Education and Self-Rated Health: Cumulative Advantage and Its Rising Importance
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The cumulative advantage hypothesis predicts that the adulthood rate of decline in health differs across levels of education in a manner that progressively enlarges the health gap across most or all of adulthood. The rising importance hypothesis predicts that the differences across levels of education in the rate of health’s decline have been growing for many decades. If both are correct, then each phenomenon tends to obscure the other when comparing the health gap across age groups in a particular year or period. The trend also can make it seem that health converges across levels of education in old age when it actually diverges. A latent-growth model of U.S. data from 1995, 1998, and 2001 supported both hypotheses. It also showed a trend toward lower age-specific self-rated health at all levels of education, but less so the higher the education. There was no significant convergence over time in older age groups.

Keywords: education; health; age-period cohort; cumulative advantage; latent-growth models

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The cumulative advantage hypothesis predicts that the age-specific rates of decline in health differ across levels of education in a manner that progressively enlarges the health gap across most or all of adulthood. The rising importance hypothesis predicts that health’s relationship to education has been growing for many decades. In other words, the rate that health diverges across levels of education in adulthood may itself be growing. Both hypotheses have currency. If both are correct, then one phenomenon tends to obscure the other when comparing age groups in a given year or follow-up period (Lauderdale 2001). In theory, older Americans have had longer for the health gap to develop, which would make the differences across levels of education increase with age at the time. However, younger adults (born more recently) may have higher rates of divergence, making the gap grow to a given size at an earlier age. That would shrink differences across age groups in a given study year or follow-up period. In addition, analyses that ignore the trends can misread them as old-age convergence in health across levels of education. To some extent, the oldest adults may have smaller health gaps and slower rates of divergence than the middle-aged because they always have had slower rates of divergence.

The remainder of this introduction reviews the theory and findings regarding cumulative advantage and adulthood growth in health differences across levels of education. It also reviews the theory and findings about a contingent but integral issue: the possible shrinking of those differences in old age, perhaps because of a longevity ceiling or mortality selection. It then reviews the theory and findings about the rising importance of education to health in newer cohorts, including attempts to distinguish education’s effect on health trajectories within cohorts from trends in its effect across cohorts.

Cumulative Advantage

The hypotheses of education’s cumulative health advantage and rising health importance come from two historically distinct areas of research that are merging. The cumulative advantage hypothesis comes from research on the development throughout adulthood of the health disparities seen in old age (House, Lantz, and Herd 2005; Lynch 2003; Ross and Wu 1996). The concept of cumulative advantage came from research on the growth of socioeconomic disparities in adulthood (Crystal and Shea 1990; O’Rand 1996). Dannefer (1987) argued that life-course studies should look at processes of self-reinforcing social differentiation more broadly. Ross and Wu (1996) applied the concept of cumulative advantage to health in their
study of education’s many links to health. According to theory, educational attainment selects for and develops generally effective abilities, habits, and attitudes and provides access to higher status positions (Mirowsky and Ross 2005b). Greater effectiveness makes individuals better at avoiding health risks and better at “self-management” when health problems occur (Goldman and Smith 2002; Mirowsky and Ross 2003; Pampel and Rogers 2004; Pincus 1996; Pincus et al. 1998; Ross and Wu 1995, 1996; Wray et al. 1998). Higher status positions insulate individuals from the stress of chronic economic hardship and alienating work (Karasek and Theorell 1990; Marmot and Mustard 1994; Mirowsky and Hu 1996; Ross and Wu 1995). Persistent differences in stress produce diverging cumulative degradation of organs or physiological processes, often called “allostatic load” (Marmot et al. 1998; McEwen 1998; Taylor, Repetti, and Seeman 1997).

The evidence for education’s cumulative health advantage is mixed but generally supports the hypothesis. Some say the gap in health across levels of education grows throughout adulthood (e.g., Mirowsky and Ross 2005a, 2005b; Ross and Wu 1996). Others say that the gap grows through much of adulthood but eventually converges as death eliminates the unhealthiest (disproportionately low education) or as the healthiest (disproportionately high education) encounter a biological ceiling on the postponement of morbidity (e.g., Herd 2006; House et al. 1994, 2005). However, one study found what appears to be a progressive convergence in number of chronic conditions and functional impairments from ages 32 through 86 (Beckett 2000). Another found a progressive convergence in self-rated health from ages 25 through 85 but divergence in functional limitations through age 55 followed by progressive convergence through age 85 (Cutler and Lleras-Muney 2006). The ambiguities have stimulated a number of ongoing studies (e.g., Hamil-Luker 2004; Quesnel-Vallee 2004; Willson and Shuey 2004).

Several analyses, or comments on them, have addressed the possibility that forces of selection in old age compress the differences in health across levels of education (Beckett 2000; Beckett and Elliott 2001; Herd 2006; House et al. 2005; Lynch 2003; Noymer 2001). The researchers generally agree that poor health increases the inability or unwillingness to participate in a survey, in part through death but also through disability and demoralization. They debate whether these selection forces can account for the apparent old-age convergence in health across levels of education. On the whole the analyses suggest that differential mortality alone cannot. However, an increase with age in the inability or unwillingness to participate because of poor health may directly compress the observed differences in health across levels of education (Noymer 2001).
Some of the ambiguity about cumulative advantage and late-life convergence may lie in the distinction between cross-sectional and follow-up results. It is possible for the average levels of health to converge even though the changes in health remain less negative for the better educated (Lynch 2003). For example, Mirowsky and Ross (2005a) compared the levels and slopes of physical impairment across categories of education. The gap in levels of impairment peaked among persons in their mid-60s. The follow-up slopes became increasingly parallel in successively older age groups but continued to be flatter for the better educated through the mid-80s. Death and the inability or unwillingness to participate in a survey because of poor health can reduce the education-based differences in health levels at a much earlier age than it eliminates the differences in follow-up health slopes, as Lynch (2003) illustrated. A trend toward larger educational differences in health trajectories can produce a similar pattern, as detailed in this study.

Some ambiguity may be resolved by distinguishing theoretically between two components of health’s adulthood trajectory (House et al. 2005; Mirowsky and Ross 2005a). The trajectory is the expected value of health as a curvilinear function of age. Theory suggests that it has two components. One is a more or less constant annual rate of decline in health that we call the erosion component. If the rate of erosion varies across levels of education, the result will be a progressive growth in the health differences as a cohort ages, consistent with the cumulative advantage hypothesis. The other component represents a biological ceiling on the postponement of morbidity. Its contribution to the trajectory is vanishingly small through much of adulthood but grows at an accelerating rate, becoming substantial in old age. We call this the disintegration component of the trajectory. It may be far less sensitive to cumulative socioeconomic advantages. Some have argued that it even may be steeper for the more advantaged, as postponed morbidity catches up (House et al. 2005). If both components exist, the slope of the trajectory at any given age sums the two. In earlier adulthood, the erosion component predominates, producing a growing health gap. Eventually, though, the disintegration component predominates, making the rate of decline in health across levels of education more and more similar at older ages. It may eventually produce a crossover, with faster declines among the better educated as their health advantage vanishes.

Rising Importance

The rising importance hypothesis states that education’s relationship to the rate of decline in health is greater in newer cohorts. In our terms above,
the effect of education on the rate that health erodes is growing. The rising importance hypothesis comes out of research on historical trends in mortality differentials. In the last third of the twentieth century, those studies began looking for declines in the socioeconomic differences expected as a result of improved public health and medical technology over the century. Instead, they found trends toward larger differences in age-specific mortality rates across levels of education (Elo and Preston 1996; Feldman et al., 1989; Lauderdale 2001; Pappas et al. 1993). Lauderdale (2001) found that the association of education with 10-year survival was increasing within cohorts as they aged, at a rate that was increasing in younger cohorts. She noted that the growth in educational differences in survival between cohorts can obscure the growth over adulthood. Older persons have had longer for educational differences to develop but lived in times when those differences grew more slowly. Lynch (2003) thought that similar trends in education’s relationship with self-rated health might obscure the growth of health differences in adulthood, perhaps even creating the false appearance of convergence in old age.

Some studies have attempted to distinguish education’s effect on the shape of health’s trajectory within cohorts from the trend across cohorts in education’s effect. Results are mixed. The studies are summarized below in progression from solely concerned with eliminating trend bias in trajectory estimates, to primarily concerned with trajectories but realizing that the results imply trends, to modeling the trends as an integral or primary focus.

Herd (2006) tried to eliminate the confounding of trajectory and trend by limiting the analysis to persons born from 1931 through 1941, aged 51 through 61 years at baseline in 1992 and aged 61 through 71 years by a fifth biennial interview in 2002. She reasoned that this effectively holds cohort constant but covers the range of ages in which divergence probably switches to convergence. Her model showed increasing educational disparities in functional limitations through age 63, followed by diminishing differences that remained substantial at age 71. However, Herd fit an age-basis latent-growth model that assumed that there was no significant trend across the 10 one-year cohorts in the sample and did not test the assumption. The model averaged across cohorts the predicted values at each age. This amounts to averaging the cross-sectional curves across waves of observation. For follow-up surveys with evenly spaced waves, the resulting curves represented the predicted levels at the midpoint of the study, which were shaped by the trends as well as the trajectory.

Using the same data but a broader range of cohorts (1904 through 1945) to study cognitive decline, Alwin and his colleagues found evidence of
statistically significant variation across cohorts in the parameters of a parabolic model (Alwin, Hofer, and McCammon 2006; Alwin, McCammon, and Hofer 2006; Alwin et al. 2005). They addressed the possibility that what looks like old-age cognitive decline is actually the result of lower levels of education in older cohorts. If so, the cross-sectional curves and follow-up changes would be flatter after adjusting for education. Their results showed just the opposite: steeper slopes after the adjustment. Alwin et al.’s (2005, 2006, 2006) time-basis models measured and tested for random variance in coefficients across cohorts (Miyazaki and Raudenbush 2000). That is not the same thing as a trend, which is by definition directional, not random. However, graphs comparing the predicted arc segments of all cohorts to one another showed a tendency for newer cohorts to have lower age-specific values. As a result, follow-up slopes within cohorts were steeper than the cross-sectional curve (the curve averaging age-specific values across cohorts). Adjusting for education increased the difference, implying that the trend toward steeper declines in cognitive function with age is greater within levels of education than on the whole. This suggests that rising levels of education partly suppress the trend, which in turn suggests that the downward trend may be slower at higher levels of education.

House et al. (2005) graphed mean levels of functional limitations by age for three categories of education (0 to 11, 12 to 15, and 16+). They compared the cross-sectional age curves at baseline (1986) to the changes in means as cohorts aged approximately 15.5 years (through 2001-2002). Both patterns of means suggested divergence in physical impairment across levels of education until about age 75, with convergence beyond. Interestingly, though, the graphs of follow-up means showed much larger 15.5-year divergence across levels of education among persons entering old age than suggested by the baseline cross-sectional means. The health gap among persons aged 75 years was notably larger at the end of the 15.5-year study period than at the beginning.

Both Alwin et al. (2005, 2006, 2006) and House et al. (2005) interpreted the differences between follow-up and cross-sectional slopes as signs of a trend. This is a crucial point and a key to measuring and modeling the trends across cohorts in the shape of the adulthood health trajectory (Mirowsky and Kim 2007). In the absence of trends, the follow-up changes would trace the baseline cross-sectional curves at each level of education, and the cross-sectional curves would be essentially the same in each study year. Differences between follow-up changes and cross-sectional curves reveal the trends. For House et al. (2005), the differences imply a substantial
trend toward higher rates of health divergence in the cohorts entering old age during the study than among those already old at the beginning of it.

Lynch (2003) took two distinct approaches to measuring the trajectories and trends, one using sequential cross-sectional data and the other using longitudinal follow-up data. The first of these is, to our knowledge, the only prior study to specify and test the three-way interaction implied by the rising importance hypothesis: that the rate at which health diverges across levels of education within cohorts is increasing across cohorts. Lynch’s sequential cross-sectional model specified a parabolic age trajectory within cohorts with its linear coefficient a function of cohort (birth year), education, and their product term.¹ That linear coefficient represented the speed at which health declines through much of adulthood. Fit to 21 years (1972 to 1993) of cross-sectional surveys measuring self-rated health, the results showed a significant three-way interaction supporting the rising importance hypothesis. The model implied that education slows the rate of decline in health by an amount that was larger in younger cohorts. This indicates a trend toward faster and longer divergence within younger cohorts as they age.

Lynch (2003) also fit a linear time-basis latent-growth model to 21-year follow-up data (1971 to 1992), with education, birth year, and their interaction as predictors of health slopes. Higher education generally flattened the predicted decline in health. The interaction between birth year and education implied that the effect switched signs for older birth years. Education flattened the expected health slopes for persons born after 1925 but steepened them for those born earlier. It is not clear whether birth year, in this model, should be interpreted as a measure of cohort per se, of the ages crossed during the period of observation, or a mixture of the two.

Hypotheses

Our analyses addressed two central hypotheses. The cumulative advantage hypothesis states that the decline in self-rated health with age is slower at higher levels of education, producing divergence through much or all of adulthood. The rising importance hypothesis states that the rate at which health trajectories diverge across levels of education within cohorts as they age is getting larger in younger cohorts. The analyses also addressed several integral phenomena that might influence estimates or interpretations: a ceiling on the postponement of morbidity, morbidity and mortality selection effects, and convergence in old age.
Methods

Model and Analyses

Methodologically, studies of education’s cumulative health advantage have progressed from comparing the cross-sectional means or regression slopes across levels of education to modeling the interaction of education and cohort in their effects on aging vectors of health. Aging-vector surveys measure each person’s level of health and change in health over a follow-up period of years. Latent-growth models can simultaneously predict the levels and slopes from age at the time, education, and their interactions. The analyses in this report used a new variant of the time-basis (also called occasion-basis) latent-growth model (Mirowsky and Kim 2007). It is similar to Lynch’s (2003) latent-growth model in two ways. First, it measures an individual’s health as a linear function of time from the midpoint of the study. Second, it predicts the individual’s health level at midpoint and health slope over the period from education, age at midpoint (an exact linear function of birth year), and their interaction. It differs in that it uses the relationship between cross-sectional and follow-up slopes to measure the trends, formalizing the kinds of comparisons and interpretations made by Alwin et al. (2005, 2006, 2006) and House et al. (2005). This makes it possible to distinguish education’s effect on the rate of divergence within cohorts from the trend across cohorts in that effect. It provides a test of the three-way interaction of age, education, and cohort in the context of a longitudinal latent growth analysis.

The analysis began with specifying a virtual cohort model (Mirowsky and Kim 2007). It defines the trajectory within a cohort as an expected value that is a curvilinear function of age, which may depend on education. It defines the trend as the effect of birth year on the shape of the trajectory, which also may depend on education. Equation 1 defines the theoretical health trajectory as a central tendency for individuals born the same year and aging across adulthood together. Individual levels and slopes may vary randomly from the trajectory over a study period, as detailed in the Appendix, but follow the trajectory in aggregate.

\[ \hat{H}_i = v_0 + v_1(A_i - 21) + v_3(A_i - 21)^3. \] (1)

The hypothetical trajectory function in equation 1 describes the predicted health of person i’s cohort at time t, where time refers to a specific calendar year measured as a deviation from the midpoint of the follow-up study. Predicted health is a function of the age of the person’s cohort at the
time of an observation, \(A_{it}\). Age is centered on 21 years to make the intercept \(v_0\) represent health near the beginning of adulthood. The trajectory has two functions of age. The linear function represents the steady decline in health that dominates the trajectory’s slope through middle age. We call its coefficient \(v_1\) the erosion coefficient. Because age is centered on 21 years, it represents the health slope at the beginning of adulthood. The cubic function represents acceleration in the rate of decline that dominates the trajectory’s slope in old age. We call its coefficient \(v_3\) the disintegration coefficient. The health slope is given by the derivative of the trajectory function with respect to age:

\[v_1 + 3v_3(A_{it} - 21)^2.\]

It is expected that \(v_3\) will be much smaller than \(v_1\), such that disintegration contributes little to the overall slope until late adulthood. Equation 2 is the hypothetical moderator function:

\[v_1 = v_{10} + v_{11}(A_{i0} - 21) + v_{12}(E_i - 12) + v_{13}(E_i - 12)(A_{i0} - 21).\]  

(2)

Equation 2 describes the rate of erosion in health as a linear function of cohort, education, and their interaction. Age at time zero \((A_{i0})\) represents cohort in Equation 2. (Measuring cohort by age at mid-follow-up simplifies the derivation of latent-growth level and slope equations from the virtual cohort model, as detailed in the Appendix.) In the moderator function, \(v_{10}\) represents the erosion coefficient of persons with 12 years of education in the cohort aged 21 years at time zero, \(v_{11}\) represents the average difference in that rate for successively older cohorts, \(v_{12}\) represents the average difference in the rate of erosion across levels of education in the cohort aged 21 years at time zero, and \(v_{13}\) represents the average change in education’s effect in successively older cohorts.

The cumulative advantage hypothesis states that \(v_{12} + v_{13}(A_{i0} - 21) > 0\): education slows the rate of erosion in health across the observed range of cohorts. The rising importance hypothesis states that \(v_{13} < 0\): education’s effect on the rate of erosion is smaller in older cohorts.

Figure 1 illustrates the three-wave, six-year latent-growth structural equation model implied by the level and slope equations derived from the virtual cohort model, as detailed in the Appendix. The structural equation model adds one more simplifying assumption, that the variance of the within-person random error \(e_{it}\) is constant across the three survey waves.

It is important to acknowledge that the virtual cohort model analyzed here is highly simplified. The model is meant to represent the essential elements of the hypothetical trajectory and trend. Clearly, it could be elaborated in several ways. To the extent that factors other than cohort and education alter the trajectory and trends, the models estimated here
represent an average given the sample composition on those factors. In this article we will elaborate the model only in very limited ways directly related to the questions of sample selection and old-age convergence. Although the model is highly simplified, it embodies a variety of ideas and observations. We will show that it fits the data well, illustrates the implications of trends in the trajectory, reproduces patterns observed in previous studies, and reveals new patterns that may guide further research.

Sample

This study used data from the survey of Aging, Status, and the Sense of Control (ASOC). It is a national telephone probability sample of 2,592 U.S. households. Sampling, pretesting, and interviewing for the surveys were conducted by the Survey Research Laboratory of the University of Illinois. Respondents were selected using a prescreened random-digit-dialing method (Lund and Wright 1994). The survey was limited to English-speaking adults. Up to 10 callbacks were made to get an interview and up to 10 to

Figure 1

Three-Wave Latent-Growth Structural Equation Model Representing the Virtual Cohort Model of Equations 1 and 2

Note: $A$ is age at time zero minus 21, and $E$ is years of education minus 12. $\hat{H}_i = v_0 + v_1(A_i - 21) + v_3(A_i - 21)^3$, and $v_1 = v_{10} + v_{11}(A_{i0} - 21) + v_{12}(E_i - 12) + v_{13}(A_{i0} - 21)(E_i - 12)$. The Appendix gives the derivation of the functions and restrictions.
complete it. Baseline interviews were completed with 71.6% of contacted and eligible persons.

The ASOC survey has an 80% oversample of people aged 60 years or older. At baseline (1995), 58% were aged 18 through 59 years (n = 1,496) and 42% were aged 60 through 95 years (n = 1,097). All results are for the entire unweighted sample unless otherwise noted. The oversample of seniors does not bias results because age is an independent variable in all of the models (Winship and Radbill 1994).

The demographic characteristics of the baseline ASOC sample match those for the U.S. adult householder population reasonably well (U.S. Census Bureau 1995). For comparative purposes, the statistics in this paragraph weight the ASOC sample to compensate for the oversample of seniors. For baseline ASOC and the United States, respectively, the percentage female is 56.2 and 51.2, the percentage White is 85.1 and 82.9, the percentage married (excluding cohabiters and those separated) is 55.7 and 55, and the mean household size is 2.67 and 2.59. For persons aged 25 years or older, the percentage with a high school degree is 85.1 and 80.9, and the percentage with a college degree is 25.6 and 22.2. The mean household income is $43,949 and $41,285.

The ASOC survey has three waves of interviews taken at three-year intervals, in 1995, 1998, and 2001, with 907 respondents who participated in all three interviews, 470 respondents who participated in the first and second interviews, 237 respondents who participated in the first and third interviews, and 978 respondents who participated only in the first. The structural equation models adjusted for attrition using partitioned full-information maximum likelihood estimation, which uses all cases regardless of their follow-up status (Wothke 2000). The procedure adjusted for data “missing at random,” meaning random given the observed values in the model. It did not adjust for nonparticipation or attrition due to residual poor health not predictable from the observed values. One of the analyses addressed the possible impact of outcome-dependent selection.

Measurement

Self-reported health was a respondent’s subjective assessment of his or her general health. Respondents were asked, “First, I’d like to ask you about your health. Would you say your health is very good, good, satisfactory, poor or very poor?” The responses were coded 5, 4, 3, 2, and 1, respectively. Self-reported health is a valid and reliable measure of general physical well-being (Davies and Ware 1981; Mossey and Shapiro 1982). It
combines the subjective experience of acute and chronic, fatal and nonfatal diseases, and general feelings of well-being, such as feeling run down and tired or having backaches and headaches. Self-reported health correlates highly with more “objective” measures, such as physician assessments and measures of morbidity, and it predicts mortality net of chronic and acute disease, of physician assessments made by clinical exam, of physical disability, and of health behaviors (Davies and Ware 1981; Idler and Benyamini 1997; Idler and Kasl 1991; Liang 1986). In fact, self-assessed health is a stronger predictor of mortality than physician-assessed health (Mossey and Shapiro 1982).

Age was measured by asking, “In what year were you born?” Age was the year of the survey minus the year of birth. The models used age at midfollow-up (1998), measured as a deviation from age 21.

Education was measured by asking, “What is the highest grade or year of school you have completed?” Answers were coded 0 through 16 years up to a college degree (BS or BA), 17 for some graduate school, 18 for a master’s degree, and 20 for a doctoral or professional degree (PhD, MD, JD, DDS, and EdD). The analyses used education reported at baseline because it is available for all respondents. We experimented with measures of education at midpoint, highest reported education, and average reported education, with Bayesian imputation of missing information on increases and truncating the sample below age 25 (baseline, age 28 midpoint). The results differed little, perhaps because of the high stability of education for most of the sample (about 7% were under age 25 at baseline, and about 14% were under age 30). Education was measured as a deviation from 12 years.

Results

Cumulative Advantage and Its Rising Importance

The results support both the cumulative advantage and rising importance hypotheses. Table 1 gives the results for the basic model of Equations 1 and 2. The model fit the data well, with normed and confirmatory fit indexes of .999 and a root mean square error of the analysis of .018. The coefficient \( v_{12} \) represents the average change in the health erosion coefficient per additional year of education for the youngest cohort in the sample. The coefficient was positive and statistically significant at \( p < .001 \). The result is consistent with the cumulative advantage hypothesis that health erodes more slowly at higher levels of education. The coefficient \( v_{13} \) represents the average change in education’s estimated effect on the rate of erosion in
successively older birth cohorts. The coefficient was negative and statistically significant at \( p < .001 \). The result is consistent with the rising importance hypothesis that education’s effect on the rate of erosion in health was smaller in older cohorts and has been getting larger in newer cohorts. Together, the two coefficients algebraically imply a positive estimated effect of education up through the cohort aged 102 years in 1998, which is above the age range of the sample. Thus, the model’s estimated coefficients are consistent with the hypotheses that health erodes more slowly for the better educated across the range of surveyed cohorts and that this has been getting more so in newer cohorts.

### Table 1


<table>
<thead>
<tr>
<th></th>
<th>Full Sample(^b)</th>
<th>Older than 24 Years Baseline(^c)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>( v_k )</td>
<td>( t )</td>
</tr>
<tr>
<td>( v_0 )</td>
<td>4.476</td>
<td>101.378***</td>
</tr>
<tr>
<td>( v_{10} )</td>
<td>(-3.095 \times 10^{-2})</td>
<td>(-8.570***)</td>
</tr>
<tr>
<td>( v_{11} )</td>
<td>(5.656 \times 10^{-4})</td>
<td>(6.146***)</td>
</tr>
<tr>
<td>( v_{12} )</td>
<td>(4.607 \times 10^{-3})</td>
<td>(7.703***)</td>
</tr>
<tr>
<td>( v_{13} )</td>
<td>(5.658 \times 10^{-5})</td>
<td>(-5.090***)</td>
</tr>
<tr>
<td>( \text{Var}(e_{0}) )</td>
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<td>23.866***</td>
</tr>
<tr>
<td>( \text{Var}(u_{0}) )</td>
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<td>22.706***</td>
</tr>
<tr>
<td>( \text{Var}(u_{1}) )</td>
<td>5.507 \times 10^{-3}</td>
<td>4.404***</td>
</tr>
<tr>
<td>( \text{Cov}(u_{0}, u_{1}) )</td>
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<td>1.376 (ns)</td>
</tr>
<tr>
<td>Mean ( H_{t0} )</td>
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<td></td>
</tr>
<tr>
<td>( SD H_{t0} )</td>
<td>7.380 \times 10^{-1}</td>
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</tr>
<tr>
<td>Mean slope</td>
<td>(-2.966 \times 10^{-2})</td>
<td></td>
</tr>
<tr>
<td>( SD \text{ slope} )</td>
<td>7.534 \times 10^{-2}</td>
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\[ a. \quad \hat{H}_t = v_0 + [v_{10} + v_{11}(A_0 - 21) + v_{12}(E_i - 12)](A_t - 21) + v_{13}(A_t - 21)^2; \]
\[ b. \quad \chi^2 = 47.302, df = 17, \Pi^2/df = 2.782, \text{normed fit index} = .999, \text{comparative fit index} = .999, \text{root mean square error of approximation} = .018. \]
\[ c. \quad \chi^2 = 27.847, df = 17, \Pi^2/df = 1.638, \text{normed fit index} = 1.000, \text{comparative fit index} = 1.000, \text{root mean square error of approximation} = .016. \]

\* \* \*\( p < .001. \) ns if \( p > .05. \)
A vector graph of the model’s equations illustrates how cumulative advantage over adulthood and rising importance across cohorts combine to shape levels and slopes (Mirowsky and Kim 2007). Figure 2 shows the vector graph of predicted levels and changes over the six-year period for every sixth one-year cohort. The origin of each arrow is the cohort’s age and predicted health at baseline ($\hat{a}_{i0} - 3\hat{a}_{i1}$, where $\hat{H}_{it} = \hat{a}_{i0} + \hat{a}_{i1} t$, as defined in the Appendix). The end of each arrow is the cohort’s age and predicted health at the end of the period ($\hat{a}_{i0} + 3\hat{a}_{i1}$). The college-degree arrow is higher and

Figure 2
Vector Graph of Predicted Levels and Slopes of Self-Rated Health for Every Sixth Birth Cohort, for 12 and 16 Years of Education

Note: The numbers to the right of the vertical axis represent the normal-distribution cumulative percentages for the $z$ scores of the predicted midpoint levels ($M = 4.005$, $SD = 0.741$).
flatter than the high-school-degree arrow within each cohort. The arrows do not meet or point toward each other in any cohort. However, the slopes become more similar across the range of cohorts, and the levels do converge across the older cohorts, as detailed later.

Trends by Level of Education

There has been a trend toward faster erosion of health at all levels of education, but more so at lower levels. This shows up in Figure 2 as disjunctions between adjoining arrows. The arrowhead predicting health for the cohort reaching a particular age at the end of the period is below the origin of the arrow for the cohort that began at that age. The trend in the rate of erosion produces a difference between the follow-up and cross-sectional slopes that the eye sees in the diagram as the disjunction. The trend is seen at both levels of education illustrated, but the disjunctions are larger at the high-school-degree level. The disjunctions also increase with the age of the individual’s cohort at midpoint because the trend is in the rate of health’s erosion across adulthood. For this model, the difference between the follow-up and cross-sectional slopes (given by the trend function at the end of the Appendix) is $-v_{11}(A_{i0} - 21) - v_{13}(E_i - 12)(A_{i0} - 21)$.

Taken together, the coefficients $v_{11}$ and $v_{13}$ suggest that the trend toward higher rates of health erosion occurred at all levels of education, but more slowly at higher levels of education. The coefficient $v_{10}$ in Table 1 represents the annual rate of change in self-rated health predicted for persons in the youngest cohort with 12 years of education. The value was negative and statistically significant at $p < .001$, indicating that health declined with age at that level of education. The coefficient $v_{11}$ represents the change in that estimate in successively older cohorts. It was positive and statistically significant at $p < .001$. Thus, the model implies that the rate of health erosion for persons with 12 years of education was lower for older cohorts. The coefficient $v_{13}$ measured the effect of education on the trend in health’s rate of erosion. The coefficient was negative and significant at $p < .001$, indicating that the trend in the rate of erosion was smaller at higher levels of education. However, even at the highest measured level of education (20 years), the trend as measured was toward faster health erosion in newer cohorts. This suggests the possibility that, were it not for rising levels of education, there might have been a trend toward faster erosion of self-rated health in adulthood.
Testing Old-Age Convergence

One may justly ask whether the disintegration coefficient $v_3$ really is constant across levels of education. We respecified the model with $v_3 = v_{30} + v_{31}(E_i - 12)$, thus allowing faster (although delayed) disintegration at higher levels of education. The resulting estimates were $-4.699 \times 10^{-6}$ for $v_{30}$ and $-1.559 \times 10^{-7}$ for $v_{31}$. The negative education coefficient is consistent with the hypothesis of faster disintegration at higher levels of education. However, the $t$ values were $-5.251$ and $-0.552$, respectively, with $p < .001$ for the disintegration coefficient at 12 years of education and $p = .581$ for the average change in it across education levels. This suggests that any slope convergence within cohorts was relatively small and/or chiefly delayed until beyond the 80s.

Apparent Convergence Phenomena

Graphs of levels and slopes show how a trend toward larger differences in health across levels of education can contribute to the impression of old-age convergence. For example, Figure 2 shows that the slopes get increasingly similar as midpoint age increases. This happens even though the model specifies no old-age convergence in cohorts as they age. Indeed, the model specifies that the differences in health across levels of education continue growing at a steady rate. The apparent convergence of slopes occurs for two reasons. One is that the differences in rates of health erosion were smaller across levels of education in older cohorts. The other is that the disintegration component $v_3(A_i - 21)^3$ increasingly dominated the sum of erosion and disintegration at older ages. It was the same in the model for all levels of education, making the slopes increasingly similar.

The trends had even more striking effects on differences in the level of self-rated health than on differences in the slope. Figure 3 shows the levels predicted at midpoint for 12 and 16 years of education. They approximate the cross-sectional age curves for 1998 and also the curves that would be produced by a cubic age-basis latent-growth model averaging across cohorts. The model and results imply divergence in level through age 60 and progressive convergence for successive ages beyond that. When restricting the model to persons over age 24, the estimates imply divergence until age 62. These are close to Herd’s (2006) age-basis estimates of convergence in physical impairment after age 63 in the Health and Retirement Survey sample.
Virtual cohort projections of the trajectories implied by the model suggest that persons with only 12 years of education in cohorts currently approaching the middle of adulthood may be heading for poor self-rated health in old age. Those with 16 years of education may barely stay ahead of older generations at the 12th grade level, averaging only fair health in old age. Figure 4 shows the virtual cohort curves implied by the model for the cohorts born in 1941 and 1965, aged 57 and 33 years at the midpoint of the

Note: The greatest difference occurred for the cohort aged 60 years at midpoint (born in 1938). Cross-sectional age curves for 1998 and curves produced by a cubic age-basis latent-growth model averaging across cohorts would be similar.

Virtual Cohort Projections

Virtual cohort projections of the trajectories implied by the model suggest that persons with only 12 years of education in cohorts currently approaching the middle of adulthood may be heading for poor self-rated health in old age. Those with 16 years of education may barely stay ahead of older generations at the 12th grade level, averaging only fair health in old age. Figure 4 shows the virtual cohort curves implied by the model for the cohorts born in 1941 and 1965, aged 57 and 33 years at the midpoint of the
Study and currently (2007) aged 66 and 42 years. The figure illustrates the combined implications of the trend toward faster erosion of health at all levels of education and the trend toward greater differences in the rate of erosion across levels of education. It is important to recognize that 20- to 40-year projections from 6 years of follow-up are extremely tentative. The virtual cohort curves should be read primarily as an illustration of the implications should trajectories and trends remain as they were during the period of observation. If they do, the self-rated health trajectories of persons with less than a college degree will be dismal.

Health Selection

The final issue is the extent that health selection influences the estimates. It seems likely to flatten trajectories, particularly the most negative
ones. As illustrated in Figure 5, health selection probably attenuates the cubic $v_3$ disintegration component more than the linear $v_1$ erosion component. Any such flattening will mostly affect estimates for persons aged 60 years and older.

To see if health selection seemed a problem, we split the sample into two age groups: 1,494 persons under age 60 at baseline and 1,098 persons aged 60 years or older. We estimated the model separately in the two groups. The results at first seemed to indicate substantial health selection effects. The estimated $v_3$ disintegration coefficient was $-7.593 \times 10^{-6}$ in the younger segment ($t = 3.561, p = .033$) but only $-0.831 \times 10^{-6}$ and not significant ($t = -0.417, p = .676$) among the seniors. However, tests of the differences in coefficients across the two groups showed that the disintegration coefficients were not significantly different ($\chi^2$ difference = 2.731, $p = .098$).
found that the estimate of $v_3$ among seniors correlated at .854 with the estimate of $v_0$ and at –.725 with the estimate of $v_{11}$. Both of those coefficients were statistically significant in both groups, and neither was significantly different across groups ($\chi^2 = 0.598$ and 2.140, respectively, $p = .439$ and .144). Setting both (or either) of those equal across the two groups greatly reduced the standard error of the $v_3$ estimate among seniors (to $0.941 \times 10^{-6}$, down from $2.020 \times 10^{-6}$).

The constrained split-sample model showed a smaller $v_3$ disintegration coefficient for seniors than for others, as expected if health selection affects the older part of the sample more. Table 2 gives the two disintegration coefficients estimated when $v_0$ and $v_{11}$ were constrained equal across the two broad age groups. The coefficient estimated for seniors was about 26.8% smaller than the one estimated for the young and middle-aged adults. However, the standard error of the estimate for nonseniors was large relative to the difference in coefficients (2.749 compared with 2.088). As a result, the difference was not statistically significant ($\chi^2$ difference = 0.769, $p = .381$). When the two were constrained to be equal, the resulting estimate was within a few percent of the one for seniors. Taken together, the results suggest that health selection effects on the disintegration coefficient may exist but are not too great when combining the full range of adult ages.

**Accelerating Trend**

Interestingly, the coefficients most different across the two large age groups were the ones associated with education. Taken together, they suggest that the trend in education’s relationship to the rate that health erodes has been accelerating. The estimated effect of education on the erosion slope ($v_{12}$) was 1.8 times larger in the younger part of the sample (or 43.2% smaller in the older part). The estimated trend toward larger effects of education ($v_{13}$) was 3.3 times larger in the younger part (70.1% smaller in the older part). This seems like good news, until one considers that the trend for the sample as a whole was toward faster erosion at all levels of education. However, this may not be equally so for the younger and older segments. In the model, the trend is represented by $v_{11} + v_{13}(E_i - 12)$, where $v_{11}$ represents the trend in the erosion coefficient for persons with 12 years of education. That coefficient was not significantly different across the two segments. In contrast, the trend in the effect of education, measured by $v_{13}$, differed by an order of magnitude. As a result, solving for the level of education at which the trend became positive yielded very different critical points. For seniors, the model predicted a beneficial trend only beyond 25 years of
education, which is well above the maximum score of 20. For the young and middle-aged, the model predicted a beneficial trend beyond 16 years, or the college degree level.

**Discussion**

The results support both the cumulative advantage and the rising importance hypothesis. Self-rated health eroded more slowly at higher levels of education, progressively enlarging the gap as the cohorts aged. The rate of divergence across levels of education was greater in younger cohorts. To our knowledge, this is the first replication of the three-way interaction in

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**Table 2**


<table>
<thead>
<tr>
<th>Under Age 60 at Baseline</th>
<th>Age 60 or Older at Baseline</th>
</tr>
</thead>
<tbody>
<tr>
<td>( v_k )</td>
<td>( t )</td>
</tr>
<tr>
<td>( v_0 )</td>
<td>4.412</td>
</tr>
<tr>
<td>( v_{10} )</td>
<td>(-2.941 \times 10^{-2})</td>
</tr>
<tr>
<td>( v_{11} )</td>
<td>7.155 \times 10^{-4}</td>
</tr>
<tr>
<td>( v_{12} )</td>
<td>7.984 \times 10^{-3}</td>
</tr>
<tr>
<td>( v_{13} )</td>
<td>(-18.017 \times 10^{-5})</td>
</tr>
<tr>
<td>( v_3 )</td>
<td>(-7.777 \times 10^{-6})</td>
</tr>
<tr>
<td>( \text{Var}(e_{it}) )</td>
<td>2.645 \times 10^{-1}</td>
</tr>
<tr>
<td>( \text{Var}(u_{it}) )</td>
<td>3.455 \times 10^{-1}</td>
</tr>
<tr>
<td>( \text{Var}(u_{it}) )</td>
<td>6.199 \times 10^{-3}</td>
</tr>
<tr>
<td>( \text{Cov}(u_{it}, u_{it}) )</td>
<td>5.936 \times 10^{-3}</td>
</tr>
<tr>
<td>( \text{Mean } H_{it} )</td>
<td>4.195</td>
</tr>
<tr>
<td>( \text{SD } H_{it} )</td>
<td>6.223 \times 10^{-1}</td>
</tr>
<tr>
<td>( \text{Mean slope} )</td>
<td>(-1.995 \times 10^{-2})</td>
</tr>
<tr>
<td>( \text{SD slope} )</td>
<td>7.968 \times 10^{-2}</td>
</tr>
</tbody>
</table>

a. \( \hat{H}_{it} = v_0 + \{v_{10} + v_{11}(A_{it} - 21) + v_{12}(E_{it} - 12) + v_{13}(A_{it} - 21)(E_{it} - 12)(A_{it} - 21) + v_3(A_{it} - 21)\}; \)

b. \( \chi^2 = 59.349, df = 36, \chi^2/df = 1.650, \text{normed fit index} = .999, \text{comparative fit index} = 1.000, \text{root mean square error of approximation} = .016. \)

*\( p < .05. **p < .01. ***p < .001. ns \) if \( p > .05. \)
Lynch’s (2003) sequential cross-sectional model. We were able to measure and test the trend in education’s effect on the rate of erosion in a longitudinal analysis by measuring and testing the implied difference between follow-up and cross-sectional slopes. Subsidiary analyses suggest acceleration in the trend in education’s effect. The differences in rates of erosion across levels of education and the trend toward larger differences in those rates were greater among the young and middle-aged half of the sample (under age 60) than among the senior half.

Given the rising levels of education in the United States, its rising importance to self-rated health may seem beneficial. Unfortunately, much of the measured trend in the rate of divergence across levels of education reflects a trend toward a faster erosion of health at lower levels of education. For the sample as a whole, the picture seems especially grim: a trend toward a faster erosion of health even at the highest level of education. With the sample split between seniors and others, things look better for the well-educated members of the young and middle-aged cohorts. For them, the results imply that 16 years of education is the boundary between a trend toward slower health erosion for those with more schooling and toward faster health erosion for those with less. On the whole, though, the trends in self-rated health are generally negative, similar to the trend in cognitive function found by Alwin et al. (2005, 2006, 2006) in the Health and Retirement Survey.

We found little evidence that a slower erosion of health through middle age for the better educated is followed by faster disintegration in old age. When added to the model, the coefficient representing old-age convergence in self-rated health had the predicted sign but was far from statistical significance. We also found little evidence that health selection greatly flattens the estimated rate of erosion in old age. With the sample split, the disintegration coefficient was smaller for the seniors than for the young and middle aged, as health selection would make it. However, the difference in coefficients between seniors and others was far from statistical significance. These observations do not rule out the possibility of significant compression or selection effects in much larger or much older samples. Rather, they suggest that those factors contribute little to the levels and slopes of self-rated health compared with the trajectory and trends described by our model.

Two elements of the trajectories and trends combine to produce what may be seen as old-age convergence in self-rated health. First, the trajectory is defined by a constant rate of erosion that dominates through middle age and a slowly accelerating rate of disintegration that becomes increasingly dominant in old age. The rate of erosion moderates substantially at higher levels of education, but the rate of disintegration does not. As a
result, the changes in health over follow-up differ greatly by level of education through middle age, but less and less so in old age. Differences in the levels of health remain large and continue to grow. Second, the trend toward larger differences across levels of education in the rate of erosion can seem like old-age convergence. Slopes are more alike among older respondents because their rates of erosion were more alike. Levels of health are more alike for the same reason. Even so, self-rated health continues to diverge across levels of education in the older cohorts as they age.

The results of this study raise questions about modeling, biological mechanisms, and sociodemographic mediators and moderators. For modeling, the split-sample results suggest an accelerating trend rather than a more or less linear one. This raises the question of how best to model that acceleration. The ideal would be a transformation of the cohort indicator $A_{i0}$ that implies acceleration but does not require additional parameters and improves model fit. Perhaps substituting $\ln(A_{i0} - 17)$ would work. Theoretically, though, an accelerating trend suggests a period of social transformation that eventually would decelerate to a new stable state. The results show what may be signs of this, in the trend toward faster erosion at all levels of education among the older cohorts but only below the college-degree level for the young and middle-aged cohorts. Modeling the rise and fall of a trend will require additional parameters and possibly nonlinear methods of estimation, larger samples, or longer follow-up periods.

The model and results also raise questions about the distinct biological mechanisms behind the separate erosion and disintegration components of the health trajectory. The function was suggested by ideas linking the delay of morbidity to the speed of its increase approaching a ceiling on longevity (e.g., House et al. 2005) and by the results of exploratory latent-growth models of physical impairment suggesting a long steady increase through middle age followed by an accelerating increase in old age (Mirowsky and Ross 2005a). The erosion component, moderated by education or something closely linked to it, probably reflects biological accumulators linked to lifestyle and chronic stress, as summarized in the introduction. Currently, those are relatively well understood (e.g., Mirowsky and Ross 2003), although much remains to be learned.

The bigger uncertainty for now is the biological mechanism of the disintegration component. What is it, and why does it seem relatively independent of the rate of erosion and the factors that regulate that rate? Cumulative oxidative damage to the mitochondrial DNA in cell lines may be responsible, or telomere limits on the number of times nuclear DNA can divide (Hayflick 1998; Wallace 1997). If so, why would these bioaccumulators be
relatively independent of the ones mediating the rate of erosion? Are they really unrelated to rising blood pressure, rising insulin resistance, declining aerobic capacity, the rise and decline of body fat, diminishing resistance to infections, and other likely mediators of the relationship between education and the rate of health erosion? Biologists sometimes speak as if the total number of calories burned per unit of body mass over the lifetime is the sole or paramount limit on life span (Perez-Campo et al. 1998), in part because mitochondria produce the adenosine triphosphate that fuels cellular processes. If that is true, why would it be relatively independent of the same forces that link education to the erosion of health, and is it? So far, too little is known about the relationship of cumulative oxidative damage and diminishing telomere length to social and behavioral factors and to the bioaccumulators that link them to rates of health erosion.

The final set of questions raised relate to sociodemographic mediators and moderators. We deliberately specified the simplest possible model embodying the central ideas of erosion, disintegration, cumulative advantage, and rising importance. Although simple in its basic form, the model’s implications are varied and complex. The results raise two sets of questions. First, what are the social and behavioral factors that mediate the cumulative advantage (or disadvantage) associated with education and the rising importance of that association? The introduction summarizes the prime candidates, and the reasons will not be elaborated here. The next step will add them to the equation describing the rate of erosion to see if the addition diminishes the coefficients associated with education and its interaction with cohort. Second, what ascribed or achieved statuses regulate the associations embodied in the model? Stated another way, what factors alter the coefficients of the model? A number of possibilities come to mind, but the most prominent is sex. Within the past several decades, women’s educational attainment has closed the gap with men’s. This is heavily a cohort phenomenon. Women now are the majority of students at colleges and universities. They now get the majority of bachelor’s and master’s degrees, and they are rapidly closing the gap at the doctoral level. The theory of resource substitution suggests that education will be more important to the erosion of women’s health than to men’s (Ross and Mirowsky 2006). Perhaps it also will be more important to the trends in women’s health.

**Conclusion**

Differences in self-rated health across levels of education grow throughout adulthood, including old age. The rate of divergence has been growing
across cohorts. Health levels and slopes seem to converge in old age partly
due to the rates of divergence being smaller in older cohorts and partly
due to disintegration rates being similar across levels of education and
increasingly dominate health changes in old age. Even so, the differences
in health across levels of education continue to grow.

Appendix

Definition of Aging-Vector Level and Slope

Measure time as a deviation from mid-follow-up:

\[ t = A_i - A_{i0} = S_i - S_0. \]

Measure individual health during the study period as a linear function of
time:

\[ H_i = a_{i0} + a_{i1}t + e_{it}. \]

Define the level \( (a_{i0}) \) and slope \( (a_{i1}) \) coefficients as combinations of
expected values and individual random deviations:

\[ a_{i0} = \hat{a}_{i0} + u_{i0}, \]

and

\[ a_{i1} = \hat{a}_{i1} + u_{i1}. \]

Derivation of the Expected Level (\( \hat{a}_{i0} \)) Equation

\[ \hat{H}_i = v_0 + v_1(A_i - 21) + v_3(A_i - 21)^3. \]

Fix \( t = 0 \).

\[ \hat{a}_{i0} = (\hat{H}_i | t = 0), \]

and

\[ \hat{a}_{i0} = v_0 + v_1(A_{i0} - 21) + v_3(A_{i0} - 21)^3. \]

Substitute the moderator function for \( v_1 \):

\[ \hat{a}_{i0} = v_0 + [v_{10} + v_{11}(A_{i0} - 21) + v_{12}(E_i - 12) + v_{13}(A_{i0} - 21)(E_i - 12)](A_{i0} - 21) + v_3(A_{i0} - 21)^3. \]
Multiply through and collect terms:
\[
\hat{a}_{i0} = v_0 + v_{10}(A_{i0} - 21) + v_{11}(A_{i0} - 21)^2 + v_{12}(A_{i0} - 21)(E_i - 12) + v_{13}(A_{i0} - 21)^2(E_i - 12) + v_3(A_{i0} - 21)^3.
\]

**Derivation of the Expected Slope (\(\hat{a}_{i1}\)) Equation**

\[
\frac{\partial H_t}{\partial A_{it}} = v_1 + 3v_3(A_{it} - 21)^2
\]

Set \(t = 0\).

\[
\hat{a}_{i1} \approx \left(\frac{\partial H_t}{\partial A_{it}}\right)|_{t = 0}
\]

and

\[
\hat{a}_{i1} \approx v_1 + 3v_3(A_{i0} - 21)^2.
\]

Substitute the moderator function for \(v_1\).

\[
\hat{a}_{i1} = v_{10} + v_{11}(A_{i0} - 21) + v_{12}(E_i - 12) + v_{13}(A_{i0} - 21)(E_i - 12) + 3v_3(A_{i0} - 21)^2.
\]

**Derivation of the Trend Function**

Define the trend function as the difference between expected follow-up and cross-sectional slopes, which equals the slope of predicted health with respect to cohort at midpoint (time zero):

\[
T_i = \hat{a}_{i1} - \frac{\partial a_{i0}}{\partial A_{i0}} = \left(\frac{\partial H_t}{\partial E_i}\right)|_{t = 0}
= -v_{11}(A_{i0} - 21) - v_{13}(E_i - 12)(A_{i0} - 21)
\]

**Notes**

1. Lynch (2006) extended the sequential cross-sectional model to analyze trends in the indirect effect of education through income but dropped the three-way interaction to simplify the path model.
2. There is a practical reason why specifying functions of time (rather than age) within individuals goes together with having level and slope coefficients differing between individuals as a function of birth year. The alternative age-basis model would describe an individual’s health as a function of age at each observation, with the coefficients influenced by year of birth. Although those models make sense intuitively, apparently they generally do not converge, presumably because of collinearity between age and year of birth. We did not find any published adulthood age-basis health trajectory model with year of birth as a predictor of its coefficients. Notably, when Miyazaki and Raudenbush (2000) developed a significance test for variance across cohorts in a trajectory’s coefficients, they used a time-basis model, presumably because an age-basis model (the usual approach in psychology) would not work.

3. We refer to the hypothetical function of age and cohort as a virtual cohort model to distinguish it from a synthetic cohort model. The latter is a special case that assumes no interaction between age and cohort. In a synthetic cohort model, the age-specific slopes are the same across cohorts, and the trajectories differ across cohorts by amounts presumed constant as the cohorts age. A synthetic cohort model has a trend in the trajectory’s intercept (constant with respect to age) but not in the coefficients governing the trajectory’s shape. The “free-intercept model” in Alwin et al. (2005, 2006, 2006) is a synthetic cohort model.

4. Equation 1 is not a level 1 within-person equation, as commonly specified in multivariate linear modeling and hierarchical linear modeling applications. It is a single between-person (level 2) equation that implicitly describes both the predicted health level at mid-follow-up and the predicted health slope over the period.

References


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