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Understanding the SES Gradient in Health among the Elderly
The Role of Childhood Circumstances

Till Stowasser, Florian Heiss, Daniel McFadden, and Joachim Winter

5.1 Introduction

It is the health economics version of the classic "chicken and egg" problem: We know that people with high socioeconomic status (SES) tend to be in better health and live longer than their economically disadvantaged counterparts but we are not sure which came first. Do economic resources determine health (hypothesis A)? Does health influence economic success (hypothesis B)? Or, are both health and wealth dependent on some third unaccounted factor (hypothesis C)? The body of literature dealing with this so-called socioeconomic gradient in health is impressive (for overviews see Smith [1999]; Cutler, Lleras-Muney, and Vogl [2011]; and Stowasser et al. [2012]).

The traditional view that causality flows from SES to health is especially common among—but not exclusive to—epidemiologists. Often-cited causal pathways are the affordability of health services, better health knowledge and lifestyles among the higher educated, environmental hazards associated with poorly paying occupations and low-income living conditions, or

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the mere psychological burden that comes with a life of constant economic struggle. Economists were among the first to argue that causality may also work its way from health to economic outcomes, the most important channel being the development of human capital: physical frailty is likely to have adverse effects on educational attainment, occupational productivity, and, consequently, the accumulation of wealth. Finally, the statistical literature stresses the point that the persistent correlation between morbidity and SES may in fact be spurious and due to unobserved individual heterogeneity with a common influence on both health and wealth; see Heckman (1981b), inter alia. Prime candidates for such hidden third factors are genetic disposition and other family effects with an impact on preferences and health-relevant behaviors.

Discriminating among these rivaling hypotheses is important since policy recommendations will critically depend on the nature and the sources of the gradient. Methodologically, the estimation of credible causal effects in population data requires addressing the challenges of simultaneity (hypothesis A vs. hypothesis B) and unobserved common effects (hypotheses A/B vs. hypothesis C).[1] The conventional solution to both of these problems is to exploit natural experiments that provide instruments for either health or SES. While this strategy of isolating exogenous variation certainly works well on paper, it is not always persuasive in practice. The main caveat is that convincing instruments are generally in short supply. As discussed by Stowasser et al. (2012), even the availability of instruments that are clearly exogenous and that have an impact on the endogenous regressor they seek to replace may cause problems if the variation they reflect is not all that *relevant* for the dependent variable of interest. Moreover, since instrumental variable (IV) strategies usually rely on rather case-specific events, any uncovered effects may well be causal in nature but of questionable external validity; Deaton (2010) discusses these issues.

For these reasons, Adams et al. (2003) propose an alternative approach of uncovering causal links that makes use of the entire variation in health and economic variables. Using panel data, they test for Granger noncausality of SES for *innovations* in health, which deals with the econometric challenge of distinguishing hypotheses A and B.[2] Their purely statistical causality concept deviates from "true" causality in a structural sense, as their approach does not specifically address the issue of unobserved individual heterogeneity. As a consequence, the detection of Granger causality would not necessarily imply the validity of hypothesis A, since unobserved third factors may be at work instead. However, a finding that economic status is *not* Granger causal for health and that the relationship is invariant across a

1. For a detailed discussion, see Stowasser et al. (2012).

2. While Adams et al. (2003) studied both wealth-to-health and health-to-wealth causation, this study concentrates on the question of whether hypothesis A is correct.

wide range of SES and health histories would be informative, as this would rule out true causality as well.[3] Applying their framework to a representative sample of US Americans over the age of seventy, Adams et al. (2003) are unable to reject the hypothesis that economic status has no causal effect on mortality and most health innovations, once health history is controlled for. Despite the fact that this result may not be overly surprising in light of the subgroup's quasi-universal access to Medicare and considering that causal links may well have been active in the past,[4] their study stimulated some controversy in the literature.

On this account, Stowasser et al. (2012) revisit the approach introduced by Adams et al. (2003) and investigate whether the original findings are confirmed when their methodology is applied to a more encompassing set of data that covers health histories of different lengths and varying age compositions. In stark contrast to the original study, they find that it is much harder to reject the existence—or the activity—of causal links in more comprehensive samples. Importantly, this result is not exclusively driven by the inclusion of younger individuals, as the mere growth in sample size already leads to higher rejection rates of Granger noncausality, which indicates that the original results were partly driven by low test power. In light of their findings, Stowasser et al. (2012) discuss three avenues for improving the approach suggested by Adams et al. (2003). First, the underlying notion of health dynamics, with health being modeled as a first-order Markov process, falls short of reflecting the stock characteristics of latent health capital as envisioned by Grossman (1972). Second, the original approach does not account for individual heterogeneity, which makes it impossible to distinguish between true causal links and third-factor effects in case Granger causality is detected. Third, even if common effects were convincingly controlled for, the tests proposed by Adams et al. (2003) are only informative about the mere *presence* of causality but not of the mechanisms through which SES influences health. Although knowledge of this general link is important in its own right, the identification of specific pathways is equally critical from a policy perspective.

The present study aims at addressing these issues and gauges whether the main conclusion of Stowasser et al. (2012), that it is impossible to statistically reject SES-to-health causality even in a retired population age sixty-five and older, is robust to these methodological refinements. The research strategy rests on the increasing availability of retrospective life-history data within large panel studies that link economic and health data, such as the

3. The rationale for this reasoning is that Granger causality—or conditional dependence across time—is thought of as a necessary but insufficient condition for causality in a more structural sense.

4. Indeed, Adams et al. (2003) find a steep gradient in the initial cross-section, suggesting that a great deal of the relationship between health and wealth has already been determined during the (unexplained) first seven decades of a respondent's life courses.

US Health and Retirement Study (HRS) used for this analysis.[5] These data innovations are the response to the rapidly growing literature on childhood health that makes the point that a meaningful analysis of the gradient should incorporate a respondent's early-life information (for an overview, see Smith [2009]; Almond and Currie [2011]; and Currie [2011]). For instance, Case, Lubotsky, and Paxson (2002) suggest that part of the adult SES gradient in health originates in early childhood, as they find a strong relationship between parental economic status and childhood health that accumulates as children age. In another cohort study, Case, Fertig, and Paxson (2005) document that these early conditions have a lasting impact on adult health and—in line with hypothesis B—other outcomes such as education, labor supply, and income. As Currie (2009) notes, these findings are supported by many, albeit not all, of the myriad of studies that complement the literature by exploiting data from natural experiments.

Not only does this evidence suggest the use of available information on childhood circumstances to avoid bias from omitted variables when studying causal pathways in adulthood—the retrospective look at the beginning of life additionally has the potential to alleviate all three of the aforementioned problems in the Adams et al. (2003) framework. First, it provides an opportunity to incorporate longer health histories and, thus, a more realistic model of health dynamics. Second, to the extent that retrospective data also covers information on family backgrounds and parental SES, it will be possible to proxy control for some of the individual heterogeneity that is suspect of exerting a common influence on health and wealth. Third, controlling for both historic and contemporary variables may elucidate *when* the association between SES and health is established, which has important policy implications: if future outcomes are predetermined during childhood, resources spent on policies that aim at improving access to health care for adults and retirees may in fact be more wisely invested into educative and financial measures for young families.

In summary, the results of this study suggest that the findings of Stowasser et al. (2012) are largely insensitive to varying models of health histories. While SES is unlikely Granger causal for innovations in acute health insults, Granger noncausality can be statistically rejected for mental health conditions, mortality, and changes in overall health. Evidence for chronic diseases and functional health is a bit more inconclusive. However, since the detection of Granger causality for these health conditions is adversely related to sample size, it is possible that we merely observe the statistical artifact—as already reported by Stowasser et al. (2012)—that test power suffers considerably in small data sets. The fact that results are also quite robust to the

5. Comparable data collection efforts targeted at the population age fifty and older include the Survey of Health, Ageing and Retirement in Europe (SHARE), the English Longitudinal Study of Aging (ELSA), or the China Health and Retirement Longitudinal Study (CHARLS).

introduction of proxy controls for individual heterogeneity lends support to a causal interpretation of the observed gradient. In line with the literature on early-life circumstances, we find that childhood health has lasting predictive power for adult health. This, however, does not render contemporary factors unimportant. Finally, we uncover strong gender differences in the intertemporal transmission of SES and health: while the link between SES and functional, as well as mental health among men is established rather late in life, the gradient among women appears to originate from childhood circumstances.

The rest of this chapter is structured as follows. Section 5.2 presents the data used for analysis. This is followed by a brief description of the methodological framework—which closely resembles that of Adams et al. (2003) and Stowasser et al. (2012)—in section 5.3. The empirical analysis is presented in section 5.4. Section 5.5 concludes.

5.2 Data

In this chapter, we use data from the Health and Retirement Study (HRS), which is a representative panel of the US population age fifty and older. The design of the analysis sample and the constructions of the variables are natural extensions of Adams et al. (2003) and Stowasser et al. (2012).[6] Due to substantial deviations in survey design, observations from the first panel wave are dropped. As a result, the main working sample consists of 8 biennial waves covering interviews conducted between 1993 and 2008. In the spirit of the original study by Adams et al. (2003), we restrict our analysis to a mostly retired population of the age of sixty-five and older. On average, each wave contains roughly 11,400 individuals with usable records on health outcomes, SES variables, and demographic information.[7] Attritors and members of refreshment cohorts are kept in the sample for as long as they participate in the survey. This ensures that sample size is kept high enough for precise estimation and that up to 8 waves can be used simultaneously.

This study differs from Stowasser et al. (2012) in that it no longer estimates the incidence of twenty separate health conditions but combines some of them into disease clusters. As a result, health dimensionality is reduced to just six outcomes, which considerably facilitates concise interpretability of results. We consider these outcomes: the number of acute—and immediately life-threatening—conditions (cancer, heart disease, and strokes); the number of chronic diseases (lung disease, diabetes, hypertension, and arthritis); the number of functional health limitations (incontinence, severe falls, hip frac-

6. For further details on HRS, you may refer to Stowasser et al. (2012).

7. Just as in Stowasser et al. (2012), we exclude individuals that generally failed to disclose information on their health. Gaps from insular item nonresponse are filled via simulation-based imputation. For missing wealth and income measures, we use imputations readily available in the public release files provided by the RAND Corporation.

tures, ADL/IADL impairments, and an indicator for obesity); the number of mental illnesses (cognitive impairment, psychiatric disease, depression, and whether interviews were conducted with a proxy respondent); self-rated health status; and mortality. Summary statistics for these health indicators as well as for all SES variables used for analysis—namely wealth, income, education, dwelling condition, and neighborhood safety—are presented in appendix table 5A.1.

This contemporary data is complemented with information from retrospective questionnaires on respondents' health, living conditions, and family backgrounds when they were children, that has subsequently become available within HRS. While this method of retrieving information about panel members' lives before the survey's baseline year provides advantages—in the form of low cost, speed, and reduced sample attrition—over longitudinal cohort studies that follow respondents from cradle to grave, one may express doubt about the accuracy of responses. After all, interviewees are asked to recall circumstances that date back at least fifty years.[8] Yet, the growing literature on the reliability of retrospective surveys finds recall bias to be generally negligible (see Berney and Blane 1997; Garrouste and Paccagnella 2010). For instance, while Smith (2009) reports some unsystematic recall error in retrospective HRS data, he finds no evidence for "coloring"—the selective recall of health histories induced by adverse health events late in life—of responses.

Retrospective information on childhood health has been introduced to HRS in two stages. A general index of self-rated health (SRH) before age sixteen—which is constructed in the same way as HRS's five-point scale measure for contemporary SRH—is already available since panel wave 4, hence covering a rather large share of the entire HRS population. On the other hand, effective sample sizes are considerably smaller for the multitude of detailed child-health measures introduced in wave 9, since these are only available for respondents who were still sample members at this late stage. The latter list of variables includes twenty-one health conditions and whether respondents missed school for more than a month due to health problems. Once again, the individual health conditions are grouped to reduce complexity. We distinguish severe health problems (such as cancer or heart disease), less severe conditions (such as ear infections or allergies), mental health problems (such as depression or psychological problems), and classic child diseases (measles, chicken pox, and mumps).

The HRS early-life data also covers the economic living conditions during childhood as well as family background measures and certain child behaviors. Again, some measures are available as early as wave 4. These include a three-point index of self-assessed family SES, information on parental education, paternal unemployment, and whether the family ever solicited

8. The HRS questionnaire defines childhood as life before the age of sixteen.

Table 5.1 HRS early life data, summary statistics

Variables	*N*	Mean	Std. Dev.
Childhood health			
Poor/fair self-rated health	25,266	0.065	0.247
No. of severe conditions	11,624	0.243	0.526
No. of less severe conditions	11,625	0.345	0.665
No. of mental conditions	11,693	0.068	0.289
No. of childhood diseases	10,565	2.228	0.982
Missed school due to health problem	11,681	0.113	0.316
Family background			
Self-rated family SES above average	25,389	0.066	0.249
Self-rated family SES below average	25,389	0.317	0.465
Family needed financial help	24,994	0.125	0.331
Moved due to financial problems	25,246	0.180	0.384
Father's education (in years)	24,806	8.9	3.5
Mother's education (in years)	26,010	9.1	3.3
Father ever unemployed	25,045	0.290	0.454
Mother always worked	17,633	0.171	0.376
Mother sometimes worked	17,633	0.327	0.469
Any parent smoked	11,677	0.634	0.482
Both parents smoked	11,677	0.169	0.375
Smoked as child	15,219	0.185	0.389
Drugs or alcohol as child	11,722	0.005	0.071
Learning problems at school	15,218	0.027	0.162
Father's age (at death) (in years)	29,482	71.6	14.4
Mother's age (at death) (in years)	29,482	75.3	15.1
Adult height (in meters)	29,482	1.69	0.10

Note: N denotes the number of respondents for whom information on the respective variable is available.

financial help or had to move due to economic dire straits. Information on maternal labor-force participation and parental smoking were added in waves 8 and 9, respectively. In addition, starting with wave 9, HRS provides information on childhood smoking, drug and alcohol use, and whether the respondent experienced significant learning problems at school. Another pair of measures—already used by Adams et al. (2003) and Stowasser et al. (2012)—that also capture family effects, but which are not considered part of HRS's retrospective module, are the ages at death (or just the ages, in case they are still alive) of the respondents' parents. Similarly, respondents' adult height is often used as a proxy for health at birth and is correlated with the uterine environment the family provides (see Case and Paxson 2008; and Currie 2011).

Summary statistics for all early-life data used for analysis are provided in table 5.1. As indicated, the number of available observations differs considerably among variables. This needs to be taken into account when deciding

which of these information to use for analysis in section 5.4, as statistical power will certainly suffer in case of severe sample-size loss.

5.3 Methodology

The econometric methods used in the present study are essentially those introduced by Adams et al. (2003), with some extensions introduced by Stowasser et al. (2012); we refer to the latter paper for a more detailed discussion. The analysis builds on a dynamic model of health incidence,

$$f(HI_{it}^{j} \mid \mathbf{HI}_{it}^{k<j}, \mathbf{H}_{it-\tau}, \mathbf{S}_{it-1}, \mathbf{X}_{it-1}, I_i), \tag{1}$$

where i denotes the respondent and t indicates time. The dependent variable, HI_{it}^{j} measures a new incidence of a given health condition, where j stands for one of the six health clusters previously introduced. As in Adams et al. (2003), health innovations are thought to be influenced by the following explanatory variables: Instantaneous causal effects from concurrent health shocks on HI_{it}^{j}—such as the development of cancer that is followed by death within the same interwave spell—are captured by $\mathbf{HI}_{it}^{k<j}$, containing the incidence variables for all health indicators (1, . . . , k) that are causally arranged upstream of indicator j.[9] Furthermore, the model controls for health histories, $\mathbf{H}_{it-\tau}$, that capture state dependence and comorbidities, respectively. The vector $\mathbf{X}_{it-1}$ includes demographic controls. The vector of main interest, $\mathbf{S}_{it-1}$, contains lagged levels of wealth, income, educational attainment, and indicators for subpar living environments. If SES is truly causal for health changes in an elderly population, we should expect significant coefficients for at least some of these variables. Moreover, the null hypothesis that

$$f(HI_{it}^{j} \mid \mathbf{HI}_{it}^{k<j}, \mathbf{H}_{it-\tau}, \mathbf{S}_{it-1}, \mathbf{X}_{it-1}, I_i) = f(HI_{it}^{j} \mid \mathbf{HI}_{it}^{k<j}, \mathbf{H}_{it-\tau}, \mathbf{X}_{it-1}, I_i), \tag{2}$$

that is, that past SES is not Granger causal for health deteriorations, should be rejected, while invariance tests, as described in Adams et al. (2003), are expected to be confirmed.

Model 1 deviates from the original specification of Adams et al. (2003) in three dimensions. First, health histories are no longer assumed to be first-order Markov, as τ may take on values larger than one, to better accommodate the stock characteristics of latent health capital. This part of the analysis, in which we estimate model 1 with alternative specifications for $\mathbf{H}_{it-\tau}$, is presented in section 5.4.1. Second, the model acknowledges

9. Similarly to Adams et al. (2003), the six health indicators are grouped in the order in which instantaneous causality is most likely to flow: acute conditions are listed first, as they can have an immediate impact on mortality. The remaining indicators are stacked as follows: acute conditions upstream of chronic conditions, upstream of functional conditions, upstream of mental conditions, and upstream of SRH.

Table 5.2 **Benchmark results, tests for Granger noncausality**

Health indicator	Test results (65+) W2–9 (N = 50,993) F	M
Acute conditions		
Mortality	○	●●
Chronic conditions	●●●	●●●
Functional conditions	○○	●●●
Mental conditions	●●●	○○○
Self-rated health status	○○○	○○○

Notes: Results are for white females (F) and males (M). Abbreviations are as follows: Granger noncausality rejected at 5 percent level (●), rejected at 1 percent level (●●), or rejected at 0.1 percent level (●●●). Empty symbols indicate that the corresponding invariance test is rejected at the 5 percent level. Blank cells indicate that Granger noncausality cannot be rejected. N denotes the number of respondent-year observations.

the hypothetical presence of individual heterogeneity, I_i, that may induce spurious correlation between health and SES (see hypothesis C). The analysis in table 5.2 seeks to contain the confounding influence of such common effects by using proxy controls for family backgrounds and behavioral factors. Of main interest is whether the finding of Stowasser et al. (2012), that SES is Granger causal for innovations in health even in an elderly population, survives when more realistic health dynamics and a richer set of control variables are incorporated. A confirmation of their results would lend support to a causal interpretation of the observed association.

The final deviation from the original model proposed by Adams et al. (2003) concerns the reduction in health dimensionality by grouping certain medical conditions together. As a consequence, model 1 is fitted by ordered probit (except for mortality and the indicator for poor/fair SRH, which continue to be estimated with a probit model). To ensure the results are not driven by this modeling choice, and to provide a benchmark to which results from section 5.4 can be directly compared, we estimate model 1 with identical health histories and controls as in Adams et al. (2003). Evidently, results are largely insensitive to the aggregation of health measures and mirror the finding of Stowasser et al. (2012) that—with the exception of acute diseases—SES Granger causality cannot be rejected for medical events after the age of sixty-five. These results are at least significant at the 5 percent level, in many cases even at the 1 percent or 0.1 percent level, although model invariance across time is not always supported in a sample that spans over all 8 available panel waves.

5.4 Empirical Analysis

5.4.1 Health Dynamics

The notion of health being a latent capital stock that reflects the entire history of medically relevant events is not new. Ever since Grossman (1972) proposed his seminal health production framework, most health economists acknowledge the existence of "long memory effects" of the human body and mind. Heiss (2011) confirms that this feature characterizes the HRS population, too, as he detects a surprisingly high degree of state dependence in respondents' SRH. Studying the first seven panel waves, he finds that, even if the maximum number of six lags of SRH are included to predict SRH in the seventh wave, all historic variables have significant explanatory power on their own.

In light of this, modeling health dynamics as a first-order Markov chain is unlikely to provide an appropriate description of the evolution of health, as discussed by Stowasser et al. (2012, 494):

> Intuitively, this is because the Markov model assumes that all relevant information about the whole past is captured in the observed variables one period ago. This is unrealistic since knowledge of longer histories would better capture the stock characteristics of health capital [. . .]. Taking functional limitations as an example, a respondent who reported difficulties with walking one year ago and no limitations previously has a different outlook than a respondent who consistently reported difficulties with walking for the last ten years.

A straightforward way to improve the original Adams et al. (2003) model of health dynamics consists of increasing the length of health histories that model 1 controls for. While the performance of higher-order Markov models probably falls short of that of a fully fledged *hidden* Markov model, such as Heiss (2011), they will likely pick up many of the same effects. More importantly, however, there are practical limits to this strategy: the more lags of health conditions that are incorporated, the smaller the effective sample size that remains for analysis. On the one hand, it excludes all respondents that have been part of the sample for fewer waves than required by the desired history length. This may affect both sample attritors and members of refreshment cohorts, meant to keep the panel representative of the underlying population. On the other hand, the sample would even shrink if the panel was completely balanced, as each additional lag of control variables requires to drop one wave for the estimation of health innovations conditional on health histories.

As discussed by Stowasser et al. (2012), such large drops in sample size constitute a problem for the Adams et al. (2003) approach because it will be unable to reject Granger noncausality if test power becomes too small as

the sample gets smaller. Given this apparent trade-off between richer health dynamics and the risk to obtain artifactual test results, the number of lags should only be increased with great care. On this account, the knowledge of health during childhood provides a promising alternative to control for even (much) longer histories without having to forego the potential scale limits in the data.

At the same time, the availability of information on child health alleviates the closely related problem of initial conditions—that is, life before respondents enter the panel (see Heckman 1981a). As Smith (2009, 388) notes,

> [k]nowing health or economic status beginning at [survey] baseline is not sufficient because the entire prior histories of health and economic trajectories may matter for current decision making. The absence of information on prebaseline health histories, including childhood health, means that researchers have to rely on a key untestable assumption: baseline health conditions sufficiently summarize individuals' health histories. If they do not, new health events unfolding during the panel may be the delayed (and perhaps predictable) consequence of some knowable part of an individual's health history. If so, health events within the panel cannot be used to measure effects of new exogenous, unanticipated events.

The extent to which retrospective data enables a look into the "black box" of early life, as compared to Adams et al. (2003) and Stowasser et al. (2012), is visualized in figure 5.1. Note that the effective health-history length is depicted to be by one wave (or two years) shorter than panel length theoretically permits.

Given these considerations, we gauge the sensitivity of model 1 to varying representations of health history by gradually increasing the lag length of adult health prevalence, by the inclusion of child health, and by combinations of the two. As argued earlier, these steps are associated with considerable reductions in effective sample size, which entails the risk of confounding any effect from longer health histories with the mere decline in test power. In order to separate these two effects, we also apply the original health-history specification of Adams et al. (2003) to these subsamples. These "dry runs" serve as the benchmarks to which results from models with more sophisticated health histories should be compared. The Granger noncausality test results for all of these specification are summarized in table 5.3.

The first alternative specification models health histories as a second-order Markov process (i.e., the number of health condition lags is increased to two), which reduces the size of the analyzable sample from 50,993 to 42,367 respondent-year observations. As is evident from comparing columns (C) and (D) with columns (A) and (B) of panel A in table 5.3, this has no significant impact on SES Granger causality tests. The same picture emerges when a third-order Markov model is used (see columns [G] and [H]). While with the latter specification, empirical *p*-values tend to be a bit higher than

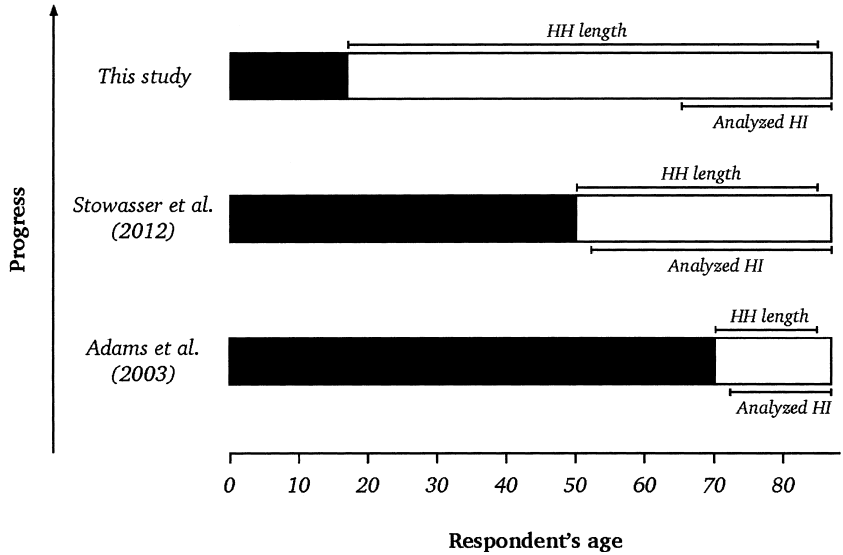

Fig. 5.1 Maximum health-history lengths, comparison between studies

Notes: White boxes indicate known health histories. Black boxes depict unknown health histories. "HH length" denotes the maximum length of health histories that can be exploited for analysis. "Analyzed HI" stands for the age range used to analyze health incidence.

with the lower-ordered Markov model (as indicated by fewer dots), this is clearly not driven by the inclusion of the additional lag but by the reduction in sample size. To see this, consider that *p*-values also increase for the benchmark case—compare columns (E) and (F) with columns (A) and (B)—whereas the actual switch to a higher-order Markov model—compare columns (G) and (H) to columns (E) and (F)—has no systematic impact at all. Results for even higher-order Markov models are not presented here, as these imply sample sizes too low to conduct meaningful analysis that stratifies by gender.

Panel B of table 5.3 contains results for specifications that use child health to incorporate longer health histories. Recall from section 5.2 that the number of respondents with data on childhood SRH greatly exceeds that of individuals for who we have detailed information on early-life health conditions. For this reason, we add these variables in two sequential steps. Results in columns (L) and (M) are for model 1 when controlling for first-order Markov health histories—the default in Adams et al. (2003)—and self-rated health during childhood. Once again, Granger noncausality tests are not systematically influenced by the incorporation of longer health histories and suggest that, with the exception of acute diseases, causal links from SES to health cannot be statistically rejected. In the second step, we additionally include the more specific data on childhood health conditions, which roughly cuts

Table 5.3: Results for varying health histories, tests for Granger noncausality

A. Higher-order Markov models

	Test results							
	Sample for 2nd-order Markov (N = 42,367)				Sample for 3rd-order Markov (N = 38,886)			
	Dry run		M2		Dry run		M3	
Health indicator	F (A)	M (B)	F (C)	M (D)	F (E)	M (F)	F (G)	M (H)
Acute conditions								
Mortality	●●●	●●●	●●	●●●	●●	●●	●●	●●
Chronic conditions	●●●	●●	●●●	●●	●●●	●	●●	●
Functional conditions	○○	●●●	○○○	●●●	○○	●●●	○○○	●●●
Mental conditions	●●●	○○○	●●●	○○○	●●●	○○○	●●●	○○○
Self-rated health	○○○	○○○	○○○	○○○	○○○	○○○	●●●	○○○

B. Childhood health

	Test results							
	Sample for SRH (N = 49,962)				Sample for conditions (N = 25,175)			
	Dry run		SRH		Dry run		HC	
Health indicator	F (J)	M (K)	F (L)	M (M)	F (N)	M (O)	F (P)	M (Q)
Acute conditions								
Mortality	●●	●●●	●●●	●●●	n.a.	n.a.	n.a.	n.a.
Chronic conditions	●●●	●●●	●●●	●●●	●●	●●●	●●●	●●●
Functional conditions	○○	●●●	○○	●●●		●●		●
Mental conditions	●●●	○○○	●●●		●●●	○○○	●●●	●●●
Self-rated health	○○○	○○○	○○○	○○○	○○○	●●●	●●●	●●●

C. Third-order Markov model and childhood health

	Test results							
	Sample for SRH (N = 34,136)				Sample for conditions (N = 19,527)			
	Dry run		M3 & SRH		Dry run		M3 & HC	
Health indicator	F (R)	M (S)	F (T)	M (U)	F (V)	M (W)	F (X)	M (Y)
Acute conditions								
Mortality	●●	●●	●●	●●	n.a.	n.a.	n.a.	n.a.
Chronic conditions	●●●	●	●	●	●	●		●
Functional conditions	○○	●●●	○○○	●●●		●●●		●●

(*continued*)

Table 5.3: (continued)

	Test results							
	Sample for SRH (N = 34,136)				Sample for conditions (N = 19,527)			
	Dry run		M3 & SRH		Dry run		M3 & HC	
Health indicator	F (R)	M (S)	F (T)	M (U)	F (V)	M (W)	F (X)	M (Y)
Mental conditions	●●●	○○○	●●●	○○○	●●●	○○○	●●●	○○○
Self-rated health	○○○	○○○	●●●	○○○	●●●	●●●	●●●	●●●

Notes: Results are for white females (F) and males (M). Abbreviations are as follows: Granger noncausality rejected at 5 percent level (●), rejected at 1 percent level (●●), or rejected at 0.1 percent level (●●●). Empty symbols indicate that the corresponding invariance test is rejected at the 5 percent level. Blank cells indicate that Granger noncausality cannot be rejected. N denotes the number of respondent-year observations. SRH stands for self-rated health during childhood. HC denotes childhood health conditions. "Dry run" stands for dry runs that use the original specification by Adams et al. (2003). M2 and M3 abbreviate second-order and third-order Markov processes, respectively. That lack in variation impedes estimation of mortality models is indicated by "n.a."

the available sample size in half (49,962 to 25,175 respondent-year observations). The corresponding results in columns (P) and (Q) require some discussion. First of all, the effect of SES on mortality can no longer be studied because information on childhood health conditions is only available for respondents who were still alive in wave 9, which happens to be the most recent wave in the working sample. Furthermore, while it is true that Granger causality of SES is no longer supported for functional health conditions among women, this seems, once again, to be driven by the substantial reduction in sample size. Also note that, while the change in results for functional conditions among men (when comparing columns [O] and [Q]) seems substantial at first sight, a look at the actual p-values reveals that the change—from 0.0089 to 0.0104—is only marginal at best.

For results in panel C of table 5.3, we combine both ways of accommodating health histories, which should arguably provide the most comprehensive description of the long memory effects of latent health capital—although this comes at the cost of even greater sample-size loss. Test outcomes in columns (T) and (U) are from a model with third-order Markov health histories and childhood SRH. This specification is then amended with the data on childhood health conditions (see columns [X] and [Y]). Overall, test outcomes depicted here corroborate the findings from panels A and B. If anything, evidence for SES being Granger causal for the development of chronic conditions becomes a little weaker, as the null hypothesis of noncausality is only rejected at the 5 percent level for men and the 5 percent to 15 percent level for women (the corresponding p-value in column [X] equals 0.141). Similarly, results for functional conditions among females do again

become barely insignificant ($p = 0.120$). While it is certainly possible to dismiss these observations as artifactual side effects of dwindling sample sizes, one should at least note that results are generally less stable for chronic and functional conditions than for mental health and SRH.

Finally, a look at the coefficients of the underlying prediction model 1—not reported here due to space limitations—confirms the earlier finding in the literature that even very long health histories have explanatory power for health innovations in an elderly population. For instance, Heiss's (2011) observation, that all lags of SRH have highly significant predictive power for current SRH, is confirmed even when controlling for SES and third-order Markov health-condition histories. The fact that the same holds true for SRH during childhood hints at an astounding degree of state dependence in latent health and confirms the long reach of childhood circumstances, established by the literature summarized in section 5.1. It is encouraging to observe that test results for Granger noncausality of SES are not significantly changed by accounting for these formerly omitted variables.

5.4.2 Common Effects

As argued earlier, the Granger causality framework proposed by Adams et al. (2003) cannot cleanly distinguish between hypotheses A and C—that is, between "true" causality and spurious correlation due to common effects. This identification problem arises because of unobserved individual heterogeneity—with respect to genetic endowment, family backgrounds, and early-life experiences—that influences both health and SES without there necessarily being a causal relationship between the two. Methodological solutions to this problem either require a set of valid instruments or the use of fixed-effects approaches. Since Adams et al. (2003), Stowasser et al. (2012), and the present chapter study whether the framework proposed by Adams et al. (2003) can serve as a viable *alternative* to IV estimation, it would not make much sense to go down the first mentioned route. Furthermore, while the HRS panel is certainly of sufficient length to estimate equations with individual fixed effects, it is not obvious that such models, which rely on the assumption that coefficients are constant over time, make sense when looking at health and wealth over a period spanning several decades.

For these reasons, this study follows a different strategy, which may well fall short of providing an outright solution to the problem, but should alleviate the confounding influence of unobserved third factors. Acknowledging the fact that the underlying problem is one of omitted variables—namely unobserved individual heterogeneity—we add control variables that should provide reasonable proxies for characteristics of the family and the home environment, as the latter are likely to play a central role in shaping individual preferences, behaviors, and genetic endowment. Naturally, the feasibility of this approach critically hinges on the data at hand. As extensively argued in the childhood-health literature, early-life data provides a number

of variables that meet the aforementioned requirement (see—among several others—Case, Lubotsky, and Paxson [2002]; Berger, Paxson, and Waldfogel [2009]; Case, Fertig, and Paxson [2005]; Smith et al. [2010]; Mazzonna 2011; and Kesternich et al. [2012]). For instance, Case, Fertig, and Paxson (2005, 384):

> [. . .] include a large set of variables in [the control vector] C, and assume that this set of variables is rich enough to capture all individual heterogeneity. Indeed, our ability to control for a large set of childhood characteristics is an advantage over much of the previous literature that examines health and SES dynamics.

The fifteen family-background variables used to proxy control for individual effects are listed in table 5.1. As was the case for childhood health conditions, the number of available observations differs substantially among variables, which is why they are also added in two sequential steps. The first tier of controls includes the four proxies for family SES, parental education, paternal unemployment status, parental age (of death), and respondents' adult height. The second tier consists of the aforementioned data to which maternal labor-force status, parental and own smoking behavior as a child, drug use, and information on learning problems in school are added. Again, we estimate benchmark dry runs like those described in section 5.4.2 to distinguish the effects of adding the controls from those that are due to reductions in sample size. Results for Granger noncausality tests, conditional on model invariance, are summarized in table 5.4.

While *p*-values slightly increase across the board by the inclusion of both tier 1 and tier 2 variables, the changes in test results are not very substantial. Overall, the conclusion that Granger noncausality is statistically rejected for nonacute health events remains intact even after controlling for family backgrounds. The notable exception is functional health, for which results are a bit inconclusive. This underscores the earlier finding that the association between SES and this health dimension appears to be weaker than for other conditions.

In a final step, we estimate a version of model 1 that combines controls for family backgrounds with a more adequate model of health dynamics as developed in section 5.4.1. Note that, inasmuch as these longer histories capture the effect of latent health capital, they may also absorb some of the endogeneity imposed by genetic traits, with severe health problems in childhood being a signal for general frailty. To achieve the most conservative assessment for the presence of Granger causality, we model health histories as third-order Markov with controls for all available childhood health conditions and include the more encompassing second tier of early-life controls. Results are presented in table 5.5 and should be compared to columns (X) and (Y) of table 5.3 and columns (G) and (H) of table 5.4. Even in this most encompassing specification—that comes at the cost of an even smaller and

Table 5.4 **Results for varying family background controls, tests for Granger noncausality**

	Test results							
	Sample for tier 1 (N = 42,271)				Sample for tier 2 (N = 21,250)			
	Dry run		Tier 1		Dry run		Tier 2	
Health indicator	F (A)	M (B)	F (C)	M (D)	F (E)	M (F)	F (G)	M (H)
Acute conditions								
Mortality	●●	●●●	●●	●●	n.a.	n.a.	n.a.	n.a.
Chronic conditions	●●●	●●	●●●	●●	●●●	●●●	●●	●●
Functional conditions	○○	●●●	○	●●		●●		●
Mental conditions	●●●	○○○	●●●	●●●	●●●	○○○	●●●	○○○
Self-rated health	○○○	○○○	○○○	○○○	●●●	●●●	●●●	●●●

Notes: Results are for white females (F) and males (M). Abbreviations are as follows: Granger noncausality rejected at 5 percent level (●), rejected at 1 percent level (●●), or rejected at 0.1 percent level (●●●). Empty symbols indicate that the corresponding invariance test is rejected at the 5 percent level. Blank cells indicate that Granger noncausality cannot be rejected. N denotes the number of respondent-year observations. "Dry run" stands for dry runs that use the original specification by Adams et al. (2003). For definitions of tier 1 and tier 2, see text. That lack in variation impedes estimation of mortality models is indicated by "n.a."

less representative sample of just 16,335 respondent-year observations—SES Granger noncausality for mental health conditions and general health status is clearly rejected, which lends credibility to the interpretation that these associations do in fact reflect causal relationships. While results for chronic and functional health conditions are certainly less robust, it is not entirely clear how much of the increase in p-values is driven by the introduction of controls—which would in fact suggest the importance of third factors—and how much is due to dwindling test power that may occult the presence of true, albeit relatively weaker, causal links. A conclusive answer to this question will have to wait for the addition of refreshment cohorts, which will eventually increase the number of available observations for early-life conditions as well.

5.4.3 Pathways between SES and Health

So far, the focus of this study has been the ability of the approach introduced by Adams et al. (2003) to discriminate between true causality and the influence of third factors in case Granger causality is detected. While this general distinction is certainly of interest in its own right, it is equally important to go beyond broad causality tests and investigate more narrowly focused questions about the mechanisms that connect specific health outcomes to specific dimensions in SES. For this reason, we complete our analysis by discussing some key parameter estimates from the underlying

Table 5.5 **Results for all controls, tests for Granger non-causality**

	Test results (N = 16,335)			
	Dry run		Tier 1	
Health indicator	F (A)	M (B)	F (C)	M (D)
Acute conditions				
Mortality	n.a.	n.a.	n.a.	n.a.
Chronic conditions	●	●		
Functional conditions		●●		●
Mental conditions	●●●	○○○	●●	●●●
Self-rated health	●●●	●●●	●●●	●●●

Notes: Results are for white females (F) and males (M). Abbreviations are as follows: Granger noncausality rejected at 5 percent level (●), rejected at 1 percent level (●●), or rejected at 0.1 percent level (●●●). Empty symbols indicate that the corresponding invariance test is rejected at the 5 percent level. Blank cells indicate that Granger noncausality cannot be rejected. N denotes the number of respondent-year observations. "Dry run" stands for dry runs that use the original specification by Adams et al. (2003). For definition of tier 1, see text. That lack in variation impedes estimation of mortality models is indicated by "n.a."

prediction model, displayed in appendix tables 5A.2, 5A.3, 5A.4, and 5A.5, as they will shed light on the question of how and when links between SES and health are established.[10]

Acute Health Conditions

Results in table 5A.2 confirm our previous observation that adult SES is unlikely to be causal for the development of acute health conditions. In fact, in all of the specifications tested, there is not a single SES marker with a statistically significant impact on this health dimension. Reaffirmingly, estimates in columns (E) and (F) show that the same holds true for family SES during childhood, which is practically unrelated with the occurrence of acute health events in a population age sixty-five and older.

However, childhood health appears to have predictive power for adverse health shocks among retirees: results in columns (C) through (F) show that the number of diseases during childhood matters for women, whereas self-rated childhood health appears to be a sufficient statistic for male respondents. At the same time, the explanatory power of adult health histories is rather low, with first-order Markov processes representing an adequate

10. Note that, as expected, the number of classic child diseases has no explanatory power for any future health outcomes and are therefore excluded from regression tables 5A.2, 5A.3, 5A.4, and 5A.5.

modeling choice for both disease state dependence and comorbidities. While all higher-order lags—whose parameter estimates are not displayed due to space considerations—enter the model with intuitive signs, their effects are not statistically different from zero.

Chronic Health Conditions

As results in table 5A.3 show, evidence for chronic health conditions is less clear-cut. When childhood circumstances are ignored, wealth, income, and education are negatively related with the development of diseases such as diabetes or emphysema. This gradient gets considerably weaker—but does not fully disappear—when controlling for health and family background during early life (see columns [C] through [E]). Recall that these changes may partly be due to dwindling sample sizes that reduce test power, since point estimates for income among women, wealth among men, and college education among men remain rather constant whereas standard errors increase substantially. There is no evidence that the link between SES and chronic diseases is established during childhood, as none of the family background measures exerts any significant influence on adult health outcomes.

Yet, as was the case for acute illnesses, the development of chronic diseases appears to be partly predetermined by childhood health. Having experienced severe health spells before the age of sixteen significantly increases the likelihood of chronic morbidity. For men, the same is true for the number of less severe conditions. This evidence for strong intertemporal dependency is corroborated by estimates—which are again omitted to save space—of adult health histories that endorse a third-order Markov specification to model the evolution of chronic health conditions.

Functional Health Conditions

Mirroring the preceding analysis in sections 5.4.1 and 5.4.2, the strong link between *adult* SES and functional health detected in columns (A) and (B) of table 5A.4 is substantially weakened—and all but disappears for women—when early-life circumstances are added to the analysis. However, this should not automatically be taken as evidence against the general causality of SES for functional impairments. In fact, results in column (E) suggest that the SES gradient does survive even for women, but that it is already established during childhood: having grown up in a family with low SES and having been raised by guardians that smoked significantly impairs functional health for female retirees. Given the substantially higher labor market participation among men, it is not surprising that their link between SES and functional health seems to work through higher education, rendering family effects insignificant in column (F).

The long reach of early life is, once again, underlined by the fact that childhood health also affects functional well-being at higher ages. For women it is the number of mental health problems that matters, whereas men are sensi-

tive to the number of less severe illnesses when growing up. With respect to adult health histories, third-order Markov processes fare much better than short-memory models. This is especially true for chronic comorbidities and indicators for subpar self-rated health, whose lagged values—not displayed here—all enter with significantly positive signs.

Mental Health Conditions

Finally, the nature of the SES gradient in mental health—under inspection in table 5A.5—closely resembles that of functional impairments. Again, the link appears to be established during childhood for women and later in life for men. Female retirees with mental health problems report that they suffered from learning difficulties, that they smoked as a child, and that their family had to change homes due to financial impasse.[11] In addition, mental health as a child is by far the strongest predictor for psychological and cognitive problems among elderly women. By contrast, childhood circumstances are far less consequential for men, whose mental well-being is primarily influenced by years of schooling and current financial wealth.

As was the case for chronic and for functional health conditions, the evolution of mental health is well described by third-order Markov models whose explanatory power clearly exceeds that of lower-order processes not reported here.

5. 5 Conclusion

This study addresses three critiques of the methodology for studying causality in the health-wealth nexus that was introduced by Adams et al. (2003). Building on Stowasser et al. (2012), we exploit the availability of retrospective data on early-life events, which allows for improved control of initial conditions and individual heterogeneity.

The first issue we address is the model of health dynamics. We implement higher-order Markov models and control for information on childhood health to accommodate the long memory effects of latent health capital. In line with the literature on early-life circumstances, we find that childhood health has lasting predictive power for adult health. This, however, does not render contemporary factors unimportant. Our analysis also suggests that—with the sole exception of acute health conditions—third-order Markov processes are a better description of health evolutions than shorter-memory models. At the same time, causality tests are largely insensitive to varying models of health histories.

Furthermore, we confirm the findings by Stowasser et al. (2012) that SES is unlikely to be causal for the development of acute health conditions but that Granger noncausality can—even in an elderly population age sixty-five

11. Note that the two latter indicators are only marginally significant at the 10 percent level.

and older—be statistically rejected for mental health conditions, mortality, and changes in overall health. Evidence for chronic diseases and functional health is somewhat inconclusive. This may simply reflect the problem that Granger causality tests require relatively large sample sizes to obtain adequate power, as discussed by Stowasser et al. (2012).

The second methodological issue is the inability to distinguish between true causal links and common effects in case Granger causality is detected. The present study alleviates this concern by conditioning on early-life events that may function as proxies for unobserved individual heterogeneity, with health problems in childhood being a signal for physical frailty, and parental SES and health-relevant behaviors capturing family effects. Results from this modification closely mirror those of accounting for longer health histories. The fact that results for mental health and overall health status are remarkably robust, lends support to a causal interpretation of the observed gradient for these health dimensions.

Ultimately, however, the assessment of this issue will depend on how narrowly one wishes to define "true" causality. In our opinion, it is fair to argue that SES may even have a causal effect—in a rather wide sense—on individual heterogeneity, rendering the distinction between hypotheses A and C almost arbitrary. In fact, there is increasing evidence that personal characteristics are not as immutable as was once believed. For instance, part of the literature on the education-health gradient argues that the years spent in education may not only change health-relevant knowledge, but also preferences, behaviors, and the way people think about their future (see Cutler and Lleras-Muney 2008). In a similar vein, Currie (2011) reports evidence that even the activation of genetic traits—once considered the holy grail of irrevocability—may depend on environmental factors as well.

Finally, we address a third critique of the Adams et al. (2003) approach, the lack of a microfoundation of the pathways between SES and health. We scrutinize the underlying prediction model, which reveals pronounced gender differences in the origin of the gradient. While the link between SES and chronic illness appears to be established rather late in life, the same cannot be said about functional and mental health conditions among female retirees. For them, low family SES and mental problems as a child are the most predictive markers for health deteriorations in late adulthood, hinting at an exceptionally high degree of intertemporal and perhaps even intergenerational transmission of health and SES. In contrast to this, the SES gradient in functional and mental health for men—whose past labor market participation is much higher than that of female HRS respondents—does not stem from childhood circumstances but is rather established during (secondary) education and adulthood.

Substantively, our findings add to the current debate about the role of early childhood circumstances for lifetime health. To the extent that future health outcomes are at least partly predetermined by childhood circum-

stances, public health policies should not neglect the importance of providing educative and financial support for young families. Our findings support the notion that social returns from such investments are likely to match those of measures that aim at altering the availability and use of health care in adulthood.

Appendix

Additional Tables

The following tables contain regression results from our underlying prediction model and summary statistics for the data set used in our analysis. Due to their large dimensions, they are each displayed on an individual page.

Table 5A.1 Contemporary health and SES variables used for analysis, summary statistics

	Wave 2 (N = 8,726)		Wave 3 (N = 9,258)		Wave 4 (N = 11,916)		Wave 5 (N = 11,953)		Wave 6 (N = 12,273)		Wave 7 (N = 12,153)		Wave 8 (N = *12,502)*		Wave 9 (N = 12,468)	
Variable	Mean	StDev.	Mean	StDev.	Mean	StDev.	Mean	StDev.	Mean	StDev.	Mean	StDev.	Mean	StDev.	Mean	StDev.
No. of acute conditions	0.541	0.685	0.659	0.743	0.643	0.743	0.658	0.746	0.671	0.753	0.698	0.767	0.710	0.776	0.721	0.779
No. of chronic conditions	1.004	0.880	1.086	0.905	1.089	0.908	1.121	0.913	1.173	0.921	1.229	0.926	1.285	0.929	1.341	0.938
No. of functional conditions	1.316	1.120	1.535	1.264	1.562	1.278	1.654	1.299	1.171	1.303	1.768	1.311	1.851	1.316	1.905	1.308
No. of mental conditions	0.587	0.874	0.635	0.900	0.601	0.897	0.628	0.921	0.642	0.924	0.619	0.909	0.589	0.874	0.580	0.866
Poor/fair self-rated health	0.358	0.479	0.341	0.474	0.373	0.484	0.328	0.469	0.327	0.469	0.335	0.472	0.334	0.472	0.329	0.470
No. of acute conditions			0.244	0.491	0.277	0.522	0.219	0.471	0.236	0.486	0.219	0.471	0.218	0.467	0.221	0.475
Died since last wave			0.104	0.306	0.101	0.301	0.108	0.311	0.115	0.319	0.098	0.297	0.100	0.300	0.101	0.301
No. of chronic conditions			0.198	0.453	0.206	0.441	0.205	0.444	0.199	0.436	0.227	0.458	0.224	0.461	0.215	0.447
No. of functional conditions			0.843	1.072	0.874	1.077	0.798	1.018	0.830	1.038	0.836	1.042	0.931	1.068	0.908	1.040
No. of mental conditions			0.260	0.561	0.236	0.530	0.199	0.488	0.205	0.498	0.169	0.447	0.163	0.432	0.156	0.425
1st quartile wealth indicator	0.255	0.436	0.230	0.421	.227	0.419	0.219	0.413	0.214	0.410	0.215	0.411	0.217	0.412	0.212	0.409
4th quartile wealth indicator	0.183	0.387	0.221	0.415	0.255	0.436	0.275	0.447	0.291	0.454	0.306	0.461	0.330	0.470	0.328	0.470
1st quartile income indicator	0.368	0.482	0.335	0.472	0.291	0.454	0.292	0.455	0.277	0.447	0.278	0.448	0.272	0.445	0.274	0.446
4th quartile income indicator	0.117	0.321	0.144	0.351	0.161	0.368	0.167	0.373	0.169	0.375	0.176	0.381	0.177	0.382	0.193	0.394
Poor/fair housing condition	0.133	0.340	0.128	0.334	0.114	0.318	0.106	0.308	0.097	0.296	0.116	0.320	0.108	0.310	0.104	0.305
Poor/fair neighborhood safety	0.145	0.325	0.132	0.338	0.101	0.302	0.089	0.284	0.075	0.263	0.086	0.281	0.096	0.294	0.094	0.291
High school (educ. > 10 y.)	0.613	0.487	0.629	0.483	0.672	0.469	0.692	0.462	0.716	0.451	0.735	0.441	0.754	0.431	0.766	0.423
College (educ. > 14 y.)	0.147	0.354	0.156	0.363	0.172	0.378	0.180	0.385	0.193	0.195	0.199	0.399	0.206	0.404	0.218	0.413

Note: Summary statistics are for the age-eligible sample (65+).

Table 5A.2 Prediction model for acute health conditions[a]

	Ordered probit regression coefficients *z*-statistics in parentheses					
	Dry run		Child health		Family	
Key explanatory variables	F (A)	M (B)	F (C)	M (D)	F (E)	M (F)
Current SES						
Wealth (1st qtl.)	0.023	0.031	0.000	0.005	0.012	0.008
	(1.02)	(1.19)	(0.08)	(1.03)	(0.24)	(1.40)
Wealth (4th qtl.)	–0.025	–0.044	0.012	–0.015	0.012	0.001
	(–1.15)	(–1.89)	(0.32)	(–0.37)	(0.29)	(0.11)
Income (1st qtl.)	–0.037	–0.044	–0.044	0.059	–0.026	0.018
	(–1.75)	(–1.86)	(–1.10)	(–1.31)	(–0.58)	(0.36)
Income (4th qtl.)	–0.005	–0.030	–0.012	–0.033	–0.009	–0.072
	(–0.19)	(–0.12)	(–0.27)	(–0.69)	(–0.17)	(–1.39)
High school	0.001	0.001	0.046	0.044	0.035	0.048
	(0.58)	(0.23)	(1.12)	(1.01)	(0.72)	(0.93)
College	–0.038	–0.001	–0.087	–0.030	–0.090	–0.010
	(–1.52)	(–0.38)	(–1.90)	(–0.68)	(–1.81)	(–0.21)
Child health history						
Poor/fair SRH			0.059	0.182***	–0.009	0.188**
			(0.94)	(2.67)	(–0.14)	(2.57)
Less severe cond.			0.034	0.054	0.050**	0.057
			(1.40)	(1.77)	(1.96)	(1.73)
Severe cond.			0.056	0.023	0.076**	0.030
			(1.87)	(0.65)	(2.28)	(0.80)
Mental cond.			0.135**	–0.129	0.180***	–0.012
			(2.45)	(–1.82)	(2.87)	(–1.52)
Family background						
Father's age	–0.001**	–0.002***	–0.000	–0.001	–0.001	–0.002
	(–2.23)	(–2.69)	(–0.11)	(–0.88)	(–0.56)	(–1.23)
Mother's age	–0.001***	–0.001**	–0.001	–0.001	–0.001	–0.001
	(–2.66)	(–2.03)	(–0.74)	(–1.33)	(–0.75)	(–0.44)

Father's education					–0.007	0.009
					(–0.90)	(1.23)
Mother's education					0.011	–0.010
					(1.33)	(–1.25)
High family SES					0.005	0.105
					(0.07)	(1.24)
Low family SES					0.042	–0.027
					(1.07)	(–0.64)
Financial help					–0.033	–0.010
					(–0.56)	(–1.57)
Need to move					0.012	0.008
					(0.67)	(0.49)
Father unemployed					0.060	–0.027
					(1.52)	(–0.62)
Mother employed					0.020	0.007
					(1.56)	(0.48)
Parents smoked					0.012	–0.066**
					(0.76)	(–2.19)
Kid smoked					0.011	0.076
					(0.18)	(1.70)
Kid alcohol/drug					–0.000	0.152
					(–0.04)	(0.40)
Kid trouble learning					0.079	0.067
					(1.30)	(1.05)
Adult health history	M1	M1	M3	M3	M3	M3
N	31,805	23,268	11,573	7,954	9,630	6,705
Log likelihood	–16,668.7	–13,880.9	–4,637.9	–3,889.4	–3,845.1	–3,266.6

Notes: Results are for white females (F) and males (M). Abbreviations are as follows: *N* denotes the number of respondent-year observations. "Dry run" stands for dry runs that use the original specification by Adams et al. (2003). SRH stands for self-rated health during childhood. M1 and M3 abbreviate 1st-order and 3rd-order Markov processes, respectively.

[a]Dependent variable: number of acute health incidences.

***Significant at the 1 percent level.

**Significant at the 5 percent level.

*Significant at the 10 percent level.

Table 5A.3 Prediction model for chronic health conditions[a]

	Ordered probit regression coefficients z-statistics in parentheses					
	Dry run		Child health		Family	
Key explanatory variables	F (A)	M (B)	F (C)	M (D)	F (E)	M (F)
Current SES						
Wealth (1st qtl.)	0.005	0.112*	0.023	0.138***	0.02	0.144**
	(0.20)	(3.81)	(0.57)	(2.76)	(0.23)	(2.52)
Wealth (4th qtl.)	–0.028	0.005	–0.036	0.032	–0.048	0.051
	(–1.36)	(0.18)	(–1.10)	(0.79)	(–1.35)	(1.19)
Income (1st qtl.)	0.065***	–0.027	0.038	–0.015	0.086**	0.010
	(3.02)	(–1.04)	(1.06)	(–0.35)	(2.16)	(0.21)
Income (4th qtl.)	–0.012	–0.015	–0.006	–0.020	–0.007	–0.035
	(–0.49)	(–0.52)	(–0.14)	(–0.42)	(–0.17)	(–0.70)
High school	–0.083*	–0.008	0.017	–0.003	0.066	0.003
	(–3.81)	(–0.31)	(0.45)	(–0.78)	(1.51)	(0.93)
College	0.007	–0.092*	–0.087**	–0.100**	–0.063	–0.074
	(0.30)	(–3.58)	(–2.19)	(–2.31)	(–1.48)	(–1.58)
Child health history						
Poor/fair SRH			–0.002	0.088	0.012	–0.136
			(–0.03)	(–1.25)	(0.17)	(–1.79)
Less severe cond.			0.038	0.079***	0.035	0.067**
			(1.71)	(2.64)	(1.43)	(2.07)
Severe cond.			0.049	0.065	0.071**	0.092**
			(1.75)	(1.90)	(2.28)	(2.51)
Mental cond.			0.037	0.001	0.011	0.032
			(0.72)	(0.02)	(0.19)	(0.44)
Family background						
Father's age	–0.001**	0.001	–0.002**	0.001	–0.003**	–0.002
	(–2.35)	(0.70)	(–2.15)	(0.60)	(–2.53)	(–1.18)
Mother's age	0.001	0.001	0.000	0.001	0.001	0.000
	(0.08)	(0.48)	(0.44)	(0.69)	(0.73)	(0.30)

Father's education					0.001	−0.005
					(0.20)	(−0.73)
Mother's education					−0.004	−0.004
					(−0.59)	(−0.47)
High family SES					0.001	0.037
					(0.02)	(0.43)
Low family SES					−0.011	0.026
					(−0.31)	(0.63)
Financial help					−0.087	0.007
					(−1.64)	(0.11)
Need to move					0.065	−0.023
					(1.55)	(−0.48)
Father unemployed					0.024	0.028
					(0.69)	(0.66)
Mother employed					−0.007	0.013
					(−0.61)	(0.92)
Parents smoked					0.000	0.035
					(0.02)	(1.20)
Kid smoked					−0.074	0.059
					(−1.35)	(1.34)
Kid alcohol/drug					−0.000	−0.028
					(−0.78)	(−0.07)
Kid trouble learning					−0.083	0.131
					(−0.58)	(1.13)
Adult health history	M1	M1	M3	M3	M3	M3
N	29,649	21,344	11,573	7,954	9,630	6,705
Log likelihood	−16,150.6	−10,997.9	−6,206.0	−4,031.0	−5,125.3	−3,389.1

Notes: Results are for white females (F) and males (M). Abbreviations are as follows: *N* denotes the number of respondent-year observations. "Dry run" stands for dry runs that use the original specification by Adams et al. (2003). SRH stands for self-rated health during childhood. M1 and M3 abbreviate 1st-order and 3rd-order Markov processes, respectively.

[a]Dependent variable: number of chronic health incidences.

***Significant at the 1 percent level.

**Significant at the 5 percent level.

*Significant at the 10 percent level.

Table 5A.4 Prediction model for functional health conditions[a]

	Ordered probit regression coefficients *z*-statistics in parentheses					
	Dry run		Child health		Family	
Key explanatory variables	F (A)	M (B)	F (C)	M (D)	F (E)	M (F)
Current SES						
Wealth (1st qtl.)	0.000	0.091*	0.024	0.081**	0.023	0.059
	(0.01)	(3.84)	(0.74)	(1.98)	(0.64)	(1.26)
Wealth (4th qtl.)	–0.012	–0.027	0.025	–0.010	0.010	–0.006
	(–0.74)	(–1.33)	(0.99)	(–0.31)	(0.37)	(–0.17)
Income (1st qtl.)	–0.009	0.020	–0.036	0.038	–0.016	0.043
	(–0.55)	(0.94)	(–1.29)	(1.27)	(–0.51)	(1.08)
Income (4th qtl.)	–0.010	0.017	–0.004	0.047	–0.006	0.030
	(–0.51)	(0.72)	(–0.13)	(1.24)	(–0.17)	(0.73)
High school	–0.044***	–0.017	–0.046	–0.009	–0.046	0.014
	(–2.61)	(–0.79)	(–1.58)	(–0.24)	(–1.34)	(0.34)
College	–0.062*	–0.060*	–0.041	–0.119*	–0.049	–0.129*
	(–3.33)	(–2.91)	(–1.35)	(–3.43)	(–1.46)	(–3.44)
Child health history						
Poor/fair SRH			–0.032	–0.011	–0.045	–0.027
			(–0.68)	(–0.19)	(–0.86)	(–0.44)
Less severe cond.			0.023	0.030	0.030	0.056**
			(1.30)	(1.22)	(1.54)	(2.09)
Severe cond.			0.054**	0.051	0.035	0.041
			(2.45)	(1.84)	(1.43)	(1.37)
Mental cond.			0.159*	0.039	0.156***	0.066
			(3.82)	(0.75)	(3.24)	(1.11)
Family background						
Father's age	0.000	0.000	–0.001	0.001	–0.001	0.001
	(0.89)	(0.63)	(–0.74)	(1.45)	(–1.17)	(1.45)
Mother's age	–0.000	0.001	0.001	–0.001	0.001	–0.000
	(–0.43)	(0.92)	(1.42)	(–0.68)	(1.05)	(–0.33)

Father's education					0.000	–0.011
					(0.04)	(–1.87)
Mother's education					0.002	0.000
					(0.44)	(0.02)
High family SES					0.084	0.127
					(1.71)	(1.86)
Low family SES					0.122***	–0.017
					(4.45)	(–0.50)
Financial help					0.059	–0.001
					(1.46)	(–0.11)
Need to move					0.072**	0.032
					(2.20)	(0.80)
Father unemployed					–0.068**	0.026
					(–2.45)	(0.75)
Mother employed					–0.008	–0.001
					(–0.90)	(–0.08)
Parents smoked					0.055***	0.044
					(2.99)	(1.84)
Kid smoked					0.035	–0.013
					(0.80)	(–0.35)
Kid alcohol/drug					0.367	–0.049
					(0.76)	(–1.36)
Kid trouble learning					0.009	0.031
					(0.08)	(0.31)
Adult health history	M1	M1	M3	M3	M3	M3
N	29,649	21,344	11,573	7,954	9,630	6,705
Log likelihood	–66,786.2	–22,219.3	–13,213.0	–7,589.5	–10,870.5	–6,372.4

Notes: Results are for white females (F) and males (M). Abbreviations are as follows: *N* denotes the number of respondent-year observations. "Dry run" stands for dry runs that use the original specification by Adams et al. (2003). SRH stands for self-rated health during childhood. M1 and M3 abbreviate 1st-order and 3rd-order Markov processes, respectively.

[a]Dependent variable: number of functional health incidences.

***Significant at the 1 percent level.

**Significant at the 5 percent level.

*Significant at the 10 percent level.

Table 5A.5 Prediction model for mental health conditions[a]

	Ordered probit regression coefficients *z-statistics in parentheses*					
	Dry run		Child health		Family	
Key explanatory variables	F (A)	M (B)	F (C)	M (D)	F (E)	M (F)
Current SES						
Wealth (1st qtl.)	0.061**	0.156*	0.065	0.223*	0.088	0.279*
	(2.40)	(4.92)	(1.42)	(3.83)	(1.69)	(4.21)
Wealth (4th qtl.)	–0.016	–0.021	–0.024	–0.011	–0.020	–0.022
	(–0.65)	(–0.71)	(–0.60)	(–0.22)	(–0.46)	(–0.39)
Income (1st qtl.)	0.072***	0.087***	0.006	0.030	–0.051	0.028
	(3.17)	(3.00)	(0.13)	(0.56)	(–1.10)	(0.46)
Income (4th qtl.)	–0.088***	–0.065	–0.079	0.015	–0.105	0.030
	(–3.03)	(–1.88)	(–1.59)	(0.25)	(–1.94)	(0.45)
High school	–0.188*	–0.174*	–0.147*	–0.147*	–0.089	–0.146**
	(–8.19)	(–6.22)	(–3.58)	(–2.87)	(–1.82)	(–2.41)
College	–0.048	–0.081***	–0.104**	–0.118**	–0.089	–0.106
	(–1.72)	(–2.65)	(–2.09)	(–2.04)	(–1.64)	(–1.70)
Child health history						
Poor/fair SRH			0.023	–0.078	0.029	–0.097
			(0.34)	(–0.92)	(0.38)	(–1.03)
Less severe cond.			0.061**	0.013	0.064**	0.033
			(2.37)	(0.35)	(2.28)	(0.81)
Severe cond.			–0.003	0.068	0.002	0.074
			(–0.08)	(1.64)	(0.07)	(1.60)
Mental cond.			0.296*	0.210***	0.271*	0.156
			(5.43)	(2.88)	(4.27)	(1.82)
Family background						
Father's age	0.001	–0.000	0.001	0.000	0.001	–0.001
	(1.04)	(–0.31)	(1.19)	(0.02)	(0.74)	(–0.36)
Mother's age	0.000	0.001	0.001	0.000	0.001	–0.000
	(0.33)	(0.65)	(0.50)	(0.22)	(0.78)	(–0.23)

Father's education					–0.008	–0.010
					(–1.10)	(–1.01)
Mother's education					–0.009	0.011
					(–1.03)	(1.05)
High family SES					–0.038	–0.018
					(–0.49)	(–1.51)
Low family SES					0.013	–0.031
					(0.33)	(–0.60)
Financial help					0.093	–0.043
					(1.54)	(–0.57)
Need to move					–0.031	0.055
					(–0.64)	(0.90)
Father unemployed					–0.057	0.034
					(–1.36)	(0.62)
Mother employed					–0.006	–0.004
					(–0.45)	(–0.25)
Parents smoked					–0.034	0.023
					(–1.20)	(0.61)
Kid smoked					0.109	–0.023
					(1.73)	(–0.40)
Kid alcohol/drug					0.246	–0.215
					(0.38)	(–0.47)
Kid trouble learning					0.338**	0.076
					(2.41)	(0.52)
Adult health history	M1	M1	M3	M3	M3	M3
N	29,649	21,344	11,573	7,954	9,630	6,705
Log likelihood	–12,117.7	–7,737.3	–4,177.3	–2,410.8	–3,410.6	–1,966.7

Notes: Results are for white females (F) and males (M). Abbreviations are as follows: *N* denotes the number of respondent-year observations. "Dry run" stands for dry runs that use the original specification by Adams et al. (2003). SRH stands for self-rated health during childhood. M1 and M3 abbreviate 1st-order and 3rd-order Markov processes, respectively.

[a]Dependent variable: number of mental health incidences.

***Significant at the 1 percent level.

**Significant at the 5 percent level.

*Significant at the 10 percent level.

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Comment

Robert J. Willis

This is the tenth anniversary of the publication of Adams et al. (2003) that introduced the idea of using Granger causality to test hypotheses about causal factors that underlie correlations between health and socioeconomic status. This paper generated a great deal of controversy about the interpretation of the Granger approach—and the meaning of causality more generally—and the implications of their empirical results in the context of conflicting literatures in epidemiology and economics about causal factors underlying the SES gradient in health. Using longitudinal data from the AHEAD cohort of the HRS containing persons age seventy and over at baseline, they found that health shocks Granger-cause changes in wealth but they rejected the hypothesis that SES Granger-causes health.

The Adams et al. (2003) finding of a causal effect of health on SES, a line of causation largely ignored by epidemiologists, was uncontroversial. However, their finding of Granger noncausation of SES on health flew in the face of an epidemiology literature in which virtually all correlations between SES and health were assumed to reflect this line of causation. Although Granger causation provides little insight into the particular mechanisms that may connect innovations in socioeconomic variables to changes in health, rejection of Granger causation may seem to undermine much of the epidemiological literature in one fell swoop because, if the noncausality results

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