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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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are taken at face value, all possible causal mechanisms by which SES affects health are shown to be statistically insignificant.

Of course, such a sweeping conclusion is too strong because the Adams et al. (2003) results were for a particular sample of given size of quite elderly people who are largely retired and covered by Medicare. Granger causation might be found in a larger sample of persons with a longer period of observation, especially in age groups in which economic and social factors might play a larger role in determining access to care or motivation for the maintenance of health. With these possibilities in mind, Stowasser et al. (2012) replicated the methodology of Adams et al. (2003), adding data from the younger HRS cohorts when they reach age sixty-five to the then longer histories of the AHEAD cohort. With the additional statistical power provided by this enlarged sample, Stowasser et al. (2012) found that they could not generally reject Granger causation from SES to health. Thus, they could not rule out any of the three possible hypotheses concerning the correlation between SES and health: SES causes health, health causes SES, or that both health and SES are caused by some third set of unmeasured factors.

In the current chapter, Stowasser et al. continue to use the Granger methodology, with a focus on seeing what they can say about the line of causation from SES to health, while doing what they can to control for unmeasured heterogeneity that might lead to spurious correlations between SES and health, by allowing for higher than first-order Markov dependence of current health on past health to capture the capital-like character of health and by including measures of childhood health and family background. Although more elaborate controls and longer lags result in losses in sample size, overall they conclude that there is credible evidence of causal impacts of SES on health, with the exception of acute conditions.

Given the original emphasis in Adams et al. (2003) on the strong implications of findings of Granger noncausality, I found it surprising that the current chapter provides little discussion of the implications of what they find to be quite strong evidence of noncausality for heart attacks, stroke, and cancer, which comprise the aggregate category they label as acute life-threatening conditions. These conditions are, of course, among the major killers in advanced societies that are well past the epidemiological transition in which death from infectious disease is replaced by death from chronic illness. A finding of Granger noncausality for SES on these conditions implies that no pathway involving economic resources would have any effect on the incidence of heart attack, stroke, or cancer among the population of people over age sixty-five.

To me, this seems to be a potentially major finding worthy of further discussion. What are the specific hypotheses put forward by economists, epidemiologists, or others that are ruled out by the finding of Granger noncausality of SES for heart attack, stroke, or cancer among older people? I am not an expert in this area, so I do not know whether this finding would

(or should) change the views of experts in epidemiology or health economics. For example, do many of the experts believe that the extra access to care afforded by the purchase of medigap insurance provides no extra protection beyond what basic Medicare provides in the case of acute disease? Actually, I suspect that most experts would say that money buys little protection against the occurrence of these diseases, but may help prevent or delay death from them once they occur by providing access to more or better quality medical care. The failure of the authors to reject noncausality for mortality would support this argument.

Stowasser et al. find significant effects of higher-order Markov dependency and, often, of childhood health and family background variables, which implies that one's current health status depends on a "long memory" of past health shocks. This important finding suggests that any causal account of the determinants of the SES-health gradient is likely to be very complex, with room for feedback loops involving causation running in both directions in a high dimensional state space. The authors present the Granger causality approach as an alternative to structural models that require some kind of exogenous instrument to identify causal effects. Valid instruments are difficult to find and, even when available, may not identify causal effects of general interest. However, the Granger approach, at best, seems to offer clues for more focused research on particular issues. For example, what should one make of their finding that mental health problems are linked to childhood conditions for females, but not for males? Assuming this differential to be a true empirical regularity, it still requires some theoretical ideas or hunches about differences in the underlying mechanisms that determine mental health to indicate which among many possible lines of focused research might provide a causal explanation of this finding.

In sum, the line of research that began a decade ago with Adams et al. (2003) has shown that it is imperative to view the determinants of health within a dynamic framework encompassing the entire life cycle. Their data-hungry research program has been aided enormously—and increasingly—by availability of longitudinal data from the HRS, which grows in length with each wave and, in recent waves, has attempted to capture early life SES and health conditions. The early hope that the Granger methodology could reject whole classes of potential true causal models has not been fulfilled. Rather, as more and better data have been brought to bear, most fully in this chapter, their findings suggest that an understanding of the SES-health gradient must include causal arrows running in both directions, along with a host of third factors that drive both SES and health in a dynamic process with many feedback loops and a long memory. Needless to say, this won't be quick work.