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Commentary

Some causal lessons from macroeconomics

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Causality has long been regarded among economists as a metaphysical minefield, best to be avoided. Yet, at the same time, the notion of cause and effect structures our ordinary lives and seems essential for understanding policy actions. Peter Adams et al.'s "Healthy, Wealthy, and Wise?" is welcome as an example of the relatively recent interest of microeconomists in overcoming their metaphysical aversions in order to say something useful about cause and effect. Their study is carefully executed and aims to resolve a well-posed causal question. The authors' panel-data approach is closely analogous to the vector-autoregression (VAR) framework of macroeconomists. I want to pursue some of those analogies as they relate to causal inference. It is important to acknowledge that, because of the small number of periods, some of the technical issues in the estimation of panel data are different from typical time-series data. The issues related to casual interpretation, however, do not depend on these differences.

Adams et al.'s notion of cause can be summarized as *time order* plus *explanatory power* plus *invariance*. I have argued (Hoover, 2001) for a structural account of causation to which each of these three elements is related in a complex way. Macroeconomic examples demonstrate that none of these elements adequately defines causality (see Hoover, 2001, especially Chapters 6 and 7). But some of the special features of macroeconomics are less salient in microeconomic contexts, and it is at least plausible that Adams et al.'s criteria are discriminating enough for their application.

To clarify ideas, consider a schematic structural model in which health (H) and wealth (W) are related in time as

$$H_t = \alpha_1 W_t + \alpha_2 W_{t-1} + \alpha_3 H_{t-1} + \varepsilon_{Ht}, \tag{1}$$

$$W_t = \beta_1 H_t + \beta_2 H_{t-1} + \beta_3 W_{t-1} + \varepsilon_{Wt}. \tag{2}$$

If the i.i.d. error terms (the ε's) are orthogonal and if the parameters are independent in the sense that setting of one does not restrict the range of the others, then (1) and (2) can be interpreted causally. Shorn of its details, Adams et al.'s strategy is to estimate a model like (1) and (2) and to use it to test whether it is invariant between

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successive waves of the AHEAD data set and whether the coefficients corresponding to α_2 and β_2 are zero. An important difference between Adams et al.'s specifications and the VAR is that they have a large number of observations on each variable at each period rather than just one as in a time series. If Adams et al. can reject invariance or fail to reject that the coefficients are zero, they believe that they have rejected the causal link between past wealth and current health and between past health and current wealth.

Their focus is on testing just those two links. They also impose strong causal assumptions that they do not test. In particular—although, for practical reasons, they decline to dismiss all contemporaneous causal connections—they do not permit current wealth to cause current health: $\alpha_1 = 0$. Furthermore, Eq. (1) is vector valued—health has many facets $(H_{1t}, H_{2t}, \ldots, H_{nt})$. These variables are causally ordered in a Wold causal chain in which H_{1t} causes H_{2t}, H_{1t} and H_{2t} cause H_{3t}, and so forth. In each case, the authors give reasons why these assumptions are credible. It was precisely the claim that the analogous identifying assumptions in macroeconomics were "incredible" that led Christopher Sims (1980) to launch the VAR program. Sims advocated estimating

$$H_t = \Pi_1 W_{t-1} + \Pi_2 H_{t-1} + \omega_{Ht}, \tag{3}$$

$$W_t = \Gamma_1 H_{t-1} + \Gamma_2 W_{t-1} + \omega_{Wt}, \tag{4}$$

where the ω's are error terms.

Sims suggested tracing out the responses of the variables to impulses given to the structural error terms. In general, however, $\mathrm{cov}(\omega_{Ht}\omega_{Wt}) \neq 0$, so that the idea of a health shock or a wealth shock is ambiguous. Sims proposed transforming the system through a transformation based on the Choleski factorization of the matrix $[\omega_{Ht}\omega_{Wt}]'$. The covariance matrix after the transformation is diagonal, so each equation is associated with a unique shock, and the contemporaneous variables form a lower triangular matrix. The transformation of (3) and (4), for example, would take the form of (1) and (2) with either $\alpha_1 = 0$ (if health is ordered ahead of wealth) or $\beta_1 = 0$ (if wealth is ordered ahead of health), and $\mathrm{cov}(\varepsilon_{Ht}\varepsilon_{Wt}) = 0$. If, as in Adams et al.'s study, H is a vector, then β_1 would be a lower triangular matrix.

As with impulse-response functions for the VAR, Adams et al.'s counterfactual simulations require structure on the contemporaneous variables. It was quickly realized that there were as many different Choleski transformations as there were variables and that were even more admissible non-Choleski orthogonalizing transformations (e.g., Cooley and LeRoy, 1985; Leamer, 1985; Sims, 1986). Each amounts to imposing a causal ordering on the contemporaneous variables. In general, judged on likelihood, each of the different orders is equivalent, since each has the same reduced form. Yet, the impulse responses (and, in Adams et al.'s study, the counterfactual simulations) based on different orders will be different. A VAR with a particular causal ordering of the contemporaneous variables is a *structural* VAR. Adams et al. follow the tactic of the typical macroeconomist and estimate a structural model with a particular Wold causal ordering based on plausible timing relationships. Plausibility, like credibility, lies in the eye of the beholder.

For cross-sectional data, Pearl (2000) and Spirtes et al. (2000) suggest using the *causal Markov condition* as a means of bringing evidence to bear on causal order. Roughly, the causal Markov condition says that two correlated variables with a common cause or an intervening cause will be uncorrelated conditional on the common (or intervening) cause. If the causal structure is $A \leftarrow C \rightarrow B$ or $A \rightarrow C \rightarrow B$, then A and B are unconditionally correlated but are uncorrelated *conditional* on C. Another important result is that if A and B are unconditionally uncorrelated and $A \rightarrow C \leftarrow B$, then A and C are correlated conditional on C. (For example, the battery being good and the switch being on may be uncorrelated, but conditional on the flashlight lighting, they are correlated.) When there is no direct causal connection between A and B, this pattern is known as an *unshielded collider*. Spirtes et al. (2000) have developed powerful algorithms for constructing mappings between graphical representations of causal relationships and the multivariate probability distribution of variables.[1] The algorithms systematically search for the screening relationships of the causal Markov condition and the induced correlations of the unshielded colliders.

Spirtes et al.'s (2000) algorithms do not work directly on time-dependent data. Swanson and Granger (1997) proposed using the VAR to prefilter the data to remove the time dependence (cf. Hoover, 2001, pp. 160–164). In essence, they suggest applying the algorithms to the estimated residuals, $\hat{\omega}_{Ht}$ and $\hat{\omega}_{Wt}$, from (3) and (4) rather than to H_t and W_t directly, and then ordering H_t and W_t in the order that the algorithm selects for the corresponding prefiltered variables. The same strategy could be adapted to Adams et al.'s panel-data framework. To the degree that cross-sectional variation dominates in the panel-data setting, it might work just as well to apply the algorithms to H_t and W_t directly.

An important theorem says that any probability distribution that can be faithfully represented in an *acyclical* graph (i.e., one without feedbacks between variables) can equally be well represented by another acyclical graph that has the same direct linkages (regardless of direction) and the same unshielded colliders (Pearl, 2000, p. 19; Spirtes et al., 2000, Chapter 4). As a result, there may be observationally equivalent causal structures in which some causal links are reversed but all unshielded colliders preserved. The algorithm, in those cases, yield only partial causal orderings.

Hoover (1990, 1991, 2001, Chapter 8) suggests invariance tests as a way of resolving unordered causal linkages in these observationally equivalent cases. To take a simple example that focuses on normally distributed contemporaneous variables, the joint distribution of H and W can be partitioned as the product of a conditional and a marginal in two ways: $N(H,W)=N(H|W)N(W)=N(W|H)N(H)$. Each can be thought of as representing a causal order. Assume that in truth W causes H and that the true partition can be represented by $N(H|W) = N(\alpha W, \sigma_H^2)$ and $N(W) = N(\beta, \sigma_W^2)$. These distributions can be estimated using regressions. Reversing the order of the variables in these regressions would allow us to estimate the other partition. Given our assumed causal order, those distributions would be

$$N(W|H) = N\left(\frac{\alpha\sigma_W^2 + \beta\sigma_H^2}{\alpha^2\sigma_W^2 + \sigma_H^2}, \frac{\sigma_W^2\sigma_H^2}{\alpha^2\sigma_W^2 + \sigma_H^2}\right)$$

[1] These algorithms are implemented in the program *Tetrad II*, described in Spirtes et al. (2000).

and

$$N(H) = N(\alpha\beta, \alpha^2\sigma_W^2 + \sigma_H^2).$$

Now imagine an intervention that alters the true distribution of W. We do not need to know exactly what it does structurally, so long as there is convincing reason to believe from historical, institutional, or other extra-statistical evidence that it directly affects W and, at best, indirectly H. One of the parameters of the W-process (either β or σ_W^2) must have changed. Notice that $N(W)$, $N(W|H)$ and $N(H)$ have all changed, yet $N(H|W)$ remains invariant. This pattern of invariance is characteristic of W causing H. (There are other patterns of invariance that may be helpful as well.) By estimating regressions that correspond to the competing partitions and checking their patterns of invariance against known interventions, it is sometimes possible to identify the true causal direction—even in the cases that are observationally equivalent in a single regime (that is, one without interventions) and, thus, not susceptible to resolution using the Spirtes et al. algorithms.

While this account of causal inference demonstrates that Adams et al. are right to regard invariance as an important indicator of a causal relationship, it also casts doubt on the cogency of their evidence. Essentially, they perform Chow tests on the *conditional* regression models. A finding of invariance in the conditional model does not indicate a true causal relationship *unless* there is simultaneously a failure of invariance in the marginal model for the conditioning variable. A policy intervention that shifted the marginal distribution for wealth might be an example of such an intervention. Without a statistically significant intervention in the marginal process, then a finding of invariance in the conditional model does not provide evidence that discriminates between wealth causing health and health causing wealth. What is needed are tests of *superexogeneity* in the sense of Engle et al. (1983) (see also Hendry, 1995, Chapter 14, Sections 16–18; Engle and Hendry, 1993; Hoover, 2001, Chapter 7, Section 3). Since the counterfactual experiments proposed in Section 7 of Adams et al.'s paper are themselves interventions of the required type, only evidence of superexogeneity would support such experiments.

On the other hand, finding a failure of invariance in the conditional model without a failure in the marginal model may be the result of misspecification—for example, an omitted variable. One reason for looking for non-statistical evidence of interventions is to convince oneself that failures of invariance correspond to genuine interventions and not to misspecification.

To sum up: the analysis of causality in a VAR framework carries important lessons for panel-studies such as Adams et al. present. Their results—both for the main causal links of interest (namely, the direct and indirect linkages among health indicators and effects of lagged wealth on health and lagged health on wealth) and for the counterfactual simulations—may be sensitive to their strong, untested assumptions (the omission of a contemporaneous link from wealth to health and the particular Wold causal ordering of the health indicators). I have suggested that there may be positive ways to bring empirical evidence to bear on these assumptions. In addition, causal inference requires more stringent tests of invariance than the authors conduct in this study.

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