 2010), smoking and drug dependence (Turner & Lloyd, 2003), and cortisol dysregulation (Gustafsson, Anckarsäter, Lichtenstein, Nelson, & Gustafsson, 2010). Hayward and Gorman (2004) found that much of the association between childhood SES and adult mortality was explained by midlife SES. Whether childhood conditions exert an indirect effect on adult chronic conditions, as suggested by Hayward and Gorman (2004), or a direct effect as seen in Haas (2008) remains an active area of research.

Given the serious individual and societal consequences of multimorbidity among older adults, it is vital to develop a better understanding of the origins of multimorbidity including both the biological and social determinants. Risk of multimorbidity varies by SES (Barnett et al., 2012), suggesting that social processes, potentially initiated in early-life, impact the development of multiple disease burden over the life course.

## Multimorbidity Trajectories Among Older Adults

The prevalence and correlates of multimorbidity in older adults has been well described (Marengoni et al., 2011; Ward, 2013). A systematic review of the multimorbidity literature found consistently higher prevalence of multimorbidity among older individuals, women, and individuals with lower SES (Marengoni et al., 2011). Higher prevalence has also been observed among racial/ethnic minorities (Fillenbaum, Pieper, Cohen, Cornoni-Huntley, & Guralnik, 2000; Tucker-Seeley, Li, Sorensen, & Subramanian, 2011). Less is known about population-level differences in the rate of accumulation of multimorbidities. Recent research has focused on race and ethnic differences in multimorbidity accumulation. While the focus of our research is not specifically racial differences in multimorbidity trajectories, this line of research provides evidence that the rate of accumulation of chronic conditions in older ages is not fully explained by adult demographics. Quiñones, Liang, Bennett, Xu, and Ye (2011) investigated racial and ethnic variation in multimorbidity trajectories among respondents aged 51 years and older, finding significant variation between individuals both in number of comorbidities at sample mean follow-up and in rate of multimorbidity accumulation, and that statistically significant differences in the rate of multimorbidity accumulation were not fully explained by their observed

sociodemographic and health measures. Brown, O'Rand, and Adkins (2012) also examined racial differences in chronic conditions among older adults and found that having a mother with more than a high school education was protective of baseline (i.e., at age 51) multimorbidity. A limitation of Brown and colleagues (2012), as in much of the work that investigates the influence of childhood conditions on adult health, is that childhood health is not simultaneously investigated with childhood SES as a possible factor in the observed differences in multimorbidity trajectories, making it difficult to determine whether it is the early experience of low SES or poor health which ultimately affects adult health. Haas (2007) noted this limitation and found strong evidence that childhood health is associated with a range of health outcomes among older adults, independent of childhood status. Luo and Waite (2005) also modeled childhood health and SES simultaneously, finding that childhood health and SES were both associated with chronic conditions among adults. However, Luo and Waite (2005) did not adjust for health behaviors of adults, an important potential mechanism linking childhood conditions with adult health. One of the strengths of the current research is that childhood SES and health are considered simultaneously in models that predict adult multimorbidity, with controls for adult health status and behaviors in the full model.

An important limitation of the present research, even with its relatively broad measures of childhood conditions and adult SES and health, is an inability to conclusively determine whether childhood conditions exert a direct or indirect effect on multimorbidity among adults. If childhood conditions are associated with number of chronic conditions after adult SES and health behaviors are adjusted for, evidence is consistent with a critical period model only insofar as all possible adult conditions through which childhood conditions could affect chronic conditions are considered. For this reason, we include several sets of measures through which childhood health and SES could plausibly influence number of chronic conditions among adults: adult SES, health insurance, healthcare utilization, adult weight status, and smoking status. Individuals raised in low SES homes are more likely to have low SES as adults compared to those from higher SES homes (Chetty, Hendren, Kline, Saez, & Turner, 2014) and are more likely to smoke (among women) (Jefferis, Power, Graham, & Manor, 2004) and be overweight or obese as adults (Brisbois, Farmer, & McCargar, 2012).

A second limitation of previous research on childhood conditions and multimorbidity is that it generally assumes the relationship between childhood conditions and chronic conditions to be constant across levels of multimorbidity (i.e., the association between poor childhood health and two chronic conditions is the same as it is for three chronic conditions, four conditions, etc.). Standard linear regression (as used in our main analysis) models the mean, ignoring the possibility that association between predictors and outcomes might differ across the entire distribution of the outcome (Beyerlein, 2014). We therefore supplement the

longitudinal random effects linear regression model with a cross-sectional multinomial logistic model to test whether the association between childhood conditions and number of chronic conditions is equal across levels of morbidity.

Drawing from LCE and the known variation in trajectories of multimorbidity, we expect early life disadvantage, as indicated by poor childhood health and low SES, to be associated with greater initial levels and accumulation of chronic conditions. Furthermore, we expect adult SES and health behaviors to attenuate the association between early life disadvantage and later life chronic conditions and explain between-person variation in number of chronic conditions.

## Methods

### Data

Data for this research come from Waves 1 to 9 (1992–2008) of the Health and Retirement Study [HRS; see Juster and Suzman (1995) for overview], conducted by the Institute for Social Research at the University of Michigan (HRS, 2011) and sponsored by the National Institute of Aging (grant number NIA U01AG009740). Data also come from a cleaned and user-friendly version of the HRS developed by RAND (version M) and funded by the National Institute on Aging and the Social Security Administration (RAND, 2013). The initial objective of the HRS was to describe the lives of late midlife U.S. adults including collecting in-depth information about their health, finances, family structure, and employment history. The original HRS cohort represented non-institutionalized U.S. adults aged 51–61 years in 1992 (born 1931–1941). Spouses were also interviewed, regardless of age. In 1992, face-to-face interviews were conducted, and 2-year follow-up telephone surveys have since been conducted. The initial sample size was 12,543 people (including age-eligible respondents and their spouses). By 1998, the first year in which respondents were asked about their childhood SES and health, 790 respondents had died and a further 287 respondents were dropped from the sample. The analytic sample includes the 10,584 respondents who were alive and responded to the 1998 survey. Information from HRS cohort respondents who did not respond to the 1998 survey but responded in subsequent waves is included in the analysis. Missing data was handled using multiple imputation using the SAS PROC MI and MIANALYZE procedures. All variables included in the analysis were used to impute five datasets. The majority of missing data came from respondents who were alive in 1998 but died in subsequent waves, resulting in missing data for number of chronic conditions. In 1998, data on chronic conditions was missing for 45 respondents; in 2000, 1,014 respondents had missing data; in 2002, 1,560 respondents had missing information. By 2008, 2,937 respondents had missing information on chronic conditions. Number of chronic conditions (the dependent variable) was included in the imputation model, but imputed

values of chronic conditions were not included in the analysis (Von Hippel, 2007).

## Measures

### Chronic conditions

Number of chronic conditions ever had at each wave was measured by asking respondents if a doctor had ever diagnosed them as having one of seven conditions. Responses were then used to construct an index ranging from 0 to 7 (RAND, 2013). Index values indicate the sum of seven conditions: diabetes, lung disease, cancer (with the exception of skin cancer), stroke, heart problems, arthritis, or high blood pressure.

### Childhood measures

Respondents were asked to recall their family's relative socioeconomic standing, whether their family moved for financial reasons, mother's education, father's education, father's occupation, and their childhood health. In order to assess general socioeconomic standing, respondents were asked to think about their family growing up, from birth to age 16, and whether their family "was pretty well off financially, about average, or poor." Responses to this question are dichotomously coded (1 = *poor*). To assess periods of hardship, respondents were asked if, before the age of 16, "financial difficulties ever cause[d] you or your family to move to a different place" (1 = *yes*). Parental education was measured using a series of dummy variables for father's and mother's education. Categories were defined as having more than a high school education (ref), high school degree or GED, less than a high school education, or missing information. Father's occupation/family status was coded as a series of dummy variables and defined as having a father who was consistently employed (ref), ever unemployed, absent, or missing information. Childhood health was assessed by asking respondents to rate their childhood health before the age of sixteen as either "poor, fair, good, very good, or excellent." A dichotomously coded poor childhood health measure was created with a value of 1 indicating a rating of poor or fair childhood health versus good, very good, or excellent health.

### Adult measures

Measures of adult characteristics include demographics, SES, and health status. Demographics were measured at survey baseline (1992). Nativity was dichotomously coded and defined as foreign born = 1. Race/ethnicity was trichotomously coded as white (reference), black, and Hispanic, with "other" not included in the analysis. Gender was dichotomously coded and defined as female = 1. Education was measured in years (0–17) and grand mean centered. Household income and wealth are logged, grand mean centered, and in constant 2008 dollars.

Four adult health status indicators at Wave 1 were included in the model: body mass index (BMI), smoking

status, health care utilization, and insurance coverage. BMI measures were based on self-reports and categorized into underweight (BMI < 18.5); normal weight (BMI ≥ 18.5 and < 25, reference category); overweight (BMI ≥ 25 and < 30) and obese (BMI ≥ 30). Smoking status was trichotomously coded as never smoked (reference), former smoker, and current smoker. Health care utilization was measured by asking respondents if they had visited a doctor in the past 2 years and an indicator of whether they had visited a doctor was created (1 = yes). An indicator was also included for whether a respondent reported having health insurance (1 = yes).

## Analysis

Trajectories of chronic conditions over time were analyzed using hierarchical linear models with random effects for intercepts, slopes, and covariance between intercepts and slopes. Conventional multiple regression models do not take into account the nested structure of the data and assume independence of the errors (Raudenbush & Bryk, 2002). This assumption is likely violated when analyzing panel data with repeated observations nested within individuals. Hierarchical linear models also offer intuitive estimates of variation explained both across persons and within an individual. A primary goal of this analysis is to test whether childhood conditions explain between-person variation in number of chronic conditions and rate of change in chronic conditions. In this analysis, Level 1 variables consist of repeated observations within individuals—the only time-varying variable included in the model is number of chronic conditions. Level 2 variables represent the characteristics of individuals that do not change over time, such as childhood health, race, or baseline age. Level 2 coefficients indicate predicted inter-individual differences in both the intercept and slope of chronic conditions. For example, a significant and positive association between poor childhood health and number of chronic conditions indicates that across individuals, poor childhood health is associated with a greater mean number of chronic conditions at sample mean follow-up. A significant and positive association between childhood health and rate of change (entered into the model as a childhood health × wave interaction) indicates that individuals with poor childhood health have a greater average increase in number of chronic conditions over time relative to those who did not report poor childhood health.

Wave-based trajectories of chronic conditions were modeled using age of respondents at the end of the interview and coded such that the intercept represents the estimated average number of chronic conditions at mean sample follow-up (between Waves 4 and 5). The slope represents the estimated change in number of chronic conditions between interviews across all waves. Although the Health and Retirement Study is designed to collect data from respondents every 2 years, some respondents may

complete surveys at varying intervals, thus our coding of time accounts for this variability, improving estimates of changes in chronic conditions over time. *Statistical Analysis Software* (SAS) (version 9.4) PROC GLIMMIX was used for all data management and analyses.

Models were estimated in the following order: Model 1 is an unadjusted means model estimating the number of chronic conditions at baseline and variation in chronic conditions that occurs between and within individuals; Model 2 estimates an unadjusted growth curve model; Model 3 adjusts for childhood SES conditions and health; Model 4 includes controls for demographics; Model 5 additionally adjusts for adult SES. Model 6, the full model, additionally adjusts for baseline adult health status and behaviors.

In order to test whether the association between childhood conditions and adult morbidity varies by number of chronic conditions, a multinomial logistic regression model was estimated. For this analysis, number of chronic conditions was recoded into five categories: zero, one, two, three, four, or five or more chronic conditions. Multinomial logistic regression estimates the likelihood of being in any given category versus a baseline category—zero conditions is used as the baseline category in this analysis. Regression coefficients therefore represent the estimated effect of having  $z$  chronic conditions versus zero chronic conditions associated with a one unit change in the predictor variable. Data for the multinomial logistic regression come from the 1998–2000 HRS data. We selected these time points because 1998 was the first year in which the entire HRS sample was asked about childhood conditions and to minimize reliance on imputed missing data due to attrition. Table 5 presents results from the multinomial model, which includes the full range of controls for demographics, adult SES, and adult health status and behaviors (equivalent to model 6 in the longitudinal random effects model).

## Results

Table 1 presents descriptive statistics of the analytic sample. At baseline, mean number of chronic conditions reported was 0.91 conditions. By 2008, mean number of chronic conditions had increased to 2.36. Approximately 7% of respondents reported poor or fair childhood health, 18% of respondents' families moved during childhood for financial reasons, and the majority of respondents reported having mothers and fathers with less than a high school education. Nineteen percent of the sample had a father who was at one time unemployed, and 9% had an absent father.

Tables 2 and 3 presents results from a series of hierarchical linear models estimating number of chronic conditions at baseline and change over time. Table 4 reports sample size, model fit estimates, and variance estimates. Based on results from the unconditional model (Tables 2 and 3, Model 1), overall number of chronic conditions across all time points is estimated to be 1.61 ( $p < .01$ ). Results from the unconditional model also indicate significant variation

**Table 1.** Descriptive Statistics for Analytic Sample ( $N = 10,584$ ), HRS Cohort 1992–2008

Variable	Mean	SD	Range
No. of chronic conditions			
1992	0.91	0.99	0–7
1994	1.06	1.07	0–7
1996	1.20	1.13	0–7
1998	1.38	1.19	0–7
2000	1.55	1.26	0–7
2002	1.75	1.31	0–7
2004	1.96	1.37	0–7
2006	2.17	1.40	0–7
2008	2.36	1.44	0–7
Demographics			
Age (years at baseline)	54.9	5.76	23–82
Proxy interview	0.05	0.20	0–1
Foreign born	0.10	0.31	0–1
Black	0.15	0.36	0–1
White	0.73	0.44	0–1
Hispanic	0.10	0.29	0–1
Female	0.55	0.50	0–1
Childhood conditions			
Low childhood SES	0.32	0.46	0–1
Poor or fair health	0.07	0.25	0–1
Family moved	0.18	0.38	0–1
Parent's education			
Mother < HS	0.55	0.50	0–1
Mother = HS	0.26	0.44	0–1
Mother > HS	0.10	0.30	0–1
Mother missing	0.09	0.29	0–1
Father < HS	0.56	0.49	0–1
Father = HS	0.21	0.41	0–1
Father > HS	0.11	0.31	0–1
Father missing	0.13	0.33	0–1
Father ever unemployed	0.19	0.39	0–1
Father absent	0.09	0.29	0–1
Father employed	0.70	0.46	0–1
Employment missing	0.01	0.11	0–1
Adult SES			
Household income (log)	11.41	1.59	0–17.47
Household wealth (log)	11.16	5.13	–14.6 to 27.86
Education (years)	12.11	3.27	0–17
Adult health			
Obese	0.24	0.43	0–1
Overweight	0.41	0.49	0–1
Normal weight	0.32	0.46	0–1
Underweight	0.04	0.19	0–1
Current smoker	0.26	0.44	0–1
Former smoker	0.36	0.48	0–1
Never smoker	0.38	0.49	0–1
Doctor visit	0.79	0.41	0–1
No health insurance	0.21	0.40	0–1

Note: HS = high school; SES = socioeconomic status.

in the intercept (Table 4, Model 1 variation in chronic conditions between individuals = 1.22,  $p < .01$ ). Approximately 28% of total variation in chronic conditions occurs within

individuals over time ( $0.47 / 1.22 + 0.47$ ), and 72% of the variation in chronic conditions occurs between individuals.

Tables 2 and 3, Model 2 includes a linear term for time and estimates an unconditional growth curve model. Across individuals, average number of chronic conditions increased between waves ( $b = 0.20$ ,  $p < .01$ ). Results from this model indicate that approximately 70% of the variation in chronic conditions *within* individuals is associated with linear changes over time ( $R^2 = 0.70$ ). No Level 2 variables are included in the model, thus there is no  $R^2$  for variation in chronic conditions between individuals (Singer & Willett, 2003). Model 3 includes measures of childhood conditions. Having poor or fair childhood health ( $b = 0.39$ ,  $p < .01$ ), low childhood SES ( $b = 0.06$ ,  $p < .05$ ), having a mother with less than a high school education ( $b = 0.15$ ,  $p < .01$ ), having a father with less than a high school education ( $b = 0.15$ ,  $p < .01$ ), and having a father who was absent ( $b = 0.14$ ,  $p < .01$ ) are associated with greater number of chronic conditions at sample mean follow-up. Only having a mother with less than a high school education, or high school education, relative to a college education, was associated with changes in chronic conditions over time ( $b = 0.02$ ,  $p < .01$ ;  $b = 0.02$ ,  $p < .05$ ). Although several indicators of childhood conditions are themselves significantly associated with mean number of chronic conditions in the unadjusted model, only about 1% ( $R^2$ , between individual = 0.01) of overall variation in number of chronic conditions at sample mean follow-up between individuals was explained by childhood conditions. Less than 1% of variation in changes in chronic conditions is associated with childhood conditions. No additional variation within an individual is explained because no time-varying covariates are added to the model.

Tables 2 and 3, Model 4 includes controls for nativity, race, and sex. Being foreign born was associated with a lower number of chronic conditions at sample mean follow-up ( $b = -0.22$ ,  $p < .01$ ), while being black ( $b = 0.31$ ,  $p < .01$ ) or female ( $b = 0.19$ ,  $p < .01$ ) was associated with a greater number of chronic conditions. Being foreign born or female were associated with a slower rate of increase in chronic conditions ( $b = -0.03$ ,  $p < .01$ ;  $b = -0.03$ ,  $p < .01$ , respectively). Taken together, nativity, race, and sex explained an additional 2% of variation in chronic conditions between individuals at sample mean-follow up.

Tables 2 and 3, Model 5 additionally includes measures of adult SES. Greater household income and household wealth were associated with a lower number of chronic conditions at sample mean follow-up ( $b = -0.02$ ,  $p < .01$ ;  $b = -0.04$ ,  $p < .01$ , respectively). Years of education was inversely associated with of chronic conditions ( $b = -0.03$ ,  $p < .01$ ). Taken together, measures of adult SES explained an additional 3% of variation in chronic conditions between individuals at sample-mean follow-up. After measures of adult SES are included in the model, the only childhood condition associated with mean number of chronic conditions was fair or poor child health. After controlling for adult SES, low childhood SES, and maternal and

**Table 2.** Hierarchical Linear Model Results for Total Comorbidities at Sample Mean Follow-Up, HRS Cohort 1992–2008

Variables	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Intercept, B0i	1.61 (0.01)**	1.65 (0.01)**	1.34 (0.04)**	1.24 (0.05)**	1.44 (0.05)**	0.64 (0.05)**
Proxy interview	—	—	–0.10 (0.06)	–0.01 (0.06)	–0.00 (0.06)	0.07 (0.06)
Foreign born	—	—	—	–0.22 (0.04)**	–0.26 (0.04)**	–0.17 (0.04)**
Black	—	—	—	0.31 (0.04)**	0.17 (0.04)**	0.04 (0.03)
Hispanic	—	—	—	0.00 (0.05)	–0.17 (0.04)**	–0.19 (0.05)**
Female	—	—	—	0.19 (0.02)**	0.17 (0.02)**	0.18 (0.02)**
Childhood conditions						
Fair or poor child health	—	—	0.39 (0.05)**	0.38 (0.05)**	0.34 (0.05)**	0.34 (0.05)**
Low childhood SES	—	—	0.06 (0.03)*	0.07 (0.03)*	0.04 (0.03)	0.04 (0.03)
Family moved	—	—	0.03 (0.03)	0.05 (0.03)	0.03 (0.03)	0.03 (0.03)
Parental education (>HS = ref)			—	—	—	—
Mother < HS	—	—	0.15 (0.05)**	0.15 (0.05)**	0.08 (0.05)	0.05 (0.05)
Mother = HS	—	—	0.06 (0.05)	0.08 (0.05)	0.05 (0.05)	0.03 (0.04)
Father < HS	—	—	0.15 (0.05)**	0.11 (0.05)*	0.03 (0.05)	0.00 (0.04)
Father = HS	—	—	0.08 (0.05)	0.05 (0.05)	0.00 (0.05)	–0.03 (0.05)
Father's employment		—				
Employed (ref)	—	—	—	—	—	—
Ever unemployed	—	—	0.05 (0.03)	0.05 (0.03)	0.05 (0.03)	0.04 (0.03)
Absent	—	—	0.14 (0.04)**	0.09 (0.04)*	0.07 (0.04)	0.05 (0.04)
Adult SES						
Household income	—	—	—	—	–0.02 (0.01)**	–0.02 (0.01)*
Household wealth	—	—	—	—	–0.04 (0.00)**	–0.03 (0.00)**
Education (years)	—	—	—	—	–0.03 (0.00)**	–0.02 (0.00)**
Adult health behaviors						
BMI (normal = ref)						
Obese	—	—	—	—	—	0.84 (0.03)**
Overweight	—	—	—	—	—	0.31 (0.03)**
Underweight	—	—	—	—	—	–0.11 (0.06)
Smoking (never = ref)						
Current smoker	—	—	—	—	—	0.37 (0.03)**
Former smoker	—	—	—	—	—	0.12 (0.03)**
Doctor visit	—	—	—	—	—	0.50 (0.03)**
No health insurance	—	—	—	—	—	–0.01 (0.03)

Notes: BMI = body mass index; HS = high school; SES = socioeconomic status.

\* $p < .05$ ; \*\* $p < .01$ .

paternal education, were no longer associated with number of chronic conditions. Having a mother with less than a high school education was associated with an increased rate of chronic condition accumulation over time ( $b = 0.02$ ,  $p < .05$ ), while having a father with a high school degree was associated with a slower rate of increase in chronic conditions ( $b = -0.02$ ,  $p < .05$ ).

Tables 2 and 3, Model 6 additionally controls for adult health status and behaviors. Compared to individuals who were normal weight, individuals who were obese ( $b = 0.84$ ,  $p < .01$ ) or overweight ( $0.31$ ,  $p < .01$ ) had a greater number of chronic conditions. Compared to individuals who never smoked, individuals who were current smokers ( $b = 0.37$ ,  $p < .01$ ) or former smokers ( $b = 0.12$ ,  $p < .01$ ) had a greater number of chronic conditions. Finally, individuals who reported going to the doctor in the past 2 years had a greater number of chronic conditions than those who did not report going to the doctor ( $b = 0.50$ ,  $p < .01$ ). Several of

the health indicators were also associated with changes in chronic conditions over time. Compared to normal weight individuals, those who were obese or overweight had a greater rate of increase in chronic conditions ( $b = 0.05$ ,  $p < .01$ ;  $b = 0.02$ ,  $p < .01$ , respectively). Compared to never smokers, current smokers and former smokers also had a greater rate of increase in chronic conditions ( $b = 0.05$ ,  $p < .01$ ;  $b = 0.01$ ,  $p < .01$ , respectively). Taken together, measures of health behaviors and health care access explained an additional 11% of variation in chronic conditions between individuals at sample-mean follow-up and 5% of the variation in mean change over time between individuals—the greatest increase in variation explained across all models. Poor childhood health continued to be positively associated with mean number of chronic conditions ( $b = 0.34$ ,  $p < .01$ ). Regarding rate of change over time, having a mother with less than a high school education continued to be associated with an increased rate of chronic condition accumulation

**Table 3.** Hierarchical Linear Model Results for Biennial Rate of Change in Comorbidities, HRS Cohort 1992–2008

Variables	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Slope, $\beta 1i$	—	0.20 (0.01)**	0.18 (0.01)**	0.18 (0.01)**	0.21 (0.01)**	0.16 (0.01)**
Proxy interview	—	—	0.01 (0.01)	0.00 (0.00)	0.00 (0.01)	0.01 (0.01)
Foreign born	—	—	—	–0.02 (0.01)**	–0.03 (0.01)**	–0.02 (0.01)*
Black	—	—	—	–0.00 (0.01)	–0.00 (0.01)	0.03 (0.03)
Hispanic	—	—	—	–0.01 (0.01)	–0.01 (0.01)	–0.01 (0.01)
Female	—	—	—	–0.03 (0.00)**	–0.03 (0.00)**	–0.03 (0.00)**
Childhood conditions						
Fair or poor child health	—	—	–0.01 (0.01)	–0.01 (0.01)	–0.01 (0.01)	–0.01 (0.00)
Low childhood SES	—	—	0.01 (0.00)	0.00 (0.01)	0.00 (0.00)	0.00 (0.00)
Family moved	—	—	0.00 (0.00)	0.01 (0.01)	0.00 (0.00)	–0.00 (0.00)
Parental education (>HS = ref)						
Mother < HS	—	—	0.02 (0.00)**	0.02 (0.01)**	0.02 (0.01)*	0.02 (0.01)*
Mother = HS	—	—	0.02 (0.01)*	0.01 (0.01)*	0.01 (0.01)	0.01 (0.01)
Father < HS	—	—	0.00 (0.01)	0.00 (0.00)	–0.00 (0.01)	–0.01 (0.01)
Father = HS	—	—	–0.01 (0.01)	–0.01 (0.01)	–0.02 (0.01)*	–0.02 (0.01)*
Father's employment						
Employed (ref)	—	—	—	—	—	—
Ever unemployed	—	—	0.01 (0.00)	0.00 (0.00)	0.01 (0.00)	0.01 (0.00)
Absent	—	—	0.01 (0.01)	0.01 (0.00)	0.01 (0.01)	0.01 (0.01)
Adult SES						
Household income	—	—	—	—	–0.00 (0.00)	–0.00 (0.00)
Household wealth	—	—	—	—	–0.00 (0.00)**	–0.00 (0.00)
Education (years)	—	—	—	—	–0.00 (0.00)**	–0.00 (0.00)**
Adult health behaviors						
BMI (normal = ref)						
Obese	—	—	—	—	—	0.05 (0.01)**
Overweight	—	—	—	—	—	0.02 (0.01)**
Underweight	—	—	—	—	—	–0.02 (0.01)
Smoking (never = ref)						
Current smoker	—	—	—	—	—	0.05 (0.00)**
Former smoker	—	—	—	—	—	0.01 (0.00)**
Doctor visit	—	—	—	—	—	0.01 (0.00)**
No health insurance	—	—	—	—	—	–0.00 (0.03)

Notes: BMI = body mass index; HS = high school; SES = socioeconomic status.

\* $p < .05$ ; \*\* $p < .01$ .

**Table 4.** Variance Components and Fit Statistics

Variance components	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Between individuals, mean follow-up	1.22**	1.50**	1.48**	1.45**	1.41**	1.25**
Between individuals, change over time	—	0.03**	0.03**	0.02**	0.02**	0.02*
Within individuals	0.47**	0.14**	0.14**	0.14**	0.14**	0.14**
N, Level 2	10,584	10,584	10,584	10,584	10,584	10,584
N, Level 1	73,220	73,220	73,220	73,220	73,220	73,220
Pseudo- $R^2$						
$R^2$ , between individuals, mean follow-up	—	—	.01	.03	.06	.17
$R^2$ , between individual, change over time	—	—	<.01	.01	.02	.05
$R^2$ , within individual	—	.70	.70	.70	.70	.70

Notes: Pseudo- $R^2$  calculated from this table values may differ from reported Pseudo- $R^2$  due to rounding.

\* $p < .05$ ; \*\* $p < .01$ .

over time ( $b = 0.02$ ,  $p < .05$ ), and having a father with a high school degree was associated with a slower rate of increase in chronic conditions ( $b = -0.02$ ,  $p < .05$ ).

### Multinomial Logistic Regression

In Table 5, results from the cross-sectional multinomial regression model support results from the longitudinal

**Table 5.** Multinomial Logistic Regression Results Estimating Likelihood of Morbidity Versus No Chronic Conditions, HRS Cohort 2000<sup>a</sup>

Variables	OR (95% CI)				
	One	Two	Three	Four	Five or more
<b>Childhood conditions</b>					
Fair or poor child health	1.45 (1.12–1.88)**	1.60 (1.22–2.10)**	2.11 (1.56–2.85)** <sup>b</sup>	2.31 (1.56–3.39)** <sup>b</sup>	3.76 (2.36–5.98)** <sup>b</sup>
Low childhood SES	1.00 (0.87–1.15)	1.02 (0.88–1.19)	1.06 (0.89–1.26)	1.06 (0.83–1.35)	1.16 (0.82–1.64)
Family moved	1.26 (1.06–1.49)**	1.20 (1.00–1.43)	1.14 (0.92–1.41)	1.41 (1.06–1.87)*	0.92 (0.60–1.42)
Mother < HS	1.11 (0.90–1.39)	1.08 (0.86–1.38)	1.21 (0.88–1.64)	1.46 (0.90–2.36)	1.81 (0.80–4.09)
Mother = HS	1.03 (0.84–1.27)	0.93 (0.74–1.17)	1.01 (0.74–1.37)	1.15 (0.71–1.87)	1.57 (0.69–3.58)
Father < HS	1.11 (0.90–1.37)	1.13 (0.90–1.43)	1.18 (0.87–1.60)	0.88 (0.56–1.36)	1.06 (0.52–2.12)
Father = HS	1.04 (0.85–1.29)	1.05 (0.83–1.33)	1.18 (0.86–1.61)	0.67 (0.42–1.07)	0.70 (0.33–1.50)
Father employed (ref)	1.00	1.00	1.00	1.00	1.00
Father ever unemployed	1.02 (0.87–1.20)	1.03 (0.87–1.23)	1.15 (0.94–1.41)	1.20 (0.91–1.58)	1.19 (0.80–1.77)
Father absent	0.95 (0.76–1.19)	1.10 (0.87–1.39)	1.23 (0.94–1.62) <sup>b</sup>	0.88 (0.60–1.29)	1.18 (0.71–1.96)
<b>Adult health</b>					
Obese (ref = normal)	1.98 (1.68–2.34)**	3.15 (2.64–3.75)** <sup>b</sup>	5.36 (4.33–6.63)** <sup>b</sup>	7.87 (5.81–10.7)** <sup>b</sup>	7.36 (4.74–11.4)** <sup>b</sup>
Overweight	1.40 (1.23–1.60)**	1.76 (1.52–2.03)** <sup>b</sup>	2.08 (1.71–2.50)** <sup>b</sup>	2.19 (1.63–2.94)** <sup>b</sup>	2.27 (1.47–3.52)** <sup>b</sup>
Underweight	0.89 (0.66–1.20)	1.00 (0.71–1.41)	1.03 (0.65–1.64)	1.19 (0.61–2.31)	1.39 (0.59–3.44)
Current smoker (ref = never)	0.98 (0.84–1.14)	0.95 (0.80–1.13)	1.33 (1.07–1.65)** <sup>b</sup>	1.62 (1.19–2.19)** <sup>b</sup>	2.26 (1.45–3.53)** <sup>b</sup>
Former smoker	1.08 (0.94–1.23)	1.18 (1.03–1.36)*	1.63 (1.37–1.94)** <sup>b</sup>	1.69 (1.32–2.18)** <sup>b</sup>	2.22 (1.52–3.27)** <sup>b</sup>
Doctor visit	2.24 (1.87–2.69)** <sup>b</sup>	4.92 (3.85–6.29)** <sup>b</sup>	7.34 (5.09–10.6)** <sup>b</sup>	10.2 (5.18–20.0)** <sup>b</sup>	14.9 (4.98–44.3)** <sup>b</sup>
No health insurance	0.89 (0.76–1.03)	0.85 (0.71–1.00)	0.65 (0.52–0.81)** <sup>b</sup>	0.58 (0.41–0.80)** <sup>b</sup>	0.64 (0.40–1.03)
N	9,570				
–2LL					

Notes: CI = confidence interval; HS = high school; OR = odds ratio; SES = socioeconomic status.

<sup>a</sup>Includes controls for proxy status, race, nativity, sex, and adult SES.

<sup>b</sup>Coefficient significantly different ( $p < .05$ ) from coefficient estimating likelihood of one versus zero chronic conditions.

\* $p < .05$ ; \*\* $p < .01$ .

random effects model: poor childhood health was associated with an increased likelihood of having one (OR = 1.45,  $p < .01$ ), two (OR = 1.60,  $p < .01$ ), three (OR = 2.11,  $p < .01$ ), four (OR = 2.31,  $p < .01$ ), and five or more conditions (OR = 3.76,  $p < .01$ ) versus having zero chronic conditions. The apparent trend in increasing odds ratios for higher categories of chronic conditions does not indicate that the overall probability of having five or more chronic conditions is greater than having one chronic condition among individuals who report poor childhood health. Rather, it indicates that individuals who report poor childhood health are more likely to report five or more chronic conditions versus zero chronic conditions than individuals who do not report poor childhood health. Tests of coefficient equality across models indicated that the coefficients for three, four, or five or more conditions were significantly different, and greater than, the coefficient indicating the odds associated with poor childhood health of having one condition versus no chronic condition.

## Discussion

Multimorbidity affects a large and growing number of older adults. We extend past research on multimorbidity risk factors by investigating the relationship between childhood conditions and trajectories of chronic conditions among

older adults, both cross-sectionally and over time. On average, respondents had one chronic condition (defined as having diabetes, lung disease, cancer, stroke, heart problems, arthritis, or high blood pressure) at survey baseline (1992), increasing to 2.3 chronic conditions by 2008. In the unconditional means and growth models, we observed significant variation in both the total number of chronic conditions and change in number of chronic conditions over time.

The main objective of this analysis was to test whether childhood conditions were associated with chronic condition trajectories among older adults, and whether this association was explained by adult factors. We found that childhood SES was associated with mean number of chronic conditions among older adults, and that this relationship was explained after adjusting for adult SES. However, the relationship between poor childhood health and number of chronic conditions as an adult was not accounted for after adjusting for adult SES and adult health behaviors. In the fully adjusted model, on average, individuals who reported having poor or fair health as a child are estimated to have more chronic conditions than those who did not report poor health as a child. Childhood conditions explained a small but significant amount of variation in mean number of chronic conditions at sample mean follow-up (1%).

Results from the multinomial logistic regression model indicated that poor childhood health had a significantly greater association with having three or more chronic condition versus zero chronic conditions than having one chronic condition versus zero chronic conditions. Poor childhood health therefore seems to be an indicator for a significantly increased risk of multimorbidity. Prior research has underscored the early-origins of specific diseases, where certain childhood conditions are thought to be directly related to the development of chronic conditions in later life (e.g., Barker, Godfrey, Fall, Osmond, Winter, & Shaheen, 1991; Hales & Barker, 1992; White et al., 2013). Poor childhood health may be directly tied to specific chronic conditions in later life such as cardiovascular or lung disease, which contributes to early onset and increased risk of secondary conditions, which, in turn, contributes to age-related multimorbidity due to the processes associated with accelerated biological aging (MacNee, 2009; Oursler et al., 2011) as well as the social processes associated with chronic disease management such as increased exposure to stressors.

By modeling the association between childhood conditions and accumulation of morbidities rather than a specific chronic condition, we adopted a GHI approach. Although this approach is not appropriate for the identification of disease-specific etiological factors, it is appropriate when the interest is in estimating the overall association between social factors and cumulative health burden. Results suggest that the overall impact of childhood SES works primarily through adult factors, consistent with an accumulation of risk model. This is not to suggest that when the outcome of interest is a specific disease, childhood SES will always work through the same adult factors. For example, the association between childhood SES and stomach cancer mortality among adults is not fully explained by adult SES, whereas the association between childhood SES and lung cancer mortality is explained by adult SES (Smith, Hart, Blane, & Hole, 1998). As with childhood SES, the impact of childhood health on chronic conditions may have multiple and distinct etiologies. Future research may also wish to examine whether the initial disease is important in the subsequent accumulation of multimorbidities, or whether the generalized impact of SES on multimorbidity is constant across patterns of disease accumulation.

Our findings highlight the potential impact of childhood SES on the risk of chronic disease burden via adult SES among older adults, which suggests that risk of multimorbidity like other health outcomes develops over the entire life course. Given the serious corollaries of multimorbidity, the social processes that lead to greater chronic disease burden among older adults should not be discounted by researchers and policymakers.

Furthermore, the robust association between childhood health and number of chronic conditions as an adult suggests that poor childhood health may be a particularly salient form of disadvantage with broad health consequences. In line with the critical or sensitive period research, after

adjusting for adult social and health factors, childhood health remained a significant predictor of the number of chronic diseases, which suggests that childhood health may have long-reaching direct effects on adult health. Yet, we cannot disregard the social factors shaping childhood health. To illustrate, maternal health, which is an important predictor of in utero conditions and fetal development, has been directly linked to birth outcomes and child development (Archer, 2015; Osmond & Barker, 2000); however, previous research underscores the importance of structural factors related to social statuses that includes access to social support and maternal-health care services as well as exposure to stress, discrimination, and hazardous environmental factors in shaping maternal health patterns (Marmot, Friel, Bell, Houweling, & Taylor, 2008; Morello-Frosch & Shenassa, 2006; Nkansah-Amankra, Dhawain, Hussey, & Luchok, 2010).

Comparing our findings broadly to previous research on multimorbidity trajectories in older adults, we observed a similar pattern of higher initial levels of chronic conditions among female and Black respondents (Quiñones et al., 2011). Furthermore, our analysis found a protective effect of education on both the baseline number of conditions and rate of accumulation, however, education was no longer associated with between-person differences in rate of accumulation after accounting for health behaviors. Our analysis suggests that there is significant within person-variation to be explained by time-varying variables such as physical activity or health status; however, as childhood conditions are time fixed, they cannot account for within-person variation in trajectories of multimorbidity. Our results also underscore the importance of modifiable risk-factors, such as obesity and smoking, in explaining between-person differences in multimorbidity and their potential as vectors for public health interventions at reducing the overall population burden of multimorbidity.

This research provides insights into the nature of the relationship between early life disadvantage and adult multimorbidity, yet it must be viewed within the context of important research limitations. The reliance on self-reports for both early life disadvantage and chronic conditions is a major limitation of this research. Blackwell and colleagues (2001) note the potential of those with current chronic conditions to construct an explanation for their adult conditions, including having had a childhood condition. Current chronic conditions are based on physician diagnosed self-reports, which is subject to healthcare access selectivity. Additionally, the early life disadvantage measures were assessed in 1998. Early experiences of inequality may lead to premature mortality, thus the delayed measurement of early life conditions may have selected for respondents less likely to have poor childhood health or low SES, affecting the generalizability of these results and potentially understating the statistical effects of childhood SES and health. We cannot be certain, however, that the estimates are conservative ones—it is also possible that the relationship

between poor childhood health and adult multimorbidity is overestimated if (a) poor childhood health is associated with reduced survival; (b) an unobserved factor also reduces survival and increases adult multimorbidity; and (c) the unobserved factor interacts with childhood health such that their combined effect on survival is lower than their separate effects (Glymour, 2007).

Lastly, but perhaps most importantly, whether the influence of childhood conditions are set early in life and work through scarring mechanisms, or play out over time across the life course through processes of cumulative disadvantage, cannot be definitively determined in this analysis. For example, measures of adult SES relevant to the link between childhood health and adult health may have been omitted in the model—measures such as neighborhood SES (Ludwig et al., 2011) or subjective socioeconomic status (Adler, Epel, Castellazzo, & Ickovics, 2000). Acknowledging the possibility that the full model omits relevant variables, our analysis provides evidence consistent with a pathway model linking childhood SES with later-life trajectories of chronic conditions, and evidence consistent with a critical period model linking childhood health with later-life trajectories of chronic conditions and increased risk of multimorbidity.

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