
Do Functional Health Inequalities Decrease in Old Age?

Educational Status and Functional Decline Among the 1931-1941 Birth Cohort

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Although the relationship between educational attainment and health is well established, there is disagreement about how this relationship changes across the life course. Some studies have shown that educational health disparities widen in middle age and then start to abate in old age, whereas others have shown that health disparities continue to widen in old age. The author used the 1992-2002 Health and Retirement Study to shed new light on this old debate. Because findings of declining health inequalities in old age are often dismissed as a product of mortality selection and cohort effects, this study primarily aimed to address these claims. The author found that cohort effects and mortality selection do not fully explain shrinking educational disparities in functional health in old age. Further work is needed to explore explanations for diminishing health disparities health in old age.

Keywords: *aging; health; disparities*

For over 30 years, sociologists and epidemiologists have shown, with both cross-sectional and longitudinal data, that people at lower educational levels, compared with those at higher educational levels, have poorer health and are more likely to die across the life course (House 2001). People without high school degrees are more likely to report lower health status; higher numbers of chronic conditions, acute conditions, and functional

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limitations; and poorer mental health (House et al. 1994; Mirowsky and Ross 2001; Smith and Kington 1997).

But despite these consistent findings, there is some controversy about how educational differences in health change across the life course. Some studies have shown that educational differences in health continue to widen in old age, maintaining the pattern exhibited across early life and midlife (Aneshensel, Frerichs, and Huba 1984). Societal disadvantages compound and accumulate across the life course and into old age, exacerbating health inequality (Ross and Wu 1996). Other studies have shown that although educational differences in health increase from young age into middle age, these differences start to shrink again in old age (Beckett 2000; House et al. 1990, 1994; Kitagawa and Hauser 1973; Newacheck et al. 1980). In short, age acts as a leveling force, shrinking the gap in health between those with low and high educational attainment.

This study sheds new light on this debate by following a single cohort (those born from 1931 to 1941) over time with a unique set of longitudinal data, the Health and Retirement Study (HRS). These data can address the primary methodological problems confounding this debate, particularly concerns about cohort effects and mortality selection. As in comparable studies, education is used as a measure of socioeconomic status (SES) because other measures, such as income, are more subject to critiques of reverse causality (that poor health causes low income).

The Current Literature

Cumulative disadvantage theory predicts that health inequalities compound and grow as people age (Lynch 2003; Ross and Wu 1996). Differences in health by educational attainment continue to get larger all the way across the life course. Unhealthy work conditions and living environments, high levels of stress, and general economic deprivation enact a mounting toll on individuals' health, gradually expanding socioeconomic health differences as people age. Ross and Wu (1996) used both cross-sectional and longitudinal data sets to show diverging educational differences in physical functioning, physical well-being, and self-reported health across the life course. Aneshensel et al. (1984) reported similar findings, though their old-age group was composed of people aged 45 and over, which is likely too broad an age sample to detect diminishing health inequalities. Their health measure was depression.

Most studies, however, support the age-as-leveler hypothesis; health inequalities expand through midlife but start to diminish just after age 60.

This holds true for both morbidity and mortality (House et al. 1994; Kitigawa and Hauser 1973; Kunst and Mackenbach 1994; Mustard et al. 1997). Longitudinal studies have confirmed the finding (Beckett 2000; House et al. 1994; Deaton and Paxson 1998). Both House et al. (1994) and Beckett (2000) specifically used measures of functional limitations. Beckett captured traditional instrumental and activity-of-daily-living limitations, whereas House et al. captured whether individuals had any difficulty doing heavy housework or walking several blocks or whether they were confined to bed. One explanation for diminishing health inequality in late life is that people with high educational attainment are catching up to those with low educational attainment. Those with high educational attainment can postpone health decline for only so long (Beckett 2000; House et al. 1994). Thus, the cause of diminishing health inequalities may be quickening rates of decline among those with high educational attainment compared with steady rates of decline for those with low educational attainment. Before age 60, people with college degrees have a very slow rate of health decline, but after age 60, their rate of health decline accelerates, producing smaller educational health disparities in old age.

The alternative explanation for declining health inequalities in late life has been given less attention. The social context changes significantly in people's 60s, which is when health inequalities diminish. Both retirement and a massive welfare state alter the social context for older Americans, particularly for those on the bottom rung of the SES ladder. In general, studies have shown that although income inequality is high among older Americans, poverty and economic hardship are lowest among this age group, likely because of Social Security and Medicare (Mirowsky and Ross 1999). Given that associations between SES and health are concentrated among those at the bottom of the ladder, changes in their social context could lead to changes in their health.

It is possible that the interactive effects of retirement, Social Security, and Medicare slow down health deterioration for those with limited educational attainment. People in occupations that have been damaging to their health can exit the labor force and be guaranteed a stable source of income through Social Security and universal health insurance through Medicare. Studies have shown that manual labor and generally occupations dominated by those with limited educational attainment have negative effects on health (Case and Deaton 2003). Furthermore, Social Security guarantees many people who had volatile incomes through their working lives a steady source of income in old age, protecting them from the negative health ramifications associated with large income drops (Herd et al. 2004). Finally, older

Americans are guaranteed health insurance for the first time in their lives. There is evidence that this may have an impact on older people's health. Those uninsured before retirement exhibit an increase in the use of medical care after obtaining health insurance through Medicare (Decker and Rapaport 2002; Dor, Sudano, and Baker 2003). Moreover, there is evidence that this uninsured population shows improvements in health after signing on to Medicare (Dor et al. 2003). If there is an advantageous effect of retirement, Social Security, and Medicare on the health of those with low educational attainment, their rate of health decline would start to slow after age 60. Thus, this slowing rate of health decline among those with low educational attainment would lead to shrinking educational health disparities.

New studies, however, are challenging whether findings of declining health inequalities in late life reflect a real relationship between education, age, and health. Studies using generalizable age-representative samples, either cross-sectional or longitudinal, may be revealing cohort effects, not age effects (Mirowsky and Ross 2003). Using both replicated cross-sections of the National Health Interview Survey and the longitudinal National Health Epidemiologic Follow-Up Study (NHEFS), Lynch (2003) showed that educational differences in self-reported health are larger among younger cohorts than among older cohorts. This result supports the hypothesis that cross-sectional and longitudinal studies showing decreasing health inequalities in old age are really displaying cohort effects, as opposed to age effects. But it is possible that cohort differences in health inequality are increasing over time and that health inequalities diminish in old age within cohorts. No study has followed an individual cohort over time to address how health inequalities change within a cohort as it ages.

Others have argued that mortality selection is producing false findings of diminishing health inequalities in old age. People at lower levels of educational attainment are more likely to die at younger ages, potentially leaving behind a more robust group of low-educated survivors relative to high-educated survivors (Beckett 2000; House et al. 1994; Lynch 2003; Noymer 2001). Thus, health disparities seem to have diminished in old age, because the sickest people at low educational levels have died, leaving a biased sample of relatively healthier persons with limited educational attainment. Robust low-educated survivors may show slowed health decline, making group differences appear smaller. The selective-mortality hypothesis would likely produce the same effect that the social-context hypothesis would produce, a slowed rate of health decline in old age for those with low educational attainment. It is critical to note, however, that mortality selection

will produce diminishing health inequalities only if high-educated nonsurvivors are not causing a parallel effect on the observed mean of this group's health. Low-educated survivors must be comparatively more robust than high-educated survivors for there to be bias.

A few studies have tried to test whether selective mortality explains findings of diminishing health inequalities (Beckett 2000; Lynch 2003). Although not often portrayed this way, these studies are really thought experiments. That is, the critique that mortality selection produces declining health inequalities is akin to saying that if people did not die (remained in the sample) and remained on their trajectories of health decline, health inequalities would continue diverging in old age. Both studies used longitudinal data. They included nonsurvivors in their samples and imputed health outcomes for the years after respondents had died. Beckett (2000) was the first to address concerns about mortality selection using two waves of the NHEFS collected in 1982-1984 and 1992. She used multiple imputation (e.g., Rubin 1987) to impute health variables for those who died between the first and second waves and did not find a meaningful impact of mortality selection. The main problem with the imputation strategy, however, was the use of survivor scores to impute health scores for decedents. If mortality selection produces diminishing health inequalities, survivors' health must be more robust than decedents. Thus, the results could be biased. Additional sensitivity analyses, however, that varied the composition of the sample provided little support for a substantial effect of mortality selection (Beckett and Elliot 2001).

Lynch (2003) used four waves of the NHEFS collected in 1971, 1982, 1987, and 1992. He used a random coefficient growth curve model. Basically, this model produces a slope for each of those who die on the basis of their individual health scores prior to death. Thus, the projected health scores of those who die are not biased by the potentially more robust survivors. Past individual health is used to predict future individual health. One limitation with the NHEFS, however, is the large gaps in time between waves. Health measurements taken between 5 and 10 years before death do not produce the most accurate estimates of future decline.

Given the contradictory findings, further analyses are clearly needed, especially ones following a single cohort over time, to evaluate the age-as-leveler versus the cumulative disadvantage hypothesis. Thus, I used the longitudinal HRS, which followed a single cohort of older Americans (aged 51 to 61 in 1992) over 10 years. This design alleviates concerns about cohort effects and allows for mortality selection to be tackled.

Research Questions

Two questions drove this research. First, do functional inequalities grow, stagnate, diminish, or disappear in old age? This was the main question addressed in this study. But I also provide preliminary evidence for a second question: If educational differences in functional health diminish or disappear, why does this happen? Is it produced by a changed trajectory for those with high educational attainment, specifically an accelerated rate of decline? This finding would lend support to biological hypotheses about why health disparities shrink in old age; educational health disparities are largely driven by the fact that those with high educational attainment are able to hold off health decline until later ages than those with low educational attainment. Their morbidity is compressed to a short period in old age when they “catch up” to those with low educational attainment. Alternatively, are diminishing functional inequalities produced by a changed health trajectory for those with low educational attainment, specifically a slowed rate of functional decline? This finding would lend support to the hypothesis that retirement and a generous welfare state diminish educational health disparities in old age. Although these findings would provide support for the previous hypotheses, however, they are not causal evidence.

Data and Methods

As already stated, the data for these analyses are derived from the longitudinal HRS. Questionnaires are administered in alternating years. The baseline year was 1992, and the last wave of available data was collected in 2002. Six waves of data were used in this analysis. The HRS had an initial sample of 12,600 persons in 7,600 households. The primary sample was those born between 1931 and 1941, but spouses were also interviewed, though they were not included in this sample to maintain the appropriate age representation. The initial sample did not cover the institutionalized population, but those present in the first wave who enter institutions (such as nursing homes) are followed. Proxy interviews are used if a participant cannot participate in follow-up interviews.

The final sample, after eliminating non-age-eligible individuals, was 9,825. Given that this was an older sample, mortality was clearly an issue. Among the 9,825 respondents, 842 individuals, or 8.6% of the sample, died

by Wave 5. In short, although those with low educational attainment were disproportionately represented among the deceased, those with high educational attainment constituted a reasonable share of the deceased. Although those without high school degrees represented 28% of the sample, they represented 36% of the deceased. College graduates represented 18.5% of the sample and 12% of the deceased.

Table 1 provides descriptive data. Independent variables included educational attainment, sex, race, geographic region (Northeast, South, West, and Midwest), and whether individuals resided in metropolitan statistical areas. Regional variables, commonly included in studies examining this question, were necessary because they predict both education and health (as do race and sex). The model was limited to these variables because introducing a variable such as income, which education and health predict, creates endogeneity problems. The dependent variable was a measure of functional status. The variable was based on these seven functional measures: Is it difficult to (1) walk several blocks? (2) get up from a chair? (3) stoop, kneel, or crouch? (4) lift or carry 10 pounds? (5) pick up a dime? (6) extend your arms over shoulder level? and (7) push or pull large objects?

The final measure was an ordered four-category variable with zero limitations, one or two limitations, three to five limitations, and six or more limitations, approximately. This reconstruction of the dependent variable was necessary to run an ordinal growth curve model. Because the jumps between each additional limitation are not equivalent, an ordinal model was more appropriate than an ordinary least squares model. That said, additional analyses (not shown) conducted using ordinary least squares produced relatively equivalent results.

There is considerable disagreement on the best measure of functional limitations. The additive approach I used is common in other studies, particularly ones using the HRS (Smith and Kington 1997; Spector and Fleishman 1998; Wallace and Herzog 1995). Furthermore, Cox proportional-hazard regressions (not shown) showed a strong and increasing predictive effect on mortality with increasing numbers of limitation on this summary functional limitation variable. The advantage to using this particular set of limitations is that it can capture more functional variation than harsher measures such as activities of daily living, which encompass severe limitations, such as an inability to eat or dress. In essence, it is far more age appropriate for this sample of near-old individuals. Similar measures can be found in the Survey of Income and Program Participation and the Medicare Current Beneficiary Survey. And more general measures of functional limitation that move

Table 1
Means or Percentages and Standard Deviations
(in parentheses) of Variables From the Health
and Retirement Study (1992, baseline)

Variable	Value
Birth year	1936 (3)
Sex	
Male	46.9
Female	53.1
Race	
Black	21.1
White	78.9
Education	
No high school degree	28.08
High school degree or more	53.45
College degree or more	18.47
Functional status	
1	38.9
2	43
3	15.4
4	2.7
Region	
West	19.26
Midwest	24.15
South	35.88
East	21.81
Community	
City	45.56
Suburb	28.09
Rural	26.34
Sample size	9,825

beyond limitations to activities of daily living and instrumental activities of daily living, which is the intent of the HRS measures, can be found in most health surveys.

Addressing Mortality Selection

The primary strategy for addressing mortality selection is to keep those who die over the survey period in the sample. But how should health values

be imputed for these decedents? There are numerous ways to impute health. And in fact, there is no perfect way. In essence, this is a thought exercise. If those exiting a survey through death cause diminishing health inequalities in old age, what happens when they are left in the sample and decline at the rate at which they were declining before they died? Does this eliminate the effect?

That said, there are two general rules that underlie the imputation strategy used. First, because the concern is that the sample of decedents is somehow "select," it is important that their predicted health trajectories are based largely on their prior health status. Because the HRS is longitudinal, there are prior health scores for decedents to accomplish this. Second, I used numerous sensitivity analyses. Unlike normal missing data problems in which there is a real value to predict, such as with a respondent who refuses to participate in follow-up waves, there is no real future health value for decedents, because they are dead. Hence, given that this is a thought exercise, it is useful to test a variety of different health trajectories for decedents.

The main imputation approach I used was a random coefficient growth curve model (Lynch 2003). Those who died or left the study were left in the analysis. Because of the concern that decedents were select, or rather more robust than survivors, imputed health measures cannot be based on surviving respondents' health. This model estimated their individual slopes largely on the basis of health data from preceding waves in which they were present. The HRS is particularly advantageous in this regard because the data waves are only two years apart. In other data sets, such as the National Health and Nutrition Examination Study, up to 10 years lapse between waves, and the number of waves is small. If I relied on the health status of individuals previous to death to predict their health decline, data from 10 years previous to death could be misleading as a predictor of their imputed future health decline.¹ One further step to address the mortality selection problem is to simply show the impact of including and excluding the individuals who die in the random coefficient growth curve analyses. If the interaction between age and education shows substantial change with the inclusion and exclusion of individuals who leave because of mortality, I would find support for a meaningful impact of selection. If the estimate differences were small, it would lend support to the idea that mortality selection is not primarily driving diminishing health inequalities in old age.

The additional sensitivity analyses I used included assuming the following health trajectories: (1) a flat rate of decline whereby the last reported health score is the imputed health score, (2) a linear rate of decline whereby decedents are forced to have an accelerated rate of decline, and (3) all decedents

are assumed to have the worst possible health score. Last, I used multiple imputation to account for the variance produced by all four different approaches in model standard errors (Rubin 1987). Because of space constraints, I report only on the main imputation strategy. These different approaches validated the central finding in this study.

Although these approaches will help address concerns about mortality selection, there is one inherent problem with the HRS sample. Although in one regard it is ideal because this age group (51 to 61 years) just precedes the point in time when the strongest debate exists as to whether health inequalities continue to expand or start to diminish, in another regard, this age group is problematic because it has already been subject to selective mortality. An ideal longitudinal sample would follow people from birth to death. Given that these data do not exist, the HRS, though imperfect, is an incredibly valuable data source. Moreover, some of these concerns can be alleviated by the fact that mortality rates are generally low until people's 60s, after this sample was selected.

The Random Coefficient Growth Curve Model

The study used a random coefficient growth curve model. The data were constructed in a person-age case fashion. For each age, an individual had one observation. The covariates predicted functional decline across individuals in their 50s and 60s and into their early 70s. A random-effects model is particularly useful for the analyses of individuals across time. It accounts for variation within and between individuals across time. Individual growth curve models address both within and between person change. The first equation, or the Level 1 model, was the within-person estimator. The second equation, or Level 2 structural model, was the between-person estimator. Individual change or individual growth rates were a function of group characteristics, including a series of non-time-varying covariates: race; sex; educational attainment; geographic region; and urban, suburban, or rural region.

Results

Do educational differences disappear, diminish, stagnate, or continue to grow? The first set of results in Table 2, which shows three different random coefficient ordinal models from the HRS data, helps answer this question. The coefficients in these models are not intuitively interpretable, but the direction and significance level tell the needed story. The first model in Table 2 includes only education. The intercept for education was positive;

Table 2
Random Coefficient Ordinal Models of Functional Decline

Variable	Model 1		Model 2		Model 3		Model 4	
	Intercept	*Age (π_j)	Intercept	*Age (π_j)	Intercept	*Age (π_j)	*Age (π_j)	*Age ² (π_{2j})
Education	0.1656***	.0038***	0.1573***	.003***	0.1236***	.0132***	.013543***	-.000584***
Women	-0.9842***	.0153**	-0.9876***	.0167	-0.9876***	.0167	.013077	.000057
Black	-0.2896**	-.0204*	-0.2073	-.0472	-0.2073	-.0472	-.0564*	.001866
New England	-0.0745	.0054	-0.1303	.0203	-0.1303	.0203	.013914	-.000522
Midwest	-0.2093*	.0222**	-0.2218*	.0267	-0.2218*	.0267	.026606	-.000252
West	-0.2519	.0212	-0.3326*	.0413	-0.3326*	.0413	.033756	-.000671
Urban	0.2696*	.0110	0.3155**	.0020	0.3155**	.0020	.01231	-.000077
Suburban	0.2429**	-.0034	0.2292*	.0016	0.2292*	.0016	.007926	-.000517
Intercept	-2.2963***	-.094***	-0.7159***	-.1189***	-0.4660	-.2031***	-.1978***	.004493
Threshold 2	2.6481***		2.6533		2.678503			
Threshold 3	5.5987***		5.6061		5.656836			

* $p < .05$. ** $p < .01$. *** $p < .001$.

those with higher education levels at age 51 were in better health (had fewer functional limitations) than those with low educational attainment. The second column in Model 1 shows how the effect of education changes as people age. Time was defined in the growth curve model as age. The positive coefficient means that the effect of education became more powerful as people aged. Those with higher educational levels had a slower rate of functional decline than those with lower educational levels.

Model 2 includes education, sex, race, and geographic covariates. The effect of education at the intercept level was diminished slightly but remained significant and positive. Women had more functional limitations than men, and Blacks had more functional limitations than Whites. A few of the regional variables, however, did not behave as expected. The inclusion of the urban, suburban, and rural variables, as well as the growth terms, led to findings at the intercept level that those in the South had better functional health relative to other parts of the country.

In column 2 of Model 2, the effect of education on functional decline continued to accelerate with age, though the addition of other controls reduced the coefficient size slightly. In regard to the other covariates, although women were initially in worse health than men, the rate of functional decline was actually faster for men as they age. The rate of functional decline for African Americans, in contrast, accelerated more quickly than for Whites.

Model 3 included all covariates and added a quadratic term to the growth rate to identify whether the relationship between age and functional health was linear or curvilinear. The education intercept term was significantly positive, indicating that at age 51, people with more years of education had lower rates of functional limitations. The interaction between years of education and age continued to be significant and positive, indicating that at least for a period, those at lower educational levels had a more rapid rate of decline than their higher educated counterparts. The third column shows that the coefficient for education interacted with the quadratic term (age^2) was significant but positive. These results indicate that though those with higher educational levels initially had slower rates of functional decline as they aged, the relationship reversed as people got older, indicating diminishing educational functional health disparities at older ages.

To give perspective on the magnitude of change, consider the difference in the log odds functional health scores between those without high school degrees and those with college degrees as people aged. Those differences expanded and reached peak at age 63. After that, the differences started to shrink but by no means disappear. By age 72, the difference in the log odds health scores had fallen from a peak of 2.36 to 1.74, which is the level it

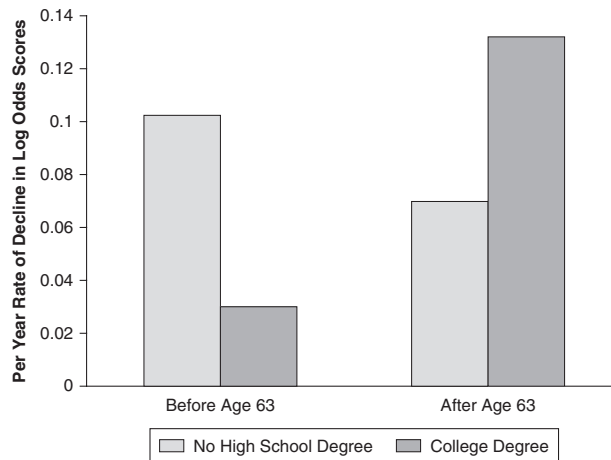
was at in the respondents' early 50s. Given that the maximum difference in scores was 3.51, a difference of 1.74 is still quite large.

Although the previous results revealed diminishing health disparities, how strongly were the results affected by the inclusion of those who died? That is, how much did mortality selection affect the results? The simplest way to cross-check this is to compare analyses that include and exclude the deceased. If the interaction between age and education shows substantial change with the inclusion and exclusion of individuals leaving the study because of mortality, I would find support for a meaningful impact of selection. If I were to find little difference in the estimates, this would lend support to the idea that mortality selection is not primarily driving declining health inequalities in late life.

Model 3 in Table 2 (on which the estimates of the previous figures are based) included those who died in the analyses and computed separate intercepts and slopes for all individuals. This allowed me to finish the thought exercise of what would have happened if they had not died. Model 4 in Table 2 then excluded those who were deceased to provide a comparison to the results in Model 3, which included the deceased. In general, these results showed little support for a large impact of mortality selection. The interaction terms are most critical in understanding how mortality affects age trends in functional decline by educational attainment. When comparing Models 3 and 4, the coefficient for education interacted with age was 3% larger in the sample that excluded people who died (Model 4), indicating little bias. There was no difference in the significance level. And the coefficient size for education interacted with age squared, which demonstrates the curvilinear relationship between age and education, was increased by just 1% in Model 4 compared with Model 3. Although there are some differences, the magnitude of the effect is small.

Why do educational health disparities diminish? Do the health trajectories change for those with low educational attainment or high educational attainment? Figure 1 shows how the rate of decline changed for those without high school degrees and those with college degrees before and after the peak age of health score differences (age 63). Before age 63, respondents without high school degrees had an annual average rate of health decline that was triple that of those with college degrees. But after age 63, the rate of health decline for those without high school degrees slowed. This supports the hypothesis that decreasing health inequalities in old age are due to enhanced social supports, such as Social Security and Medicare, which would primarily affect the health of those with limited education. If mortality selection is not properly addressed, however, this finding could also reflect a more robust sample of low-educated survivors.

Figure 1
Rate of Functional Decline (log odds scores) Among
Those Without High School Degrees and Those
With College Degrees, Before and After Age 63,
in the Health and Retirement Study



In contrast to those without high school degrees, respondents with college degrees had a rapidly accelerated rate of functional decline after age 63, which supports biological explanations for declining health inequalities in old age. Specifically, it supports the hypothesis that those with high educational attainment are able to delay the onset of poor health until later ages than those with low educational attainment. This accelerated decline, which helped produce diminishing educational health disparities, cannot be explained by mortality selection.

These additional findings did vary a bit depending on the imputation strategy. But two key aspects remained consistent. After the age of peak difference (which did vary depending on the imputation strategy) between those with low and high educational attainment, the rate of decline for those with high educational attainment accelerated rapidly, whereas the rate of health decline for those with low educational attainment never exceeded the rate prior to that peak age of inequality.

Conclusion

The results of this study provide support for the age-as-leveler hypothesis with regard to how education and functional health interact across the life course. Educational functional health disparities consistently widen until age 63, after which they start to diminish. By age 72, they return to the average level of those at age 53. Nonetheless, the differences remain quite large given that these differences start to expand in early adulthood and peak around age 60. Furthermore, these analyses can follow the respondents only into their early 70s. It is possible that as individuals continue to age and their health worsens, health inequalities could begin to accelerate again. Future analyses of the HRS in the coming years will be able to document whether health inequalities continue to diminish or whether they reverse themselves into individuals' mid- to late 70s and 80s.

This is the first study that examined how educational health disparities change as people age by following a single cohort over time. By looking at a single cohort, I am not concerned that diminishing health inequalities are due to larger disparities in health by educational level among younger cohorts relative to older cohorts. It may be the case, however, that other cohorts may age differently. Thus, it is possible that this is a period effect. I argue, however, that this is unlikely. Additional analyses of older cohorts in the National Health Interview Survey 1970 to 2000 (not included in this article) found a similar pattern to the 1931-1941 birth cohort.

The other oft proposed alternative explanation for diminishing health inequalities in late life is mortality selection. The longitudinal nature of the study design, however, provides the means to address it. These analyses provide no convincing evidence that mortality selection is primarily responsible for declining health inequalities in old age. It is the case, however, that only a longitudinal study following individuals from early adulthood through the next 80 years could rule out mortality selection with absolute certainty.

This study also provides some very preliminary evidence on why inequalities decline. The rate of health decline for those with college degrees started accelerating rapidly after age 63, providing support for biological hypotheses. Particularly, individuals with high educational attainment have the onset of functional decline at later ages than those with low educational attainment. In contrast, the rate of health decline for respondents without high school degrees slowed after age 63, providing support for a social welfare effect in old age. Again, these findings do not provide causal evidence, but they do provide support for both hypotheses.

The policy implications of this study are twofold. First, though health inequalities do shrink some in old age, they remain quite large. Thus, proposals to increase the age of eligibility for Social Security and Medicare would have very different implications for those with low educational attainment compared with high educational attainment. Although many have argued that improved health and capacity among the aged is a justification for this policy change, it is clear that many older Americans have relatively significant health problems that would limit their ability to be employed.

The second policy implication is the need for more research. Ultimately, the finding that health inequalities diminish in old age could provide an important clue about how health inequalities can be offset. Of particular interest is the extent to which societal explanations, specifically Social Security, retirement, and Medicare, may affect health. People at the bottom of the SES ladder have the most health problems and are most sensitive to changes in their environments and economic resources. Evidence that poverty and economic distress is quite low among the elderly relative to other age groups, largely as a result of a large welfare state, may partially explain why their rate of decline in functional health slows in their 60s (Mirowsky and Ross 1999). Furthermore, there is evidence that Medicare has a positive impact on older people's health, particularly for those who did not have health insurance previous to retirement (Dor et al. 2003). From childhood right up until early old age, socioeconomic differences in health grow. Thus, the finding that health inequalities diminish some in old age is very intriguing. Old age, and all that goes along with it, may provide clues that could help shrink health disparities at younger ages. Future research must tackle why health inequalities decline in late life.

Note

1. Sample attrition among survivors is less of a concern because individuals in the HRS are followed up if they enter institutions such as nursing homes. Thus, the probability is low that those survivors who left the study were not randomly missing. Random coefficient models are perfectly suited for this type of analysis.

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