Early Life Influences on Population Differences in Aging-Vector Trajectories of Functional Limitations

Steven A. Haas
Katsuya Oi
Zhangjun Zhou

Department of Sociology
Population Research Institute
Pennsylvania State University

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INTRODUCTION
Over the past two decades substantial progress has been made into understanding the determinants of population health and the distribution of health states within and between populations. Among the many strands of work within the expanding interdisciplinary field of ‘population health’ two of the most fruitful have focused on a) leveraging comparisons across populations to illuminate processes, and b) investigating the salience of life course processes and early life factors. While each of these lines of inquiry has provided important insights, very little work has attempted to integrate them on either a theoretical or empirical level. However, a number of important insights are possible only at their intersection. The present study steps into that intersection by investigating between-country differences in later-life trajectories of functional health and the role played by factors across the life course in determining within and between country heterogeneity in those trajectories.

BACKGROUND

*International Differences in Adult Health*

Recently a number of studies have compared the health and mortality of late adult/elderly populations across international contexts (Crimmins, Preston, and Cohen 2010). Such studies have been made possible by the ever growing number of high quality and internationally comparable data sets covering aging populations around the world. These include the Health and Retirement Study family of international data sets (HRS, SHARE, ELSA etc.) and the WHO Study on Global Aging (SAGE). To date these studies have usually compared population across Western Europe, sometimes including explicit comparisons to the US and have found substantial differences in a variety of health outcomes including mortality, disability, and chronic disease incidence and prevalence (Solé-Auró et al. 2015; Banks et al. 2011; Avendano et al. 2005; 2009a-b; Mackenbach et al. 2008; Thorpe, Howard, & Galactionova 2007; Banks et al. 2006).
One potential source of international variation in later life health is compositional differences in the prevalence of behavioral psychosocial and material risk factors. For example, a substantial portion of the gap in diabetes between the US and England results from the former’s higher rates of obesity (Banks, Kumari, Smith, and Zaninotto 2010). In addition, differences in smoking prevalence appears to play a partial role in international variation in mortality after age 50, especially for men (Preston, Glei, and Wilmoth 2010). However, the broader literature suggests that behavioral risk factors such as obesity, physical activity, and smoking appear to play a relatively minor role in driving current international differences in chronic disease prevalence and later life mortality risk between the US, England, and Europe (Alley, Lloyd, and Shardell 2010; Steptoe and Wikman 2010; Avendano, et al 2009a). Nor are differences likely to be driven by population differences in the level of social integration and social support (Banks, Berkman, Smith, Avendano, and Glymour 2010). Evidence does suggest that some difference may be driven by differing degrees of socioeconomic inequality and the role it plays in determining access to important salubrious resources such as health services (Banks, Berkman, Smith, Avendano, and Glymour 2010).

**Life Course Determinants of Health**

The life course perspective, which begins with the fundamental premise that health in later life is not static nor can it be divorced from the cumulative impacts of lived experience. This lived experience includes deleterious exposures or advantages associated with one’s individual placement within social and economic hierarchies and early life health insults. An essential organizing principal to the life course perspective is the idea that the life trajectories of individuals are *embedded in time and place* (Elder 1998). The contextual elements of time and place are essential because they define the opportunity and constraint structures within which
individuals express their agency. This, in part, gives rise to the idea of cohorts as an important element in life course research. Cohorts expose their members to particular constellations of sociohistorical circumstances that shape their lives as they unfold in historical time. A second, related principal of the life course perspective, is the notion that the impact that a particular set of events or transitions has on particular individuals is contingent on its timing within their lives (Elder 1998). Therefore, though the members of different cohorts may experience a common set of historical circumstances, they inevitably do so at different ages and life stages and with a unique set of prior experiences. Thus the same historical circumstances may yield very different impacts on the members of the different cohorts that experience them due to differences in timing. Prior analysis has found substantial cohort influences on trends in health and mortality. For example, Yang (2008) found that large secular declines in US mortality in the second half of the 20th century was largely driven by cohort processes. Similarly Manton et al (1997) found large increases in survival and in maintaining functional capacity across cohorts born in the late 19th and early 20th centuries. Recent research has also found a strong role played by cohort processes in shifting racial inequalities in mortality (Kramer et al. 2015; Masters et al. 2014).

A recent significant contribution of the life course perspective has been to examine what’s come to be known as ‘the long arm of childhood’ (Hayward & Gorman 2004). This refers to the lasting impact the childhood health and socioeconomic conditions have on health and life chances throughout the life course. This literature suggests that substantial gains in understanding adult health can be made from better knowledge of its determinants over the life course. It also suggests that the broad parameters of individual health trajectories and socioeconomic gradients therein may, in part, be forged very early in life, as unhealthy and
socioeconomically disadvantaged children become unhealthy and socioeconomically disadvantaged adults.

Previous research has found that those from disadvantaged backgrounds have more health-related risk factors (Blane et al. 1996) and increased risk of chronic diseases, including depression (Gilman et al. 2002), cardiovascular disease (Notkola et al. 1985), and stroke (Hart et al. 2000). Those from disadvantaged social backgrounds also tend to have worse self-rated health (Rahkonen et al. 1997), higher mortality rates (Davey Smith et al. 1997) low physical functioning at midlife (Lou and Waite 2005) and importantly for the current study, disability trajectories (Haas 2008). This body of work suggests that the impact of early life socioeconomic insults persist into adulthood net of adult social conditions, but that the later play a large role in mediating the former.

There is also an extensive body of research linking childhood health status to adult health outcomes. Much of the early research in this area was based on the use of height as a proxy of early life health and nutrition (Floud et al. 1990). Such studies typically found a negative association between achieved adult height and adult morbidity and mortality (Fogel and Costa 1997). A few studies have directly investigated the relationship between childhood and adult health using various population-based surveys. Among these are a small number of prospective investigations using the British cohort studies (Kuh and Wadsworth 1993; Kuh et al. 2002; 2006). More recently studies have investigated this relationship using retrospective reports of childhood health in US-population-based studies finding significant associations between serious infectious disease in childhood and various adult chronic diseases including cardiovascular disease, cancer, and lung conditions (Blackwell et al. 2001). Similarly, previous research has found that poor childhood health to be associated with poor self-rated health, work-limiting
disability, and chronic disease (Haas et al. 2008), functional health trajectories (Haas 2008), and physical performance (Haas 2007). The empirical evidence further suggests that the impact of childhood health insults persists net of subsequent socioeconomic attainment and health-behavior profiles.

An important limitation of life course health research is that empirical analyses have tended to focus almost exclusively on within-population processes. Very little research has explored how health or its life course determinants may vary across international contexts. This is rather odd as the life course and international-comparative perspectives would seem to have a natural affinity for each other. Nation states inherently foster differences in the experience of time and place. For example, while the 1940 birth cohort in the US and the UK share many sociohistorical circumstances there are substantial differences. For American members of the 1940 cohort their first 10 years of life were experienced as war on far-off foreign soil. While this often resulted in absent fathers, working mothers, and a degree of economic rationing, it was also followed by the post war economic boom. The British members of this cohort their first 10 years were shaped by the bombing of their cities, evacuation to the countryside, and substantial post war deprivation and reconstruction. Nation states also structure life course outcomes because they form the core of the policy environment that individuals are subject to. For example, that same 1940 British birth cohort would also experience the birth of the National Health Service in 1948- an experienced not shared by their American counterparts. Nation states vary widely in their welfare state structure and the extent to which they are centralized/fragmented, universal or exclusionary. More importantly they vary in degree to which they invest in health, buffer populations from the vicissitudes of the market (employment/income protection), provide public/social services, are active agents of economic opportunity and resource redistribution, to
insulate children and adolescents from socioeconomic deprivation and insults to their health, or
to ameliorate their more pernicious consequences (Esping-Anderson 1990; Ferrera 1996; Wood
and Gough 2006). Therefore, examining how the life course shapes the health trajectories of a
common group of cohorts across international contexts may yield valuable insights that are not
otherwise possible.

Unfortunately, little effort has been made to synthesize international comparative and life
course/developmental perspectives. Two studies have examined early life conditions as
determinants of between population differences in adult health. The first is a study by Banks et
al. (2011) was limited to a comparison of the US and England. They find significant differences
in childhood health status and the odds of transmission of childhood illness into adulthood.
However, the findings also suggest that such differences may play only a minor role determining
differences in later life health across the two populations. A larger study by McEniry (2014)
examined a much wide range of countries and focused on the role of early life conditions and the
timing of the epidemiologic transition. This study found large differences across populations in
risk of heart disease and diabetes and these were generally related to early life conditions and the
timing of and pace of the epidemiologic transition (McEniry 2014). An important limitation of
that study was that it was limited to indirect and proxy measures of childhood health status such
as rural place of birth and aggregate country-level caloric intake during childhood.

As is often the case with comparative research, one of the major obstacles limiting
progress has been the dearth of comparable population-based data across international contexts.
The growing availability of internationally comparable data funded or otherwise supported by
NIA has made cross-national comparisons easier than ever before. The current study leverages
this newly available data to begin to fill this research void by integrating the life course
perspective into international-comparative research on later life health. The goals of this paper are three fold. First, we will estimate country-specific aging-vector models of functional health for a common set of cohorts. This allows us to model trajectories of functional health over time uniquely for a given set of birth-year cohorts. Second, we will empirically compare functional health trajectories across countries. Finally, we will test for between-country differences in the relative impact of poor childhood health and socioeconomic disadvantage on functional health trajectories.

METHODS
Data
This study will utilize data from several sources. Preliminary analysis is based on data from the US and England. Data for the US comes from the Health and Retirement Survey (HRS). Begun in 1992, the HRS is a long-term panel study of approximately 28,000 Americans over the age of 50 and born before 1959, designed to investigate the economic and health transitions associated with retirement (Juster and Suzman 1995). It combines extensive information on both socioeconomic and health status. The original data collection took place using in-home face-to-face interviews and a standard survey instrument. Follow-up takes place every second year. Data for England come from the English Longitudinal Study of Ageing (ELSA) (Chesire et al. 2012). ELSA is a sample of approximately 11,000 English men and women aged 50 and older and their partners and was begun in 2002. Five follow up waves have since been completed at two-year intervals. At wave 3 (2006-07) and extensive life history survey was completed including childhood health histories. The ELSA sample covers the birth cohorts between 1908-1956. For comparability we constrain the HRS sample to these same birth cohorts. For PAA we plan to extend the analysis to continental Europe using data from the Survey of Health, Aging, and Retirement in Europe (SHARE). SHARE sampled 45,000 individuals aged 50 and older in
Central Europe. Wave 1 (2004) included Austria, Belgium, Denmark, France, Germany, Switzerland, Italy, the Netherlands, Sweden, Greece, and Spain. Wave 2 (2006-07) added Czech Republic and Poland. Wave 3 (2008-2009) consisted of the SHARELIFE survey, which collected extensive life history data including childhood health histories (Börsch-Supan and Jürges 2005). More recent waves have added Israel, Slovenia, Estonia, and Luxemburg. The structure and content of ELSA and SHARE were modeled after the HRS. The resulting strong concordance between the data sets facilitates their integration and comparison. In addition, RAND has produced harmonized versions of all the data sets used. This project will draw upon both the raw data as well as the RAND harmonized versions.

**Measurement**

Functional limitation represents the sum of 8 dichotomous items in which respondents were asked if they had any difficulty with a series of common physical tasks (Nagi 1969). These include walking several blocks, sitting for two hours, getting up from a chair after having sat for a long period, climbing a flight of stairs without resting, lifting or carrying weights over 10lbs, reaching/extending one’s arms above the shoulders, pulling or pushing large objects, and picking up a coin from a table.

The assessment of Childhood Health Status used in this analysis is based on retrospective reports. In 1998, respondents were asked to “consider your health while you were growing up, from birth to age 16. Would you say that your health during that time was excellent, very good, good, fair, or poor?” We create a dichotomous measure of childhood health disadvantage that codes those who report experiencing fair or poor childhood health as 1 and those reporting good, very good, or excellent childhood health as 0.
Previous research has analyzed the quality of retrospective childhood health histories in large nationally representative samples including the overall subjective childhood health measure used here. Haas (2007) presents the first comprehensive treatment of this measure. Using data from the PSID and the HRS he shows that the retrospective measure of overall childhood health is reliably reported over time, especially when the measure was dichotomized into a good/very good/excellent vs. fair/poor comparison. Quality of measurement did not vary substantially by gender or age. There is also no evidence that retrospective reports are subject to anchoring, by which current health status contaminates reports of health in childhood. In terms of their validity, retrospective childhood health reports have also been shown to correlate with birth weight (Haas 2007). Smith (2009) found strong correspondence between prevalence estimates of various childhood conditions drawn from retrospective reports and estimates based on external contemporaneous sources. Additionally, cohort trends in retrospective reports of measles and mumps corresponded favorably to secular declines in prevalence rates following the introduction of vaccines for these diseases. Haas and Bishop (2010) provide additional evidence of the validity of this measure using the Wisconsin Longitudinal Study. Retrospectively reported overall subjective childhood health status was strongly associated with a wide variety of common childhood conditions and activity limitations.

**Statistical Analysis**

The analysis utilizes aging-vector models to estimate trajectories functional limitations (Mirowsky and Kim 2007). Aging-vector models are a class of multilevel latent growth models in which latent intercept (baseline level) and slope (rate of change) are derived from multiple longitudinally observed moments. The within-individual model for functional limitations \( Y \) for person \( i \) at time \( t \) is a linear function of time:
\[ Y_{it} = a_{i0} + a_{i1}t + e_{it} \]  

where \( a_{i0} \) represents the individual intercept and \( a_{i1} \) represents the individual slope and \( e_{it} \) represents the individual and time specific error. Individual intercepts and slopes can then be expressed as a function of age at baseline \( (A_i) \) centered to the mean age at baseline \( (k) \), a vector of covariates \( X \), and individual random effects \( (u_{i0} & u_{i1}) \):

\[ a_{i0} = a_{00} + a_{01}(A_{i0} - k) + a_{02}(A_{i0} - k)^2 + \beta X + u_{i0} \]  
\[ a_{i1} = a_{10} + a_{11}(A_{i0} - k) + \beta X + u_{i1}. \]  

Age \( (A_{it}) \) for individual \( i \) at time \( t \) is defined as the difference between the calendar year of the survey \( S_i \) and i’s birth year \( B_i \) reflecting the age, period, cohort triad.

\[ A_{it} = S_i - B_i \]

The aging-vector model has a number of characteristics that distinguish it from more common applications of growth curve models such as cohort sequential models. Perhaps most important is that the within-individual model (1) estimates change as a function of time \( (t) \) rather than age \( (A_i) \). Age appears in the between-individual models (2) and (3). In a longitudinal study of multiple birth cohorts observed at overlapping or adjacent ages this allows each individual-year birth cohort to have its own aging trajectory parameters rather than averaging across the experiences of multiple cohorts observed at the same ages. Empirically this frees the cohort-specific aging trajectories from the functional form observed in the cross-sectional age curve (synthetic cohort) (Mirowsky and Kim 2007).

A path diagram for the model is presented in figure 1. The factor loadings of observed moments on the intercept (not shown for clarity) are the vector \([1,1,1,1,1,1,1,1,1,1]\). This establishes the intercept as the baseline value. The factor loadings for the slope (not shown)
represent time since first observation [0,2,4,6,8,10,12,14,16,18]. This establishes the slope as the annual rate of change over the observation period (1992-2010). In this model the intercept is a function of both linear age and a quadratic age term while the slope is constrained to linearity. This allows the cross sectional (and implied synthetic cohort) trajectory to take on a curvilinear shape while not imposing that functional form upon each segment of the life course experienced by any birth-year cohort. However, other functional forms are possible. The model is estimated using Mplus with the full information maximum likelihood (FIML) estimator, which accounts for sample attrition while utilizing all available observations.

[Figure 1 here]

**Preliminary Analysis**

Our preliminary analysis presents a simplified aging-vector model of functional limitations. It is limited in that it examines only the US and England and that it includes only a few covariates of interest including sex, race, and poor childhood health. FIML estimates for the HRS and ELSA are presented in table 1. Each country is modeled separately. The results for the English sample show that for the intercept there is a positive main effect for age and quadratic age term. This demonstrates that more recent cohorts have lower baseline levels of functional limitation and that the initial level of limitation is higher among each subsequent birth-year cohort. However, in the US sample the main effect of age is slightly negative with a positive quadratic age term. This reflects the trend among the most American cohorts to have higher initial levels of functional limitation compared to the cohorts that immediately preceded them. However, starting with cohorts who were in their mid-60s at the start of observation this trend is reversed. The effect of age on the latent slopes is nearly identical in the English (.005) and American (.006) samples. In both samples women and blacks have higher baseline levels of functional limitation with gender differences being slightly larger in the US while racial difference being larger among the
English. However, in the English sample there is a negative impact of being black on the latent slope. Thus while blacks have higher initial levels of functional limitations at all ages, for each cohort the slope is less steep leading towards convergence in black-white differences in functional limitation over the observation period. This is not the case in the US where a significant positive effect of being black on the latent slope reveals continued divergence between whites and blacks in the level of functional limitations.

[Table 1 here]

For both samples there was a large and significant positive impact of poor childhood health on the latent intercept. Thus across all cohorts individuals who reported experiencing poor health in childhood had .748 and .944 more initial functional limitations than their healthy English and American peers. In the US sample there is a negative effect of poor childhood health on the latent slope suggesting that the negative impact on functional health may moderate over time. To illustrate the patterns figure 2 presents aging-vector graphs for the English and American samples by childhood health status. For the sake of clarity only every 6th birth year cohort is shown. The US (HRS) is presented in the left panel and England (ELSA) is presented in the right panel. In general the shape of the two curves are similar. At the youngest ages (<60) the US has higher rates of functional limitation. However, because rates of increase among those cohorts is lower in the US by age 70 the English sample has caught up. Across all age groups there is overlap in the vectors for adjacent cohorts in the US in which the end points of preceding cohorts are higher than the starting points of succeeding cohorts. This reflects increasing levels of functional limitations (earlier onset and or more rapid accumulation) for more recent cohorts. In the English sample such over doesn’t appear until older ages.

[Figure 2 here]
Planned Analysis
For PAA we plan to extend this preliminary analysis in a number of ways. First we will extend the analysis to the SHARE dataset to examine a wider array of functional health trajectories and their early life determinants. Second, we plan to expand the range of early life factors beyond childhood health to include parental education and father’s occupation. In addition, we will include a wider array of both time varying and time invariant covariates as potential confounders or mediators of both between country differences and early life effects. These will include information on adult health behaviors and chronic disease, and adult socioeconomic attainment. Finally, we will conduct formal tests of trajectory parameters and their determinants across country contexts.
CITATIONS


Table 1. FIML Estimates From Aging-Vector Models for England and the US Cohorts Born 1908-1956

<table>
<thead>
<tr>
<th></th>
<th>England¹</th>
<th></th>
<th></th>
<th>US²</th>
<th></th>
<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Intercept</td>
<td>Slope</td>
<td>Intercept</td>
<td>Slope</td>
<td>Intercept</td>
<td>Slope</td>
</tr>
<tr>
<td>(Age-60)</td>
<td>.033*** (.003)</td>
<td>.005*** (.000)</td>
<td>-.008** (.003)</td>
<td>.006*** (.000)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Age-60)²</td>
<td>.001*** (.000)</td>
<td>—</td>
<td>.002*** (.000)</td>
<td>—</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Poor Childhood Health</td>
<td>.748*** (.080)</td>
<td>.019 (.010)</td>
<td>.944*** (.072)</td>
<td>-.012* (.005)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>.679*** (.047)</td>
<td>.001 (.006)</td>
<td>.732*** (.031)</td>
<td>.002 (.002)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Black</td>
<td>.684*** (.190)</td>
<td>-.051* (.023)</td>
<td>.462*** (.045)</td>
<td>.008* (.003)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>.884*** (.038)</td>
<td>.043*** (.004)</td>
<td>.795*** (.024)</td>
<td>.068*** (.002)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Residual variance</td>
<td>3.025*** (.085)</td>
<td>.020*** (.001)</td>
<td>3.607*** (.062)</td>
<td>.011*** (.000)</td>
<td></td>
<td></td>
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<tr>
<td>Residual correlation</td>
<td>-.040*** (.008)</td>
<td>—</td>
<td>-.075*** (.004)</td>
<td>—</td>
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</tr>
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*χ² (df)                      178.65 (26)        3725.12 (91)
*p < .05; **p < .01; ***p < .001

¹ English Longitudinal Study on Ageing
² Health and Retirement Study

Note: All coefficients are significant at the .001 level.
Figure 1. Unconditional Aging-Vector Model of Functional Limitations over 20 Years
Figure 2. Aging-vector graphs for US (HRS) and England (ELSA) by Childhood Health Status