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The Developmental Origins of Health and Disease in International Perspective

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Abstract

The developmental origins of health and disease and the comparative international approach are two important strands of research exploring population health. Despite the potential insights to be gained from integrating the two approaches, their nexus remains an underexplored frontier. The current study investigates international variation in the early life origins of health among aging cohorts in 13 countries. We examine cross-national differences in exposure to poor childhood health and socioeconomic disadvantage, whether the long-term health associations with those exposures vary across contexts, and whether they persist in the face of subsequent accumulation of socioeconomic and behavioral risk. Finally, we investigate whether childhood health and socioeconomic circumstances help explain between-country differences in later life health. The findings suggest substantial international variation in the exposure to early life health and socioeconomic insults. We also find variation in their association with later life health. However, early life factors appear to play a modest role in explaining international differences in later life health in the contexts examined here.

Keywords

Europe; SHARE; ELSA; TILDA; Life course; Co-morbidity; ADL; Mobility limitations

Introduction

The *developmental origins of health and disease* (DOHD) approach to population health has documented the indelible impacts that childhood health and social conditions have on adult health. While the DOHD literature has yielded valuable insights into the processes that generate variation in health, the vast majority of this work has focused on single populations. Scant research has sought to explicitly integrate DOHD and international-comparative

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approaches to population health. However, there is reason to believe that the nexus of these approaches may yield important insights. For example, very little is known about whether the association between early life exposures and adult health varies across contexts. Nor do we know the extent to which international variation in population health may result from differential exposure to early life insults or to variation in their long-term sequela. The current study begins to bridge this gap by examining the DOHD in international perspective.

Background

The Life Course and Developmental Origins of Health and Disease

Over the past two decades the life course perspective has become a central orienting framework with which to understand population health. The key value of the life course approach is that it provides the theoretical basis with which to understand the ‘social-biological interface’ (Bartley 2017 pp.169). It provides a framework with which to describe the etiological implications of socially embedded developmental and biological transitions as individuals grow, develop, and exercise agency bounded by the socio-historical context in which they live (Hertzman 1999). Specifically, DOHD research offers three processes (critical periods, cumulative disadvantage, and chains of risk) to describe how health trajectories may be shaped by social, material, and psychological forces experienced across the life course.

Critical period effects illustrate the nature of these socially embedded developmental transitions. In *critical period* processes, health shocks that occur during developmentally critical or sensitive periods can lead to irreversible adaptations in the structure and functioning of important biological systems (Ben Shlomo and Kuh, 2002). For example, intrauterine nutritional deprivation resulting from resource scarcity can alter a number of physiological processes in the body of the gestating fetus that persist throughout life. This includes cardio-metabolic processes (Barker, 2007), immune function (Cohen et al. 2004; McDade, 2005) and inflammatory pathways (Crimmins & Finch, 2006). Such adaptations may increase fetal survival in the short term, while manifesting in disease pathology decades later. Accordingly, four decades of research have documented the relationship between early life health and adult disease, physical functioning, and mortality (Bartley, 2017; Blane, Netuveli, & Stone, 2007).

The foundational insight of the life course perspective is that individual outcomes can only be understood within the context of the cumulative impact of lived experience. That includes insults and investments resulting from the social and physical conditions one is exposed to across the life course, from the intrauterine environment to late life. Therefore, individual health trajectories are shaped by the process of *accumulation*, in which salubrious inputs and noxious risks deriving from social, environmental, and behavioral exposures accumulate over the life course. An established body of research has shown that those from socially disadvantaged backgrounds have increased risk of chronic disease (Hart, Hole, & Davey Smith, 2000), steeper disability trajectories (Haas, 2008), and higher mortality (Davey Smith, Hart, Blane, & Hole, 1998). The impacts of early life health and social conditions are compounded by subsequent socioeconomic deprivation and health insults. Alternatively,

they may be ameliorated by upward social mobility and healthy investments (Tushar, Chin, & Jung, 2018).

In the *pathways* or *chains of risk* process, early life factors influence later life health by generating subsequent etiologies of risk. For example, childhood socioeconomic position is thought to influence disease risk not through its own independent influence per se, but through its role as a determinant of adult status attainment and that it is adult socioeconomic characteristics that are the central etiologic pathway. Thus, early life may matter only in so far as it initiates chains of risk that cascade throughout the life course (Power & Mathews, 1997). Though often presented as such, these processes need not be mutually exclusive. While risk accumulation is thought to be a fundamental process, critical periods and chains of risk are important complimentary mechanisms, helping to understand the specific etiologic processes connecting early life to adult disease (Blane et al. 2007).

More recent work has proposed a fourth process in which an array of cognitive and noncognitive human capital attributes developed very early in life, such as intelligence, conscientiousness, and sense of control act as a *selection* mechanism, sorting individuals into higher socioeconomic positions and also leading them to make health producing choices and investments, improving later life health (Mackenbach, 2010). Thus at least some of the social variation in adult health can be attributed to selection processes involving personal psychosocial attributes (Oi & Alwin, 2017; Chapman, Fiscella, Kawachi, & Duberstein, 2009; Singh-Manoux, Ferrie, Lynch, & Marmot, 2005).

Developmental Origins in International Context

Previous research has documented substantial heterogeneity in adult health across international contexts, most frequently comparing high income contexts (Solé-Auró, Michaud, Hurd, & Crimmins, 2015; Mackenbach et al., 2008). This literature has often focused on either institutional/welfare state regimes or on compositional differences in psychosocial/behavioral risk factors as determinants of international variation and has given little consideration to the life course processes discussed above. Likewise, the DOHD literature has largely lacked a comparative-international focus. The scarcity of comparative DOHD research is surprising given that a central tenet of the life course perspective is that lives are structured by the unique circumstances associated with *time* and *place*, which determine the array of opportunities and constraints within which individuals exercise agency (Elder, 1998). Consideration of national context in DOHD research is critical given that the factors that shape the distribution of risks within and between populations can be quite different than those that put particular individuals at risk (Rose, 2001). We propose that national context may influence population health through structuring the life course processes discussed above.

One way in which national context may influence population health through life course processes is through generating differential *exposure* to noxious and salubrious forces during developmentally critical/sensitive periods. As children, members of currently aging cohorts were exposed to substantial international variation in epidemiologic environments as countries differed in the timing and pace of the epidemiologic transition. For example, in the 1930s the infant mortality rate in Switzerland was 47 per 1000, while it was 66 in the UK,

80 in France, and over 100 in Italy and Spain (authors' calculations of Human Mortality Database). Indeed, geographic variation (within countries) in infant mortality was central in early DOHD research (Forsdahl, 1977). Prior research has shown that the timing and pace of the epidemiologic transition is associated with international differences in later life chronic disease risk (McEniry, 2014). Similarly, there is substantial variability in exposure to socioeconomic conditions in early life such as childhood poverty rates or human capital investments. For example, 79% of the older population in Spain has not completed upper secondary schooling while only about 15% of older Germans failed to do so (author's calculations of SHARE data). The same cohorts in those two contexts faced very different opportunities for human capital accumulation and the health benefits that derive from it.

National environmental and institutional conditions may also modulate life course processes of *accumulation* and *chains of risk*. The last century has seen a great expansion in the "social capacity for health" resulting from technological and biomedical innovation and the rise of educational and social welfare institutions (Hayward & Sheehan, 2016 pp. 355). However, the timing, degree, and nature of this health capacity expansion has been uneven. For example, across high income Western European contexts there is substantial heterogeneity in welfare state institutions in terms of orientation, structure, and generosity. This includes the extent to which they are centralized vs. fragmented and universal vs. exclusionary, the degree to which they decommodify labor and buffer individuals and families from the vicissitudes of the market, and act as agents of resource redistribution (Esping-Anderson, 1990; Wood & Gough, 2006). Such institutional differences may play an important role in creating heterogeneity in health within and between populations. Context-specific factors such as labor market conditions during the transition to adulthood, or rates of social mobility, are likely to modulate the relationship between early life socioeconomic conditions and adult health (Cutler, Huang, & Lleras-Muney, 2015). More egalitarian orientations towards human capital investment may facilitate intergenerational socioeconomic mobility, helping to ease the pernicious impacts of prior health insults or socioeconomic disadvantage. For example, recent work has found that investments made by the Indonesian government in the 1970s to expand access to primary schooling were strong enough to completely eliminate inequalities associated early life resource shocks (Tushar et al. 2018). Conversely, the effect of childhood deprivation on adult health may be magnified in societies where human capital investment was/is more heavily dependent on private/familial resources. Substantial research has investigated the population health impacts of welfare state regimes. Some evidence suggests that institutional arrangements can influence life course accumulation processes and their influence on health trajectories (Sacker, Worts, & McDonough, 2011). However, the welfare state literature overall has provided mixed, often counterintuitive, results with findings frequently dependent on the welfare state typology used (Bartley, 2017; Bergqvist, Yngwe, & Lundberg, 2013; Bambra, 2011).

The Present Study

In the present study, we address three research questions that emerge at the nexus of the DOHD and comparative-international approaches. First, to what extent does exposure to health insults and socioeconomic conditions in the critical/sensitive period of childhood among aging cohorts vary across international contexts? Second, to what extent does

variation in early life exposures explain variation in late-life health across countries? Finally, does the association that childhood exposures have with later life health vary across national context? Scant research has tackled the first and third question while a pair of studies have examined the second. However, those studies were limited in important ways. Banks, Oldfield, and Smith (2011) compared the US and England and found significant differences in childhood health status and the odds of transmission of childhood illness into adulthood, though such differences played only a minor role determining differences in health across the two populations. That study was limited to comparing two contexts. McEniry (2014) examined a wide range of countries and found large differences in risk of heart disease and diabetes and that these were related to early life conditions and the timing and pace of the epidemiologic transition. However, that study was limited to only indirect measures of childhood health including rural birth and national measures of childhood caloric intake. Incorporating newly available data, our strategy overcomes these limitations to examine a range of health outcomes among a common set of cohorts across thirteen European countries.

Data & Methods

This study utilizes data from three sources covering thirteen European countries: England, Ireland, Austria, Germany, Sweden, Denmark, Netherlands, Belgium, France, Switzerland, Spain, Italy, and Czech. These countries share a number of conceptually important characteristics (affluent market democracies, similar historical demographic trajectories and medical infrastructure, and substantial economic integration). They have also been front and center in both the DOHD literature and recent research on international differences in adult health. Similarly, the vast majority of research on the health effects of welfare state regimes has focused on Western Europe. Therefore, the results can speak directly to prior research in these areas.

Data for England come from the English Longitudinal Study of Ageing (ELSA) (Stephens, Breeze, Banks, & Nazroo, 2013). Begun in 2002, ELSA is a sample of approximately 11,000 English men and women aged 50 and older. Five follow-up waves have been completed at two-year intervals. At wave 3 (2006–07) a life history survey was completed including a childhood health history. Data for Ireland come from The Irish Longitudinal Study of Ageing (TILDA). Begun in 2009, TILDA includes approximately 8,500 Irish men and women aged 50 and older. A second wave of data was collected in 2012 (Sava, 2011). Data for continental Europe come from the Survey of Health, Ageing, and Retirement in Europe (SHARE). SHARE began in 2004 and has sampled 45,000 individuals aged 50 and older (Börsch-Supan & Jürges, 2005). Wave 3 (2008–2009) collected life history data including childhood health histories. Because of data incomparability, we excluded Greece and Poland. Both SHARE and ELSA fielded surveys in 2004. To facilitate cross-sample comparability we use 2004 as our central observation point for health outcomes and time varying covariates (e.g. smoking, income) for SHARE and ELSA. For TILDA, we use the first observation (2009). To ensure comparability of measurement we utilize both raw data as well as the RAND harmonized data.

Measurement

Outcomes—The analysis takes an integrative approach to health, examining the presence of disease as well as its subsequent cascade of physical and functional sequela (Hayward & Sheehan, 2016). We examine three outcomes. The first, a dichotomous indicator of *Multi-morbidity*, takes on values of 1 when the respondent reported having two or more of 7 common physician-diagnosed chronic conditions, and 0 otherwise. These include diabetes, stroke, high blood pressure, cardiovascular disease, cancer, chronic lung/respiratory disease, and arthritis. The other two measures are summary counts of *Functional Mobility Limitations* (Nagi, 1969), and *Activities of Daily Living Limitations* (ADLs) (Katz, 1983). Functional limitations reflect difficulty performing a series of 8 physical/mobility tasks (e.g. climbing a flight of stairs). ADLs assess difficulty performing 4 tasks necessary for independent living (i.e. bathing, toileting, dressing, and feeding). Because of skewness, mobility and ADL limitations are log transformed in the analytic models.

Exposures of Interest—Childhood health is measured using a retrospective subjective assessment of overall childhood health based on the question “*how would you rate your health as a child?*” Childhood refers to the period from birth through to 16. We dichotomize this measure as a comparison between excellent/very good/good vs fair/poor as suggested by prior literature (Haas, 2007). Previous work has shown that retrospective childhood health histories perform well. They have shown to be reliable over time and are correlated with objective measure of early life health including birth weight (Haas, 2007) and are internally consistent with retrospective reports of specific common childhood conditions (Haas & Bishop, 2010).

Childhood socioeconomic conditions were assessed by an index of parental socioeconomic characteristics similar to that used by Montez and Hayward (2014). Maternal and paternal education were standardized to the International Standard Classification of Education (ISCED). Low (ISCED=0–2), middling (ISCED=3), and high education (ISCED=4) were assigned values of 0, 1, and 2, respectively. The occupational standing of the father/main breadwinner was similarly coded into three categories representing low (e.g. manual, bluecollar), medium (e.g. white-collar service, sales, clerical, administrative) and high (e.g. professional, managerial) status and assigned values of 0, 1, and 2, respectively. The childhood socioeconomic index sums over these 3 measures and takes on values ranging from 0–6 with higher scores representing more advantaged childhoods. Because of skewness, when the childhood socioeconomic index is used as an outcome we use a log transformation.

Adult socioeconomic position is captured by three measures. Educational and occupation attainment were each coded to the same low, medium, and high categories used for parents. Finally, we include a measure of the total household income, inflation and currency adjusted to constant 2004 US dollars. Respondents were then grouped into three categories representing high (top quartile), medium (middle 50%), and low (bottom quartile) income based on their position in their country-specific income distribution.

Controls and Other Covariates—We include controls for respondent's body mass index (kg/m^2) and indicators of whether they are a current or a former smoker. Finally, we include controls for age, birth cohort, and sex. For most variables there were low levels of missing data ($\approx 5\%$ or less). However, approximately 20% of respondents were missing data on parental education. Missing data on covariates was dealt with through the use country-specific multiple imputation via the chained equations routine in Stata. Following the recommendations of Graham et al. (2007) 20 imputed data sets were constructed and included all study variables (except outcomes) as well as sampling weights.

Analytic plan

We employ mixed-effects models in a generalized linear mixed modeling (GLMM) framework. Mixed-effects modeling simultaneously estimates fixed and random-effects predicting a given outcome. The general form of the GLMM can be expressed in matrix notation as:

$$\mathbf{y} = \mathbf{X}\boldsymbol{\beta} + \mathbf{Z}\mathbf{u} + \boldsymbol{\epsilon}$$

Where $\mathbf{y}$ is a $n \times 1$ vector of outcomes; $\mathbf{X}$ is the $n \times p$ fixed-effects design matrix; $\boldsymbol{\beta}$ are the fixed effects; $\mathbf{Z}$ is the $n \times q$ random-effects design matrix; $\mathbf{u}$ are the random-effects; and $\boldsymbol{\epsilon}$ is a $n \times 1$ vector of residuals. For continuous outcomes, the model is specified using the identity link function

$$\eta = g(E(Y)) = E(Y).$$

Dichotomous outcomes utilize the logit link function

$$\eta = \text{logit}(\pi) = \log\left(\frac{\pi}{1-\pi}\right).$$

Hausman tests supported the use of random-effects over country fixed-effects to model between country variation for all outcomes. To examine cross-national heterogeneity in exposure to health insults and socioeconomic conditions during childhood we estimate random-intercepts models for poor childhood health and the childhood socioeconomic index and test for significant variation in the random-intercepts. To test whether the inclusion of socioeconomic conditions and health in childhood helps explain cross-national differences in health, we estimate a series of nested random-intercepts models of multi-morbidity, mobility, and ADL limitations. Base models control for age and sex. Model 2 adds childhood circumstances. Model 3 adds adult socioeconomic characteristics. Model 4 adds smoking and BMI. The nesting structure allows us to examine critical/sensitive period, accumulation, and chains of risk processes by testing the extent to which the association of poor health and socioeconomic characteristics in childhood with later health are mediated by subsequent socioeconomic exposures and health behaviors. While mediation analysis is conventionally done by the comparison of the estimates across nested models, we additionally conducted formal mediation tests using the Kohler, Holm, and Breen method, which is applicable to all link functions in the GLMM (Kohler, Karlson, & Holm, 2011). The formal mediation

analysis confirmed the results described below. Finally, to test whether international context modulates the relationship between childhood health and social conditions and later life health, we test random-slopes for poor childhood health and childhood socioeconomic index.

Results

Table 1 presents descriptive statistics by country. In England, the mean number of functional mobility limitations (1.73) is more than three times larger than in Switzerland (0.53). ADL differentials are even more pronounced with the older English experiencing 8.25 times more limitations than their Swiss peers. The English also have the highest rates of multi-morbidity with 21.9% experiencing two or more chronic conditions. This is more than twice the rate among the Swiss (8.6%). There are also differences in the prevalence of poor childhood health. Only 6.3% of the Irish sample reported having experienced fair or poor childhood health, while the prevalence in Austria was more than twice as large (12.9%). Large differences are observed for the index of childhood socioeconomic conditions. Austria experienced the highest mean childhood socioeconomic position (2.49), while Ireland was the most disadvantaged (0.63).

Table 2 presents formal analysis of cross-national differences in exposure to poor childhood health and socioeconomic conditions. For both poor childhood health and socioeconomic conditions there is significant variance in the random-intercept. To illustrate this variation we generate country-specific predicted prevalence rates for poor childhood health and the mean of the childhood socioeconomic index. The top pane of figure 1 presents predicted prevalence of Fair/poor childhood health. Prevalence of poor childhood health ranged from a low of 6.1% in Ireland to nearly double that (11.9%) in Austria. Low prevalence was also observed in Italy (6.4%), and the Czech Republic (6.7%). In addition to Austria, high prevalence of poor childhood health was also found in England (11.6%), Germany (11.2%), and the Netherlands (10.5%). The bottom pane presents predicted means of the childhood socioeconomic index. On average the most advantageous childhood socioeconomic conditions were found in Germany (2.25), the Czech Republic (1.96), and Switzerland (1.94), and, while the most disadvantageous conditions were found in Ireland (0.33), England (0.59), Spain (0.99), and Italy (1.03). Importantly, countries that did well in terms of low prevalence of poor childhood health tended to not be the same countries that performed well vis-à-vis childhood socioeconomic conditions. The two sets of early exposures appear to not be strongly correlated at the national level among these cohorts.

Table 3 presents exponentiated GLMM logit estimates (odds-ratios) predicting the probability of multi-morbidity. The variance of the random-intercept is significant and robust to model specification. Adding childhood health and social conditions to the model results in no change in the variance of the random-intercept, and only a slight decline in the intraclass correlation. The experience of poor childhood health is associated with a 1.57 times greater odds of having two-or more chronic conditions. Each additional increase in the index of childhood socioeconomic status is associated with 7% lower odds of multi-morbidity ($OR = 0.93$). The addition of adult socioeconomic status also has little effect on the random-intercept variance and intraclass correlation. However, the addition of adult SES results in a slight

decline in the estimate for poor childhood health and reduces the estimate for childhood SES by half.

Controlling for the distribution of smoking and body mass (model 4) further reduces the between-country variation in disease risk (random-intercept and intraclass correlation). Both current and former smokers are at increased risk of multi-morbidity as are those with higher body mass. Controlling for adult health behavior does result in a slight yet attenuation of the estimated association of childhood SES. With the inclusion of all covariates, childhood health remains significantly associated with multi-morbidity, while childhood SES does not. There also remains significant variance in the random-intercept such that population-level heterogeneity in the risk of chronic disease exists, net of the compositional differences in early life conditions, adult SES, and health behaviors.

Table 4 presents GLMM estimates for the log number of mobility functional limitations. Non-linear (e.g. negative binomial) specifications yielded substantively similar results. For ease of interpretation we present the linear results. Model 1 shows that the random-intercept is also significant, confirming between-country variation in the underlying level of functional health. As shown in model 2, those who experienced poor health in childhood reported 35% ($e^{0.30}=1.35$) more mobility limitations than those who had healthy childhoods. A one-unit increase in the childhood SES index was associated with a 3% ($e^{-0.03}=0.97$) decrease in the number of limitations. However, the inclusion of childhood conditions results in modest attenuation of country differences in health. The variance in the random-intercept is reduced slightly (from .15 to .14) and remains significant. The intraclass correlation is reduced by 10%.

Adult socioeconomic status is also negatively associated with functional health in later life. After the inclusion of adult socioeconomic status, the association with childhood SES is no longer statistically significant. The intraclass correlation is also slightly reduced but remains significant. Including all covariates (model 4) largely confirms the result of prior models. The estimate for childhood health is largely unchanged while that for childhood SES is completely eliminated. In addition, there remains a significant random-intercept.

Table 5 presents GLMM estimates of log ADL limitations. The significant random intercept reveals between-country differences in the underlying level of ADL limitations. Adding childhood health and socioeconomic circumstances to the model (model 2) results in no attenuation of the random-intercept and only a slight decline in the intraclass correlation. Those who experienced poor childhood health reported 8% ($e^{0.08}=1.08$) more ADL limitations than their healthy childhood peers. Similarly, those who experienced higher socioeconomic status during childhood reported fewer ADL limitations.

Adjusting for adult socioeconomic attainment (model 3) reduces the association between childhood socioeconomic status and ADLs to nearly zero, while the association with poor childhood health remains unchanged. The random-intercept variance and intraclass correlation do not change with the inclusion of individual adult SES. Even with all covariates in the model (model 4), childhood health continues to be independently associated with ADL limitations. However, adjusting for adult health lifestyle factors and

SES eliminates the association with childhood socioeconomic conditions. Interestingly, there is no difference in the random-intercept variance between model 1 and model 4.

Finally, we formally tested whether international context modulates the estimates for early life health and socioeconomic conditions by adding random-slopes to model 4. These results are presented in the final column of tables 3–5 (model 5). For multi-morbidity, only childhood SES showed significant variance across countries. For mobility and ADL limitations the variance estimates for the random-slopes for both poor childhood health and childhood SES were significant. To illustrate this variation, country-specific predictions for the random-slopes of poor childhood health and childhood SES are presented in table 6. With the exception of multi-morbidity, variability in estimates occurs for childhood health. For example, in Switzerland those with poor childhood health experienced 27% ($e^{0.241}=1.27$) more mobility limitations than their healthy childhood peers. However, in Ireland that difference is 33% ($e^{0.285}=1.33$). Similarly, poor childhood health is associated with an 8% ($e^{0.079}=1.08$) more ADL limitations in Ireland, while in Spain it is associated with 5% ($e^{0.049}=1.05$) more ADL limitations. Although variation in the estimates for childhood SES is also significant for multi-morbidity and mobility limitations, they varied within a substantively narrower range. In additional analysis (not shown) we find that in models without controls for birth cohorts that variation in estimates for poor childhood health and childhood SES was notably larger, suggesting important cohort variation in exposure to and long-run impact of early life forces across contexts.

Discussion

The present study is one of the first to integrate the comparative international perspective into the developmental origins of health and disease model by testing for international differences in exposure to childhood health insults and socioeconomic conditions, their association with later life health, and if early life factors help explain international variation in adult population health. We find that the among currently aging cohorts, exposure to childhood health insults and socioeconomic conditions significantly varied across Western Europe. The results also reveal that poor health in childhood has lasting negative associations with later life health, independent of adult socioeconomic conditions and behavioral factors. Childhood socioeconomic conditions on the other hand, are largely mediated by adult circumstances. Furthermore, depending on the specific health outcome, we find that the magnitude of the association between later life health and early life health and socioeconomic conditions can vary substantially across contexts. Despite this heterogeneity, the contribution of early life factors to explaining international differences in later life health across the countries, cohorts, and outcomes examined here was generally modest.

The present study makes a number of empirical contributions. It presents new evidence documenting international differences in both the prevalence of early life exposures and their association with subsequent adult health. In addition, whereas prior work by Banks et al. (2011) was limited to a comparison between the US and England, the current study investigates early life factors across a much broader range of contexts across Western Europe. Furthermore, we extend the work of McEniry (2014) by looking at a wider variety

of, and more direct measures of childhood health and socioeconomic circumstances rather than indirect, or proxies such as aggregate nutritional intake or rural birth.

More importantly, the results have important implications for the DOHD literature. In keeping with prior research, poor childhood health is a risk factor in every country we examined regardless of adult circumstances. The ubiquity of this linkage suggests empirical grounds to be considered a critical/sensitive period effect. There are also specific plausible biological mechanisms that underpin this relationship (Barker, 2007; Cohen et al. 2004; McDade, 2005; Crimmins & Finch 2006). The results also confirm that not all life course processes follow the same pathways. Some early life exposures, such a poor childhood health, appear to operate primarily through latent critical/sensitive period processes, while others (childhood socioeconomic position) through more generalized processes of risk accumulation (Blane et al. 2007). While there was very little attenuation of the association between childhood health and later life health through adult socioeconomic attainment and health behaviors, the association between later life health and childhood socioeconomic conditions was largely accounted for by adult factors. This is consistent with the accumulation and chains of risk approaches.

For policy makers, the results confirm that timely investments that improve the health of children and adolescents can have long-term payoffs that reduce later life disease and disability. Such investments are more productive and cost-efficient than investments later in life (Heckman & Masterove, 2007). At the same time, it is important to note that the constellation of childhood exposures to which these aging cohorts were exposed (e.g. infections and injuries) are quite different than those experienced by current cohorts (e.g. asthma, psychosocial behavioral disorders, obesity) (Van Cleave, Gortmaker, & Perrin, 2010). Many of the conditions that afflict more recent cohorts may be more amenable to subsequent investments. This could result in a decline in the association between early and later life health among more recent cohorts. Conversely, there is also reason to suspect a strengthening of the association between childhood and later life disease. For example, there has been a significant rise in childhood obesity in affluent societies (Wang and Lobstein 2006). This is a condition that tends to persist across the life course and has well-documented links to a host of pathological sequela including cardiovascular disease and diabetes.

There are some notable caveats and limitations to the findings. One caveat concerns the scope of contexts examined. As stated above, the results suggest that, at least for the contexts and outcomes examined here, early life factors may play a modest role in explaining between country variation in health. However, this may reflect the particular cluster of European countries examined. While important differences exist between these contexts, they tend to share similar levels of affluence, demographic histories, medical infrastructure, and substantial economic integration. In addition, while the timing and specific institutional arrangements varied, all of these countries implemented a broadly similar portfolio of social welfare policies over the course of the 20th century. This includes the mass expansion of education, labor market regulation (child labor laws, unionization, unemployment insurance/income protection, pensions), and universal health care systems. It may be the case that the relative importance of early life conditions in generating variation in later life health across

international contexts may be muted by the force of this shared expansion of the social capacity for health (Hayward & Sheehan 2016). It is also important to note that the countries examined here represent but a small slice of international variation in social, technological, and institutional conditions, adult health, and in life course etiologic exposures. As data linking early and later life health become available for more countries it will be crucial to extend the analysis to a wider array of sociohistorical, geographic, and cultural contexts of aging around the globe.

While documenting international variation in the relationships between life course factors is an important and necessary first step, unfortunately, we were not able to directly examine the structural and institutional mechanisms responsible. The second-order question of explaining the macro and micro social process that underlie the patterns documented here is an important next step. This work will, by its nature, emerge through further integration of the life course perspective into comparative studies of macro-structural and institutional processes. A good starting point will be to document the specific childhood health conditions most responsible for international variation in early life health insults, particularly those with the strongest connection with later life health and its proximate and contextual determinants. It will also be critical to examine the array of structural and institutional arrangements that moderate the long-term impact of early life health insults. This will include structures and policies directly tied to the treatment and management of ill-health in childhood and across the life course, but also those associated with human capital accumulation, labor market integration, family formation, and other downstream processes that may modulate the impact of early life insults. It will also require further delineation of period and cohort effects as these manifest in life course trajectories. Furthermore, it will necessitate multiple analytic strategies including the type used here (e.g. simultaneously examining a broad array of countries), as well as studies that are more narrowly focused on very detailed examination of particular population contexts (Zhang, Gu, & Hayward, 2008; Lumey & Van Poppel, 1994).

Another limitation of the current study is that in all countries the samples were subject to mortality selection over the life course. We are only able to observe members of aging cohorts that survived to make it into the various study samples. As there are substantial between country differences in mortality across the life course there are likely also differences in the impact of early life health and socioeconomic insults on mortality in childhood, adolescence and throughout adulthood. Thus, those societies where early life insults were most prevalent or have their most pernicious effects are likely to have experienced the strongest degree of mortality selection. That would lead to a regression towards the mean resulting in the present results underestimating true between country differences.

Finally, measurement is always a potential vulnerability in comparative studies. While much effort has been made to make the present studies comparable, there may be unknown contextual differences in key measures. While prior research has demonstrated the quality of retrospective childhood health histories, we are not aware of any work that has validated such histories across cultural contexts. To the extent that there are systematic cultural differences in the interpretation of and propensity to report particular response categories,

net of underlying health status, then our results may be biased. As research seeks to leverage the use of retrospective childhood health histories to answer important questions about the life course, it will be critical to empirically validate their cross-cultural comparability. The persistence in the associations across countries suggests that measurement issues may not be important.

The life course approach and the international comparative perspective have individually yielded important insights into the distribution of health within and between populations. The present study suggests that additional purchase can be gained at their nexus. The results dovetail with recent work showing that international variation in adult health trajectories derive, in part, from varying cohort dynamics, themselves the product of heterogeneous life course processes (Haas, Oi, & Zhou, 2017). Future research would be wise to leverage the unique insights available to a conceptual and empirical approach that combines a rich appreciation of life course/developmental processes and consideration for how national context, including institutional forces and cohort dynamics may modulate them.

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Highlights

We study international variation in life course influences on health.

Early life health & socioeconomic exposures vary across countries.

The long-term impacts of early life exposures also vary across countries.

Life course processes explain a modest portion of variation in adult health in Europe.

Figure 1.
Predicted Childhood Circumstances By Country

Table 1.

Weighted descriptive statistics by country: means (with standard deviations in parentheses) or percentages.

	England	Ireland	Austria	Germany	Sweden	Denmark	Netherlands
Functional Limitations	1.73 (2.24)	1.15 (1.81)	1.02 (1.57)	0.95 (1.77)	0.68 (1.51)	0.66 (1.35)	0.75 (1.65)
ADL Limitations	0.33 (0.85)	0.14 (0.59)	0.08 (0.41)	0.11 (0.56)	0.05 (0.42)	0.08 (0.44)	0.08 (0.52)
Two or More Chronic Conditions (%)	21.9	14.2	12.8	18.4	13.6	13.3	12.7
Age	65.6 (10.2)	63.6 (10.0)	63.9 (10.1)	63.3 (11.8)	63.4 (12.5)	63.0 (9.9)	62.6 (11.2)
Female (%)	53.7	52.0	56.1	52.0	53.2	52.8	52.1
Born before 1930	22.5	8.8	18.2	14.6	15.7	14.6	13.6
Born 1931–1935	13.4	9.4	11.7	10.1	10.7	10.2	10.0
Born 1936–1940	15.5	10.5	14.8	17.6	13.8	12.0	13.2
Born 1941–1945	18.9	12.9	17.8	16.9	17.8	19.2	15.9
Born 1946–1950	24.3	18.0	22.3	16.6	20.6	21.5	24.6
Born After 1951	5.6	40.4	15.1	24.2	21.3	22.6	22.8
Poor Childhood Health (%)	11.9	6.3	12.9	12.0	8.4	7.7	11.1
Childhood SES	1.08 (1.60)	0.63 (1.27)	1.94 (1.37)	2.49 (1.86)	1.53 (1.46)	2.04 (1.41)	1.47 (1.32)
Education (%)							
Low (ISCED 0–2)	55.3	63.4	32.0	15.7	47.5	21.6	50.2
Medium (ISCED 3)	9.2	18.1	44.3	52.8	20.5	42.5	25.7
High (ISCED 4)	35.4	18.5	23.7	31.5	32.0	35.9	24.1
Occupation (%)							
Low	36.7	48.0	62.1	45.5	35.6	44.3	40.7
Medium	53.9	35.0	29.4	43.1	39.3	38.3	39.0
High	9.5	17.0	8.5	11.4	25.1	17.4	20.3
Household Income (mean \$ by quartile)							
Low (bottom 25%)	10460 (5328)	10345 (9763)	9193 (7706)	9216 (7818)	15676 (9192)	15531 (8318)	10540 (8591)
Medium (middle 50%)	19843 (5872)	27972 (17058)	20639 (7879)	22708 (10205)	29959 (9209)	32452 (10349)	24972 (10111)
High (top 25%)	47114 (84646)	113851 (254841)	65582 (83715)	72978 (108218)	76772 (88984)	77591 (95424)	72074 (115736)
Smoking (%)							
Current Smoker	15.5	19.3	17.1	18.7	17.5	28.0	24.3
Former Smoker	47.8	37.8	18.5	26.5	36.6	33.6	37.4
Body Mass Index	27.4 (6.9)	28.7 (6.3)	27.1 (4.9)	26.7 (4.9)	26.0 (4.7)	25.8 (4.2)	26.3 (4.9)
<i>N</i>	5,973	8,160	873	1,856	2,282	2,111	2,282

	Belgium	France	Switzerland	Spain	Italy	Czech
Functional Limitations	1.00 (1.72)	0.95 (2.20)	0.53 (1.10)	1.25 (2.10)	1.19 (2.16)	1.01 (2.18)
ADL Limitations	0.13 (0.60)	0.09 (0.70)	0.04 (0.23)	0.11 (0.63)	0.11 (0.61)	0.08 (0.65)
Two or More Chronic Conditions (%)	13.8	13.1	8.6	13.7	16.8	20.2
Age	64.7 (11.4)	64.1 (14.0)	64.2 (10.8)	64.5 (12.1)	64.3 (12.7)	63.8 (14.5)
Female (%)	55.4	54.9	55.2	52.2	53.5	56.0
Born before 1930	20.4	18.6	18.3	18.2	16.8	11.0
Born 1931–1935	12.6	12.0	10.7	14.2	13.4	12.0
Born 1936–1940	13.4	12.1	14.2	14.4	15.9	12.0
Born 1941–1945	14.7	14.4	17.2	14.9	15.1	19.3
Born 1946–1950	19.0	21.7	18.7	17.8	21.7	22.3
Born After 1951	19.9	21.2	20.9	20.5	17.0	23.4
Poor Childhood Health (%)	8.6	9.6	9.9	10.0	6.8	6.7
Childhood SES	1.60	1.53	2.20	1.09	1.11	2.37
Education (%)	(1.30)	(1.54)	(1.44)	(0.93)	(0.82)	(2.71)
Low (ISCED 0–2)	46.7	42.6	36.4	79.2	72.6	44.1
Medium (ISCED 3)	25.6	35.4	34.1	10.4	18.8	39.0
High (ISCED 4)	27.7	22.0	29.5	10.5	8.6	16.9
Occupation (%)						
Low	51.8	47.5	31.7	70.9	28.3	48.2
Medium	31.9	39.1	50.7	21.9	63.4	42.1
High	16.3	13.4	17.6	7.2	8.3	9.8
Household Income (mean by quartile)						
Low (bottom 25%)	8119 (4012)	9155 (6008)	15457 (12078)	4149 (5292)	5501 (6333)	4201 (3223)
Medium (middle 50%)	18938 (5170)	22499 (8066)	37725 (15949)	10929 (5994)	13985 (6182)	6175 (1311)
High (top 25%)	69269 (131846)	65098 (103825)	113301 (170164)	46635 (145194)	41442 (81968)	12402 (12651)
Smoking (%)						
Current Smoker	15.8	14.7	19.3	18.2	19.9	22.4
Former Smoker	30.7	28.3	23.8	18.9	25.9	23.3
Body Mass Index	26.3 (4.5)	26.0 (5.9)	25.3 (4.3)	27.4 (5.1)	26.4 (4.9)	27.4 (6.6)
<i>N</i>	2,767	2,490	1,218	2,081	2,554	1,812

Table 2.

Generalized linear mixed model estimates (95% confidence intervals) of poor childhood health and childhood socioeconomic status (ELSA, TILDA, SHARE)

	Poor Childhood Health ¹		Log Childhood SES	
<i>Fixed Effects</i>				
Age	1.00	(0.99, 1.01)	0.01	(−0.02, 0.02)
Female	1.22	(1.14, 1.32)	0.01	(−0.00, 0.02)
Born in 1931/1935	1.00	(0.94, 1.00)	0.02	(−0.01, 0.05)
Born in 1936/1940	1.15	(0.83, 1.19)	0.06	(0.02, 0.10)
Born in 1941/1945	1.07	(0.92, 1.44)	0.10	(0.05, 0.15)
Born in 1946/1950	0.94	(0.81, 1.42)	0.11	(0.05, 0.18)
Born after 1950	0.91	(0.67, 1.34)	0.13	(0.06, 0.22)
Childhood SES	0.97	(0.60, 1.40)		
Poor childhood health			−0.02	(−0.05, 0.03)
Intercept	0.09		0.48	
<i>Random Effects</i>				
σ^2 (Intercept)	0.06	(0.03, 0.14)	0.11	(0.05, 0.23)
Intraclass correlation	.021		.209	
<i>N</i>		36,459		36,459

¹. Odds-ratios

². Born before 1930 as the reference

Table 3.

Generalized linear mixed logit model estimates (odds ratios with 95% confidence intervals) of multi-morbidity (ELSA, TILDA, SHARE)

	1	2	3	4	5
<i>Fixed Effects</i>					
Age	1.03 (1.02, 1.04)	1.03 (1.02, 1.05)	1.03 (1.02, 1.04)	1.04 (1.03, 1.05)	1.04 (1.03, 1.05)
Female	0.84 (0.80, 0.89)	0.83 (0.79, 0.89)	0.82 (0.78, 0.87)	0.90 (0.85, 0.96)	0.90 (0.85, 0.96)
Born in 1931/1935	1.01 (0.89, 1.14)	1.01 (0.89, 1.14)	1.00 (0.89, 1.14)	0.99 (0.88, 1.12)	0.99 (0.88, 1.12)
Born in 1936/1940	0.93 (0.80, 1.10)	0.94 (0.80, 1.10)	0.95 (0.81, 1.12)	0.98 (0.83, 1.15)	0.98 (0.83, 1.15)
Born in 1941/1945	0.80 (0.65, 0.98)	0.80 (0.66, 0.99)	0.83 (0.68, 1.02)	0.86 (0.70, 1.05)	0.86 (0.70, 1.06)
Born in 1946/1950	0.60 (0.47, 0.78)	0.61 (0.48, 0.79)	0.63 (0.50, 0.82)	0.69 (0.53, 0.90)	0.70 (0.54, 0.90)
Born after 1950	0.44 (0.32, 0.60)	0.45 (0.32, 0.61)	0.46 (0.34, 0.63)	0.54 (0.40, 0.74)	0.54 (0.40, 0.74)
Poor childhood health		1.57 (1.42, 1.71)	1.54 (1.40, 1.69)	1.57 (1.43, 1.72)	1.57 (1.43, 1.72)
Childhood SES		0.93 (0.91, 0.96)	0.98 (0.94, 1.00)	0.98 (0.95, 1.01)	0.98 (0.95, 1.01)
Education (low=reference)					
Medium			0.85 (0.78, 0.93)	0.88 (0.80, 0.96)	0.88 (0.80, 0.96)
High			0.83 (0.75, 0.90)	0.88 (0.80, 0.96)	0.88 (0.80, 0.96)
Occupation (low=reference)					
Medium			0.92 (0.86, 0.98)	0.95 (0.88, 1.01)	0.94 (0.88, 1.01)
High			0.83 (0.74, 0.93)	0.85 (0.76, 0.96)	0.85 (0.76, 0.96)
Income (low = reference)					
Medium			0.94 (0.88, 1.00)	0.94 (0.88, 1.01)	0.95 (0.88, 1.01)
High			0.83 (0.76, 0.92)	0.84 (0.76, 0.96)	0.84 (0.76, 0.93)
Smoking (never=reference)					
Current smoker				1.22 (1.12, 1.34)	1.22 (1.12, 1.34)
Former smoker				1.39 (1.30, 1.48)	1.39 (1.30, 1.48)
BMI				1.07 (1.07, 1.08)	1.07 (1.07, 1.08)
Intercept	0.03	0.01	0.04	0.00	0.00
<i>Random effects</i>					
σ^2 (Intercept)	0.27 (0.18, 0.41)	0.26 (0.16, 0.38)	0.25 (0.16, 0.38)	0.23 (0.15, 0.36)	0.21 (0.14, 0.33)
σ^2 (Poor childhood health)					0.00 (0.00, 0.00)

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	1	2	3	4	5
σ^2 (Childhood SES)					(0.01, 0.08)
Intraclass correlation	.021	.020	.019	.014	.013
-2 log likelihood	-15410	-15398	-15382	-15001	-15001
N	36,459	36,459	36,459	36,459	36,459

Table 4. Generalized linear mixed model estimates (95% confidence intervals) for log of functional mobility limitations (ELSA, TILDA, SHARE)

	1	2	3	4	5
<i>Fixed Effects</i>					
Age	0.02 (0.02, 0.03)	0.02 (0.02, 0.03)	0.02 (0.02, 0.03)	0.02 (0.02, 0.03)	0.02 (0.02, 0.03)
Female	0.33 (0.32, 0.35)	0.33 (0.31, 0.34)	0.31 (0.30, 0.33)	0.35 (0.33, 0.37)	0.35 (0.33, 0.37)
Born in 1931/1935	-0.12 (-0.16, -0.07)	-0.12 (-0.16, -0.07)	-0.117 (-0.16, -0.08)	-0.131 (-0.17, -0.09)	-0.131 (-0.17, -0.09)
Born in 1936/1940	-0.14 (-0.20, -0.10)	-0.14 (-0.20, -0.10)	-0.13 (-0.18, -0.08)	-0.14 (-0.19, -0.08)	-0.14 (-0.19, -0.08)
Born in 1941/1945	-0.11 (-0.17, -0.41)	-0.10 (-0.17, -0.41)	-0.08 (-0.15, -0.02)	-0.087 (-0.15, -0.02)	-0.087 (-0.15, -0.02)
Born in 1946/1950	-0.08 (-0.16, 0.04)	-0.07 (-0.16, 0.04)	-0.042 (-0.12, 0.04)	-0.036 (-0.12, 0.04)	-0.036 (-0.12, 0.04)
Born after 1950	-0.07 (-0.17, 0.03)	-0.06 (-0.17, 0.03)	-0.03 (-0.13, 0.07)	-0.054 (-0.10, 0.09)	-0.01 (-0.10, 0.09)
Poor childhood health		0.30 (0.26, 0.32)	0.29 (0.26, 0.32)	0.29 (0.26, 0.32)	0.27 (0.22, 0.31)
Childhood SES		-0.03 (-0.04, -0.03)	-0.00 (-0.01, 0.00)	-0.00 (-0.01, 0.00)	0.00 (-0.01, 0.02)
Education (low=reference)					
Medium			-0.10 (-0.13, -0.08)	-0.09 (-0.12, -0.07)	-0.09 (-0.12, -0.07)
High			-0.13 (-0.16, -0.11)	-0.10 (-0.13, -0.08)	-0.10 (-0.13, -0.08)
Occupation (low=reference)					
Medium			-0.08 (-0.10, -0.06)	-0.07 (-0.09, -0.05)	-0.07 (-0.09, -0.05)
High			-0.11 (-0.14, -0.08)	-0.10 (-0.13, -0.07)	-0.10 (-0.13, -0.07)
Income (low = reference)					
Medium			-0.05 (-0.07, -0.03)	-0.05 (-0.07, -0.03)	-0.05 (-0.07, -0.03)
High			-0.13 (-0.16, -0.10)	-0.12 (-0.15, -0.09)	-0.12 (-0.15, -0.09)
Smoking (never=reference)					
Current smoker				0.164 (0.14, 0.19)	0.16 (0.14, 0.19)
Former smoker				0.077 (0.06, 0.10)	0.08 (0.06, 0.09)
BMI				0.026 (0.02, 0.03)	0.03 (0.02, 0.03)
Intercept	-1.63	-1.6	-1.4	-2.38	-1.89
<i>Random effects</i>					
σ^2 (Intercept)	0.15 (0.10, 0.22)	0.14 (0.10, 0.21)	0.14 (0.09, 0.20)	0.12 (0.08, 0.18)	0.14 (0.09, 0.20)
σ^2 (Poor childhood health)					0.06 (0.03, 0.13)

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	1	2	3	4	5
σ^2 (Childhood SES)					(0.01, 0.04)
Intraclass correlation	.032	.029	.028	.023	.028
-2 log likelihood	-44812	-44564	-44311	-43842	-43841
<i>N</i>	36,459	36,459	36,459	36,459	36,459

Table 5.

Generalized linear mixed model estimates (95% confidence interval) for logged number of ADL limitations (ELSA, TILDA, SHARE)

	1	2	3	4	5
<i>Fixed Effects</i>					
Age	0.01 (0.01, 0.01)	0.01 (0.01, 0.01)	0.01 (0.01, 0.01)	0.01 (0.01, 0.01)	0.01 (0.01, 0.01)
Female	0.03 (0.02, 0.04)	0.03 (0.02, 0.04)	0.03 (0.02, 0.03)	0.03 (0.02, 0.04)	0.03 (0.02, 0.04)
Born in 1931/1935	-0.05 (-0.07, -0.03)	-0.05 (-0.07, -0.03)	-0.05 (-0.07, -0.03)	-0.05 (-0.07, -0.03)	-0.05 (-0.07, -0.03)
Born in 1936/1940	-0.02 (-0.05, 0.01)	-0.02 (-0.05, 0.01)	-0.02 (-0.05, 0.01)	-0.02 (-0.05, 0.01)	-0.02 (-0.05, 0.01)
Born in 1941/1945	-0.02 (-0.01, 0.05)	0.02 (-0.01, 0.05)	0.03 (-0.01, 0.05)	0.03 (-0.01, 0.06)	-0.02 (-0.01, 0.05)
Born in 1946/1950	0.05 (0.02, 0.10)	0.05 (0.02, 0.10)	0.06 (0.02, 0.10)	0.06 (0.02, 0.10)	0.06 (0.02, 0.10)
Born after 1950	0.10 (0.05, 0.14)	0.10 (0.05, 0.14)	0.10 (0.05, 0.15)	0.10 (0.05, 0.15)	0.10 (0.06, 0.15)
Poor childhood health		0.08 (0.07, 0.09)	0.08 (0.06, 0.09)	0.08 (0.04, 0.09)	0.06 (0.03, 0.09)
Childhood SES		-0.01 (-0.01, -0.03)	-0.00 (-0.01, 0.00)	0.00 (-0.01, 0.00)	0.00 (-0.00, 0.00)
Education (low=reference)					
Medium			-0.03 (-0.04, -0.02)	-0.02 (-0.04, -0.01)	-0.03 (-0.04, -0.01)
High			-0.03 (-0.05, -0.02)	-0.03 (-0.04, -0.01)	-0.03 (-0.04, -0.01)
Occupation (low=reference)					
Medium			-0.02 (-0.03, -0.01)	-0.02 (-0.03, -0.01)	-0.02 (-0.03, -0.01)
High			-0.02 (-0.04, -0.01)	-0.02 (-0.04, -0.01)	-0.02 (-0.04, -0.01)
Income (low = reference)					
Medium			-0.01 (-0.04, 0.01)	-0.01 (-0.02, 0.01)	-0.01 (-0.02, 0.01)
High			-0.02 (-0.04, -0.01)	-0.02 (-0.03, -0.01)	-0.02 (-0.03, -0.01)
Smoking (never=reference)					
Current smoker				0.05 (0.04, 0.06)	0.05 (0.04, 0.06)
Former smoker				0.02 (0.01, 0.03)	0.02 (0.01, 0.03)
BMI				0.00 (0.00, 0.01)	0.00 (0.00, 0.01)
Intercept	-1.32	-1.32	-1.27	-1.48	-1.06
<i>Random effects</i>					
σ^2 (Intercept)	0.05 (0.03, 0.08)	0.05 (0.03, 0.07)	0.05 (0.03, 0.07)	0.05 (0.03, 0.07)	0.05 (0.04, 0.08)
σ^2 (Poor childhood health)				0.04 (0.02, 0.07)	0.04 (0.02, 0.07)

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	1	2	3	4	5
σ^2 (Childhood SES)					(0.01, 0.01)
Intraclass correlation	.017	.017	.018	.016	0.02
-2 log likelihood	-16872	-16801	-16712	-16615	-16591
N	36,459	36,459	36,459	36,459	36,459

Table 6.

Predicted random slopes for poor childhood health and childhood SES

	Multi-Morbidity ^I		Log Mobility Limitations		Log ADL Limitations	
	Poor Childhood Health	Childhood SES	Poor Childhood Health	Childhood SES	Poor Childhood Health	Childhood SES
Total	1.59	0.976	0.273	0.000	0.065	0.003
England	1.59	0.957	0.284	-0.003	0.074	-0.001
Austria	1.59	0.980	0.269	-0.006	0.060	0.003
Germany	1.59	0.985	0.272	-0.004	0.060	0.003
Sweden	1.59	0.973	0.272	0.000	0.059	0.004
Netherlands	1.59	0.973	0.270	0.005	0.061	0.004
Spain	1.59	0.975	0.270	0.000	0.049	0.004
Italy	1.59	0.977	0.267	-0.003	0.057	0.003
France	1.59	0.969	0.262	-0.001	0.056	0.004
Denmark	1.59	0.975	0.269	0.000	0.061	0.004
Switzerland	1.59	0.974	0.241	0.004	0.055	0.004
Belgium	1.59	0.978	0.267	0.007	0.059	0.003
Czech	1.59	0.966	0.263	0.000	0.052	0.004
Ireland	1.59	0.993	0.285	-0.001	0.079	0.003

^I. Odds-ratios