

# Probability and Structure in Econometric Models

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19 September 2007

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An earlier draft was presented to the 13<sup>th</sup> International Congress of Logic, Methodology, and the Philosophy of Science at Tsinghua University, Beijing, China 8-16 August 2007. I thank Julian Reiss for comments on that draft.

**Abstract**  
**of**  
**“Probability and Structure in Econometric Models”**

The difficulty of conducting relevant experiments has long been regarded as the central challenge to learning about the economy from data. The standard solution, going back to Haavelmo’s famous “The Probability Approach in Econometrics” (1944), involved two elements: first, it placed substantial weight on *a priori* theory as a source of structural information, reducing econometric estimates to measurements of causally articulated systems; second, it emphasized the need for an appropriate statistical model of the data. These elements are usually seen as tightly linked. I argue that they are, to a large extent, separable. Careful attention to the role of an empirically justified statistical model in underwriting probability explains puzzles not only in economics, but more generally with respect to recent criticisms of Reichenbach’s principle of the common cause, which lies behind graph-theoretic causal search algorithms. And it provides an antidote to the pessimistic understanding of the possibilities for passive observation of causal structure in econometrics and related areas of Nancy Cartwright and others.

## **Probability and Structure in Econometric Models**

### **I. Econometrics and the Problem of Passive Observation**

For nearly two centuries – at least since John Stuart Mill (1844 [1967], p. 327) – philosophers have observed, and economists have lamented, the barriers to turning economics into an experimental science. As important as it is, the recent rise of a field known as experimental economics is, and promises to be, far too limited in scope to alter radically the fact that most empirical economics must trade in passive, rather than experimental, observation. At one point, the lack of scope for controlled experiments was seen as a serious barrier to the application of modern, probability-based statistics to economics. The situation was saved – or, at least, economists were comforted – with the publication of Trgyve Haavelmo’s “The Probability Approach in Econometrics” (1944) and the subsequent development of the theory of econometric identification by the Cowles Commission (Koopmans 1950; Hood and Koopmans 1953; see Boumans 2007 for an excellent history of these developments).

There were two critical ideas in the new approach. The first is that statistical controls, accounting for covariates, could take the place of experimental controls (see Morgan 1990, pp. ch. 8, esp. pp. 246-248). Before Haavelmo, passive observations, especially of temporally ordered data (time series), had been seen as unlike repeated observations on, say, a field of an agricultural research station. Rather than repeated draws from a fixed distribution, observations were drawn from different distributions, the relationships of which were opaque. Haavelmo proposed that an economic process could be partitioned into a deterministic and a random part. If the causal structure of the deterministic part were articulated fully and accurately enough, the random part would conform to the laws of probability. Each variable would be generated in part from a

random “error,” and the error-terms could be seen as independent draws from a constant multivariate probability distribution. Or, equally, all the errors for a set of observations could be seen as a single, independent  $n$ -dimensional draw from draw from the same distribution – one of a hypothetically infinite number of such draws. Characterized in either of these ways, standard statistical techniques applied.

The second critical idea was that the causal structure of the deterministic part of the economic process had to be accurately articulated. Haavelmo suggested that *a priori* economic theory could provide the needed causal articulation. The second idea was the necessary prop of the first. The Cowles Commission took up Haavelmo’s idea and analyzed the conditions under which, conditional on knowing the causal connections among variables, the strengths of those connections could be measured through statistical estimation. For reasons that are not important here, the Cowles Commission initiated a conversion in econometrics from explicit talk of cause and effect to the, at best, implicitly causal “endogenous” and “exogenous” variables.<sup>1</sup> What is important is the discovery that causal structure is richer than, or, at least, distinct from, the probability structure.

Take a very simple example. Suppose that  $A$  causes  $B$ , where  $A$  and  $B$  are two stochastic variables. Their relationship can be represented graphically as  $A \rightarrow B$  and algebraically, with some additional structure, as:

$$(1) \quad A \leftarrow \varepsilon_A,$$

$$(2) \quad B \leftarrow \alpha A + \varepsilon_B,$$

where  $\varepsilon_A$  and  $\varepsilon_B$  are random error terms; for convenience we assume that each is distributed independent normal with mean zero and variances  $\sigma_A^2$  and  $\sigma_B^2$  (notated

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<sup>1</sup> See Hoover (2003) for an historical discussion.

$\varepsilon_A \sim$  independent  $N(0, \sigma_A^2)$  and  $\varepsilon_B \sim$  independent  $N(0, \sigma_B^2)$ . Each is independent across successive draws and independent from each other (which implies that the covariance of  $\varepsilon_A$  and  $\varepsilon_B$  is zero ( $\text{cov}(\varepsilon_A, \varepsilon_B) = 0$ ). The coefficient  $\alpha$  is a fixed parameter. And the arrowhead on the equal sign turns it into an assignment operator, a reminder that the model incorporates the asymmetry of causation.

The causal structure of (1) and (2) determines its probability structure. Substituting (1) into (2) yields what econometricians refer to as *reduced forms*, which completely characterize the probability structure of the variables:

$$(3) \quad A = \varepsilon_A = E_A,$$

$$(4) \quad B = \alpha\varepsilon_A + \varepsilon_B = E_B.$$

$E_A$  and  $E_B$  are themselves distributed  $E_A \sim$  independent  $N(0, \sigma_A^2)$  and  $E_B \sim$  independent  $N(0, \alpha^2\sigma_A^2 + \sigma_B^2)$ . But they are not independent of each other; in fact,  $\text{cov}(E_A, E_B) = \alpha\sigma_A^2 = \Sigma \neq 0$ .

The sense in which the causal structure is richer than the probabilistic structure is that implication runs one-way: (1) and (2) imply (3) and (4), but not the other way round. In fact, if  $B \rightarrow A$  with a causal structure analogous to (1) and (2), instead of  $A \rightarrow B$ , we can generate the reduced forms:

$$(5) \quad A = \varepsilon'_A + \beta\varepsilon'_B = E_A,$$

$$(6) \quad B = \varepsilon'_B = E_B.$$

The important thing is despite the difference in causal structures (reflected in the difference in the middle terms in the two sets of equations), both equations (3) and (4) and equations (5) and (6) define the same random terms,  $E_A$  and  $E_B$ . And these terms

have the same interdependence in each case:  $\text{cov}(E_A, E_B) = \beta\sigma_B^2 = \Sigma$ . They define the same probability distribution; they are observationally equivalent; or, in the argot of econometrics, they are not identified.

The observational equivalence of the two sets of equations means that we can work backwards to form estimates of the parameters only if we are willing to commit to a particular causal structure. If we believe that theory (or some other extra-statistical source) tells us that (1) and (2) constitute the correct causal structure, then we can use observations on  $A$  and  $B$  to estimate the parameter  $\alpha$ . But what if, as economists often believe, causation is mutual ( $A \leftrightarrow B$ )? For example, suppose that the causal structure is

$$(7) \quad A \leftarrow \beta'B + \varepsilon_A'',$$

$$(8) \quad B \leftarrow \alpha'A + \varepsilon_B''.$$

Then there are infinite combinations of causal strengths connecting them, so that the equivalence class is itself infinite.<sup>2</sup> There is no way to recover estimates of  $\alpha'$  or  $\beta'$  without further non-empirical assumptions (e.g., about their relative strengths). This is the classic *identification problem* in econometrics.

The classic solution is to imagine that  $A$  and  $B$  are subject to independent experimental control. Suppose

$$(9) \quad X \rightarrow A \leftrightarrow B \leftarrow Y,$$

where  $X$  is a means of intervening on  $A$  independent of  $B$  or  $Y$ , and  $Y$  is a means of intervening on  $B$  independent of  $A$  or  $X$ . The Cowles Commission showed that, in such

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<sup>2</sup> The reduced forms are  $A = \left( \frac{1}{1 - \alpha'\beta'} \right) (\varepsilon_A'' + \beta'\varepsilon_B'') = E_A$  and  $B = \left( \frac{1}{1 - \alpha'\beta'} \right) (\alpha'\varepsilon_A'' + \varepsilon_B'') = E_B$ . Once again, they define the same probability distribution as indicated by the right-hand terms.

circumstances, unique estimates of  $\alpha'$  or  $\beta'$  could be recovered. If we have independent reasons for thinking that the world is structured like (9) and that  $X$  and  $Y$  can somehow be observed, then passive observations could replace controlled experiments.<sup>3</sup> In macroeconomics, the analogy with controlled experiments is the basis for techniques of causal inference based on patterns of invariance and noninvariance (Hoover 2001a, chapters 8-10); while in microeconomics, it motivates the search for “natural experiments” (Angrist and Krueger 1999). The Cowles Commission itself and econometric orthodoxy in the second half of the 20<sup>th</sup> century downplayed experimental analogues, emphasizing instead the role of *a priori* economic theory in selecting the warranted causal structure such as (9), in which  $X$  and  $Y$  are just additional observed variables, now christened *instrumental variables* (or just *instruments*).

The Cowles Commission’s strategy opened an era of optimism about the possibilities for passive observation and the articulation of causal structure. Soon, however, pessimism set in: where does our confidence in the causal structure come from (Liu 1960)? Christopher Sims (1980) famously stigmatized the assumed causal order as relying on “incredible” identifying restrictions (i.e., assumptions about which variables are *not* causally connected). For a while, some economists were resigned to using reduced forms only, but one can say so little about policy problems without a causal understanding that a whiff of *a priori* theory was soon reintroduced; and, for those who were still skeptical of *a priori* theory, natural experiments became the lodestone.<sup>4</sup>

Behind the alternating optimism and pessimism lies perhaps the biggest questions in empirical economics: how, and exactly what, can economists learn from passively

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<sup>3</sup> Scheines (2005) provides a careful exposition not only of the logic of such inference from natural experiments, but also of the close analogy with the logic of controlled experiments.

<sup>4</sup> See Hoover (2006) and Hoover (forthcoming) for fuller accounts.

data? Marcel Boumans (2007) characterizes the Cowles Commission as having given up on discovery and testing in favor of the measurement of parameters. This seems only partly right, since the set of restrictions on a causal structure needed to identify it for estimation is generally smaller than the set of all possible restrictions, so that testing the so-called *overidentifying restrictions* on the maintained assumption of a minimal identifying set is still possible. Nevertheless, the official methodology of the Cowles Commission has little to say about discovery. And despite practical successes in empirical econometrics, the official methodology is a form of rationalism that provides no account of how one could ever infer causal structure from data.

## **II. Probability Models: Function and Inference**

In clarifying the identification problem, however, the Cowles Commission raises issues that go beyond economics. Nancy Cartwright (1989) maintains that econometrics provides the clearest example of how probability should function in physical, as well as social, sciences. What impresses Cartwright are the detailed theoretical assumptions that inform theoretically identified econometric models. These correspond, in experimental contexts to experimental controls and shielding, which she argues allow “nature’s capacities” to display themselves without conflating interference, just as the set of instrumental variables allow the strengths of causal connections to be measured in econometric models. Cartwright (1999, p. 173) emphasizes the stringency of the conditions needed to throw capacities into clear relief:

My claim is that it takes hyperfine-tuning . . . to get a probability. Once we review how probabilities are associated with very special kinds of models before they are linked to the world, both in probability theory itself and in empirical theories like physics and economics, we will no longer be tempted to suppose that just any situation can be described by some probability distribution or other. It takes a very



special kind of situation with arrangements set just right – and not interfered with – before a probabilistic law can arise.

And while she is grateful to econometricians for clarifying the logic of the problem, she is pessimistic with respect to the project of applied econometrics, seeing econometricians as having themselves laid the groundwork for showing it to be a quite hopeless undertaking (Cartwright 1999, chs. 6 and 7).<sup>5</sup>

Cartwright’s pessimism about econometrics is based in what it teaches her about the application of probability. First, probabilities are not simply there in the world to be invoked whenever it suits our inferential purposes. Rather, following Ian Hacking (1965), she argues that probabilities are ways of codifying the propensities of physical (and perhaps social) machinery to display behavior with frequencies that follow certain patterns. The propensity of a coin to display heads half the time and to provide no evidence of dependence between successive tosses (in short, to follow a binomial probability model with the key parameter set to  $\frac{1}{2}$ ), requires that the coin, the flipping device, and the environment to be constituted in highly particular ways, and only then can we expect the probability model to apply. Physical experiments are just examples of such *nomological machines*; while what an applied economic theory describes is a *socioeconomic machine*. But whereas a description of a nomological machine may be a true report, a description of a socioeconomic machine is almost always a work of science fiction – they may be edifying stories, but the machines that they describe can be built rarely, if at all.

Second, Cartwright asks, how do probabilities attach to the world? Her answer is that probabilities attach to the world through models. It is the actual success of the

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<sup>5</sup> Hoover (2001b, 2002) shows that Cartwright’s position is genuinely and unnecessarily, if only *implicitly*, pessimistic.

binomial probability model in describing the frequencies generated in the coin-flipping nomological machine that warrants claims about probabilities – for example, claims about how often five heads in a row ought to be expected.

Two of the theses that I will elaborate and defend in this paper are reactions or qualifications of the lessons that Cartwright draws from econometrics. First, her position that probabilities arise only in chance setups is correct up to a point. The generation of stable frequencies is a property of the real-world (not just physical) systems appropriately configured. But consistent with Hacking (1965, pp. 24-25), it is not the frequencies directly but the frequencies on particular kinds of trials that exhibit chance behavior. The same data may be viewed on different kinds of trials: “there is nothing unusual about regarding one event under several aspects” (Hacking 1965, p. 25). Different probability models may be applied to the same data without conflict or contradiction.

And this connects to my second thesis: Cartwright is correct to stress the role of models, but their role is not merely to attach probabilities to the world, but to create probabilities. Without the models, there are no probabilities to discuss. This is not an anti-realist thesis. For I suppose that that some models are better than others when judged in relation to actual frequencies from a particular aspect or point of view and that different points of view may lead to different, but not contradictory, probability models. The upshot of my theses is that a (perhaps, *the*) central problem of econometrics is to establish appropriate probability models. While there are plenty of statistical tools devoted to specification testing and specification search, the logical role of probability models in econometric inference is a relatively neglected topic.<sup>6</sup>

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<sup>6</sup> However see Hendry (1995), esp. chs. 1 and 15, Juselius (1999), and Johanssen (2006).

I also want to defend a third thesis that is well illustrated by the equivalent probabilistic implications of the causal structures  $A \rightarrow B$ ,  $A \leftarrow B$  and  $A \leftrightarrow B$  (see equations (1)-(8) above): namely, probability models do not in general require causal presuppositions. In saying this, I do not wish to contradict another of Cartwright’s (1989) well-known principles that an output of causal knowledge is delivered only by inputs of causal knowledge. Rather I want to defend the weaker claim that, while prior causal knowledge may be useful and, sometimes at least, essential, some causal claims may be supported by facts about probability models that do not depend on assumptions about the truth of these very same causal claims.

To illustrate, consider a simple causal structure:  $A \rightarrow C \leftarrow B$ , where

$$(10) \quad A \leftarrow \varepsilon_A,$$

$$(11) \quad B \leftarrow \varepsilon_B,$$

$$(12) \quad C \leftarrow \alpha A + \beta B + \varepsilon_C,$$

and  $\varepsilon_i \sim$  independent  $N(0, \sigma_i^2)$ ,  $i = A, B, C$  and  $\text{cov}(\varepsilon_i \varepsilon_j) = 0$  for all  $i \neq j$ . The arrangement is one with a *common effect* of two independent causes, sometimes known as an *unshielded collider* (Spirtes, Glymour, Scheines 2000, p. 10). The causal structure itself cannot be directly observed, but we can observe realizations of the variables ( $a_j$  for  $A$ ,  $b_j$  for  $B$ , and  $c_j$  for  $C$ , where  $j = 1, 2, \dots, N$  indexes particular observations).

Now suppose that we want to infer the causal structure from the data. We begin by trying to establish a probability model of the data. A normal distribution is often a good place to start. There are standard statistical tests for normality. The joint normal model of three variables can be described by nine parameters: three means (call them  $\bar{A}, \bar{B}, \bar{C}$ ), three variances ( $\sigma_A^2, \sigma_B^2, \sigma_C^2$ ), and three covariances or, equivalently, population

correlations  $(\rho_{AB}, \rho_{AC}, \rho_{BC})$ .<sup>7</sup> A particularly simple model of the data assumes that the three variables are distributed independent normal with constant means and variances:

Model 1 
$$(A, B, C) \sim N(\bar{A}, \bar{B}, \bar{C}; \sigma_A^2, \sigma_B^2, \sigma_C^2; 0, 0, 0),$$

where the last three arguments indicate that each of the population correlations is zero.

Probabilistic independence can be defined formally as occurring when

$P(X, Y) = P(X)P(Y)$ . Informally, it means that the probability distribution of a variable is the same whatever realization is taken by another variable. Probabilistic independence implies that the corresponding population correlation is zero; a population correlation of zero does not, in general, imply probabilistic independence, though the cases in which the two diverge are fairly special. Thus, Model 1 stated as a probability distribution is actually slightly weaker than the intended relationship that the three variables be probabilistically independent, not just uncorrelated.

Each probability model sees the data from a point of view. Is Model 1 a good model? The answer, of course, depends in part on our purposes. If we, subscribe to an inferential scheme that requires judgments about probabilistic dependence (for example, the various causal-search algorithms in Peter Spirtes, Clark Glymour, and Richard Scheines (2000) or Judea Pearl (2000)), then it is not a good model, since it *assumes* that there is no probabilistic dependence. A better model would be

Model 2 
$$(A, B, C) \sim N(\bar{A}, \bar{B}, \bar{C}; \sigma_A^2, \sigma_B^2, \sigma_C^2; \rho_{AB}, \rho_{AC}, \rho_{BC}).$$

There is no loss from taking this point of view, since Model 2 nests Model 1: if we decide that on our best estimates  $\rho_{AB} = \rho_{AC} = \rho_{BC} = 0$ , then Model 2 collapses to Model 1.

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<sup>7</sup> The relationship of the population correlation to the covariance is, for example,  $\rho_{AB} = \frac{\text{cov}(A, B)}{\sigma_A \sigma_B}$ .

Whether a model is good also depends on its relationship to the data. Various statistical techniques allow us to estimate – that is, to assign values to – the parameters of Models 1 and 2. It is not to our purpose to discuss them in any detail. Other statistical techniques test whether the data appear to be normal, based on the parameter estimates. And others allow us to test the hypotheses that the population correlations are individually or jointly zero (in the later case, we might accept Model 1).

The assertion that a probability model truly describes some portion of the world is a conjecture from which we can deduce that the model accounts for the co-occurrences of the data (both observed and yet-to-be-observed) except for some random residual. We can define “random,” as Hacking (1965, p. 119) does as the property of probabilistic independence among the terms of a series. There are various ways that independence can fail and various statistical tests to check for its failure, so that judgments of randomness are always relative to some set of statistical tests. Conjectures about probability models, like all scientific conjectures, are accepted because they are supported by the right kind of data. They are never deductively certain, and they always remain open to doubt and criticism. Serious criticisms must be adjudicated in the light of the data and may lead to a reassessment of the appropriateness of a probability model. The crucial point is that the probability model is not a directly observable fact about the frequencies displayed by the data; rather it is a conjecture, the support for which depends on a complex of statistical inferences.

Returning to our illustration, suppose that we have obtained estimates for the parameters of Model 2, we have tested it for normality, and we have tested and rejected Model 1 as a special case. (These by no means exhausts all that the statistician might do

to convince himself that Model 2 is a good model.) Now, here is a principle invoked in many causal search algorithms:

**Principle of the Common Effect:** if  $X$  and  $Y$  are unconditionally probabilistically independent, but are probabilistically dependent conditional on  $Z$ , then  $Z$  is the common effect of  $X$  and  $Y$  (or  $Z$  forms an unshielded collider on the path  $XZY$ ).

I do not wish to defend this principle here, but instead consider the logic of its application.

We start with an estimate of Model 2. Suppose that a statistical test tells us that we cannot reject  $\rho_{AB} = 0$  and that the correlation of  $A$  and  $B$  conditional on  $C$  does not equal zero ( $\rho_{AB|C} \neq 0$ ). Then, we are working with a particularization of Model 2, call it

Model 2' 
$$(A, B, C) \sim N(\hat{A}, \hat{B}, \hat{C}; \hat{\sigma}_A^2, \hat{\sigma}_B^2, \hat{\sigma}_C^2; 0, \hat{\rho}_{AC}, \hat{\rho}_{BC}),$$

where, in the custom of econometricians, the “hats” indicate estimated values and the estimates  $\hat{\rho}_{AC}$  and  $\hat{\rho}_{BC}$  are constrained to fulfill the condition  $\hat{\rho}_{AB|C} = 0$ .<sup>8</sup>

Probabilistic dependence is a property of probability distributions and not of realized data. The important judgments here are  $P(AB) = P(A)P(B)$  and  $P(AB|C) \neq P(A|C)P(B|C)$ . The crucial point is that these are deductive consequences of Model 2' and are not unmediated consequences of observed data  $(\{a_j, b_j, c_j | j = 1, 2, \dots, N\})$ . This is easily seen by noting that Model 1 is, for other purposes and from other points of view, a perfectly acceptable model of the data; and Model 1 does not imply  $P(AB|C) \neq P(A|C)P(B|C)$ . Given Model 2', we can *deduce* that the antecedents of the Principle of the Common Effect are fulfilled and, therefore,

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<sup>8</sup> Which requires in particular that  $(\hat{\rho}_{AC}^2 - \hat{\rho}_{AC}\hat{\rho}_{BC})(\sqrt{1 - \hat{\rho}_{AC}^2}\sqrt{1 - \hat{\rho}_{BC}^2})^{-1} = 0$ , so that, given  $\hat{\rho}_{AB} \neq 0$ , any nonzero values for both  $\hat{\rho}_{AC}$  and  $\hat{\rho}_{BC}$  are sufficient.

conclude that the data, through the mediation of Model 2', imply  $A \rightarrow B \leftarrow C$ , which we know by assumption is the causal structure that generated the data.

There are two points to take away from this illustration. The first is that an inference such as the one from the Principle of the Common Effect is a two-step process. Step 1 establishes the probability model through statistical inferences; step 2 deduces the probabilistic (in this case, causal) consequence from the inferential principle applied to the probability model. Commentators on causality frequently seem confused on the two-step nature of the inference because the parameters of common probability models frequently have easily calculated analogues among descriptive sample statistics. For example, Pearson's sample correlation coefficient  $r_{XY}$  is analogue to  $\rho_{XY}$ .<sup>9</sup> One cannot, however, work directly with the sample correlation coefficient or other descriptive statistics without reference to the probability model. a) Parameterization is distribution-specific; some distributions may have no parameter closely related to  $\rho_{XY}$  in the normal distribution and, so, nothing to which  $r_{XY}$  can serve as an analogy. b) Even when the analogy holds,  $r_{XY}$  may not coincide with the best estimate of  $\rho_{XY}$ , since the parameters of a probability distribution are typically estimated jointly (e.g., by maximum likelihood methods) rather than individually. c) Sample descriptive statistics are calculated without the aid of a probability distribution, and it is only through one or other distribution that they can have any bearing at all on probability – to act otherwise is to commit a category mistake.

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<sup>9</sup>  $r_{XY} = \left( \sum_{j=1}^N (X_j - \bar{X})(Y_j - \bar{Y}) \right) / \left( \sum_{j=1}^N (X_j - \bar{X})^2 \sum_{j=1}^N (Y_j - \bar{Y})^2 \right)^{1/2}$  and  
 $\rho_{XY} = E((X - E(X))(Y - E(Y))) / (E(X - E(X))^2 E(Y - E(Y))^2)^{1/2}$ .

While I do not believe that proponents of causal-search algorithms, such as Spirtes *et al.*, are confused on the two-step nature of the inferential process – their inferences are clearly defined relative to facts about probability distributions and not sample statistics – their way of describing the algorithms typically elides the step from facts about frequencies to the probability distribution and gives the impression that the linkage is direct from data to causal model. In effect, the algorithms test various hypotheses, leading to particularizations of the probability distribution, similar to the move between Model 2 and Model 2', while simultaneously drawing inferences about probabilistic dependence and causal order from the more general probability distributions that are robust to further particularizations. In the end, they have produced simultaneously an inferred causal structure and described the data-supported probability distribution from which it can be deduced. But the temporal simultaneity of this process should not be allowed to obscure the logical priority of statistical inference to a probability distribution over both deductions of relationships of causal dependence and, in turn, of causal structure.

The principal interest of most researchers with respect to causal search is in the second step of inferring causal structure from patterns of probabilistic dependence. They frequently take for granted that the problem of justifying a particular probability model from the data has been (or can easily be) solved. Clark Glymour (1999, pp. 73-74, quoted by Reiss 2007, p. 189) is perhaps somewhat cavalier when he writes:

Applying the [causal search algorithm] to real data requires a lot of adaptation to particular circumstances: variables must often be transformed to better approximate normal distributions, decisions must be made about modeling with discrete or continuous variables, data must be differenced to remove autocorrelation, and so on and on.



Glymour’s statement is open to the (surely unintended) interpretation that data preparation is just a matter of applying some cookbook techniques to prepare the raw material for further processing, obscuring the fact that inferring an appropriate probability model from data is a subtle statistical problem of the first order and that the search algorithms logically operate on facts about the distributions rather than on the data directly. Lack of clarity on this point, as I will show presently, contributes mightily to misunderstanding and misprizing causal search algorithms and, indeed, graph-theoretic causal analysis more generally.

The second point to take away from the illustration is that, in inferring the pattern of causal dependence from which causal order is itself inferred, we nowhere refer to the facts about causal structure that form the endpoint of our inferential chain (namely, the connection of  $A$  and  $B$  to their common effect  $C$ ). That is not to say that we do not use causal knowledge at all. In restricting our model to three variables, we have implicitly judged that none of the other causal connections that our three variables has is structured in such a way as to interfere with the appropriateness of Models 2 or Model 2’. Such a judgment may, of course, be challenged, suggesting further investigation. That we cannot step out of a causal context notwithstanding, the key point is that we have begged no question.

People often intuitively think of probabilistic dependence as a causal notion.

Hacking (1965, p. 20) writes:

Two events are commonly said to be independent of each other if neither causes the other, and if knowledge that one occurred is no aid in discovering if the other occurred.

But Hacking also agrees that  $X$  and  $Y$  are independent if  $P(XY) = P(X)P(Y)$ . Not only is no assumption about causation cited in this statement, the statistical tests of independence are based on patterns of co-occurrence without causal reference. Whether knowledge of one aids in discovering whether the other occurred depends importantly on what knowledge we have in mind and what we mean by “aids.”

Generally, two watches give knowledge of each other: if I know that my watch says 2:39 PM, then it is a fair bet that my neighbors watch is pretty close. My watch even gives me knowledge of what a watch in Australia is likely to read, knowing the difference in time zones between the east coast of the United States and, say, the west coast of Australia. And although we do not have a common reference point, I suspect that, if there were Martians and Martians had watches, then an hour passed on my watch would be an hour passed on a Martian’s watch (suitably adjusting Martian units to our own). But generally, it is a well-supported conjecture that my watch is probabilistically independent of my neighbor’s, the Australian’s, and the Martian’s watches.<sup>10</sup>

For example, take the standard time signal provided by the U.S. National Institute of Standards and Technology and the U.S. Naval Observatory as a reference time.

Define the random variable  $A$  = the difference between the time on my watch and the reference time and  $B$  = the difference between the time on my neighbor’s watch and the reference time. I maintain that typically we will find a well-supported probability model in which  $P(AB) = P(A)P(B)$ . More directly, if  $b$  is a particular realization of  $B$ , then we will find  $P(A|B = b) = P(A)$ . That watches convey knowledge about the likely behavior of other watches and clocks explains their widespread use. That watches are typically

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<sup>10</sup> Cf. Steel (2003), section 2, and Reiss (2007), p. 181.

probabilistically independent of each other explains why, when the power has been cutoff, we can usefully look to our wristwatches to reset the clock on the microwave.<sup>11</sup>

Contrary to Hacking, it would be more accurate to say that

*two random variables are independent of each other if the realization of one conveys no information about the distribution of the other.*<sup>12</sup>

Formulated this way, probabilistic independence does not invoke causal order conceptually, nor do statistical tests of independence presuppose causal order.

### III. The Principle of the Common Cause

The importance of clarity with respect to the two-step inferential process – from data to probability model, from probability model to causal structure – is thrown into relief by recent discussions of the Principle of the Common Cause, a version of which lies at the heart of the graph-theoretic analysis of causal structure and related search algorithms.

Hans Reichenbach (1956, p. 157) provides the original statement:

**Principle of the Common Cause (Reichenbach):** “If an improbable coincidence has occurred, there must exist a common cause.”

Julian Reiss (2007, p. 184) gives a version, which he attributes to Hoover (2003, p. 548), that is clearer for the issues to hand:

**Principle of the Common Cause (Hoover):** “If variables  $X$  and  $Y$  are probabilistically dependent . . . , then either  $X$  causes  $Y$  or  $Y$  causes  $X$ , or  $X$  and  $Y$  are the joint effects of a common cause.”<sup>13</sup>

Reiss, however, states the principle mainly to criticize it.

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<sup>11</sup> I say “typically,” because, for example, two old-fashioned electric clocks on the same circuit that use the cyclicity of the household electricity to control the speed of their motors may be probabilistically dependent after all.

<sup>12</sup> And two *events* are independent of each other if each is a realization of a mutually independent random variable.

<sup>13</sup> Reiss states he needs to modify my statement of the principle to make it consistent with his own paper. I agree that this formulation is better than my original formulation, which was specific to a particular context.

The background for Reiss’s criticisms is Elliot Sober’s (1994, 2001) putative counterexample.<sup>14</sup> In Sober’s example, bread prices in England and sea levels in Venice are both rising and *ex hypothesi* not causally connected. In a sense that is less than perfectly clear, Sober maintains that bread prices and sea levels are correlated and, therefore, probabilistically dependent, which contradicts the Principle of the Common Cause.

Reiss categorizes various reactions to Sober’s counterexample as following one of two strategies: the first strategy argues that Sober’s claim that there is a genuine probabilistic dependence between bread prices and sea levels is defective; the second proposes to defuse the counterexample by showing that it fails to apply to data when they are appropriately prepared.<sup>15</sup> An example of the second strategy would be to apply the Principle of the Common Cause only to the first differences of trending data. Reiss treats these two strategies separately, but observes that they may be complementary. I would put the point more strongly: if “data preparation” (second strategy) is understood appropriately and if the first strategy is stated in its positive form (patterns of probabilistic dependence may support causal inference when genuine), then there is no legitimate way to separate the two strategies, for the second is part of establishing the *bona fides* of the probability model necessary for the first. Although Reiss (2007, p. 186) also claims that the two steps of the inferential process cannot always be separated, his illustrations imply that there is a route directly from the data to a causal conclusion that employs the Principle of the Common Cause without the mediation of a probability

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<sup>14</sup> Hoover (2003) offers a detailed refutation of Sober’s counterexample. While I remain convinced of its argument, at some key points the exposition apparently misled some readers about its essence. I hope to be clearer here.

<sup>15</sup> Reiss offers Hoover (2003) as an example of the first strategy and Spirtes *et al.* (2000), Steel (2003), and Arntzenius (2005) as examples of the second.

model. Where I argue that Reiss’s two strategies are – rightly interpreted – elements of a single strategy, Reiss denies that the step of establishing a probability model, which I regard as key to that single strategy, is necessary at all.

Although Sober does not offer any formal measures of correlation between bread prices and sea levels, he does provide some cooked data (Sober 2001, p. 334) and notes that “higher than average sea levels tend to be associated with higher than average bread prices” (p. 332). Unlike Reiss, Sober (2001, p. 343) acknowledges the two-step inferential process involved in applying the Principle of the Common Cause; so, we are entitled to ask what the sample association of bread prices and sea levels says about probabilistic dependence. Note that on a common measure of sample association, Pearson’s correlation coefficient ( $r$ ), which was previously defined in footnote 8, bread prices ( $B$ ) and sea levels ( $S$ ) are highly correlated ( $r_{BS} = 0.99$ , where  $-1 \leq r \leq 1$  and  $|r| = 1$  indicates perfect correlation, whereas  $r = 0$ , indicates no correlation.). We cannot interpret this high correlation in terms of probability without a probability distribution. This is obvious, since it is exceedingly rare to find a correlation coefficient that is *exactly* zero; we must judge whether it is *effectively* zero or not relative to an assumed probability distribution.<sup>16</sup>

The stationary, multivariate normal distribution is the workhorse of statistics. It has the nice property that  $r$  is an analogue for its population parameter  $\rho$ , and that it can be shown that, as the sample size increases, the expected value of  $r$  converges to  $\rho$ . Roughly speaking, a distribution is stationary when its moments (mean, variance, and higher moments) are constant through time. But a stationary distribution is not a good

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<sup>16</sup> Notice that this is true whether we accept classical statistical testing or a decision-theoretic approach.

model of Sober’s data. A stationary distribution implies that a time-series will cross and re-cross its sample mean frequently. Sober’s data cross their sample means only once. While this is a nice clue, there are also formal tests for non-stationarity. There are a number of non-stationary alternatives to the stationary, multivariate normal distribution – none of which display the correspondence between the sample correlation coefficient  $r$  and a fixed population parameter, like  $\rho$ .

One alternative is the random-walk in which the best expectation of the value of a time series at  $t+1$  is its value at time  $t$ . If the data were generated by two probabilistically independent random walks, then  $r$  would be a worse-than-useless measure of probabilistically dependence; for the expectation of  $r$  as the sample size grows converges not to a single value but to a uniform distribution over the interval  $-1$  to  $1$  (Hendry 1995, p. 128). This means that when the world is populated by random walks that it is easy (and meaningless) to find high levels of sample correlation among some of them.

The paradigm random walk can be expressed as:

$$(13) \quad X_{t+1} = X_t + \varepsilon_t$$

or equivalently as

$$(13') \quad \Delta X_{t+1} = \varepsilon_t,$$

where  $\varepsilon_t$  is a stationary random error term (e.g., normal). The form (13') suggest to some commentators (e.g., Forster 1988; Papineau 1992; Hausman and Woodward 1999) a quick fix. If we difference the non-stationary time series  $X$ , it becomes stationary, and the correlation coefficient between two such differenced, non-stationary time series is an indicator of probabilistic dependence. The problem with this approach is that, while the differences of nonstationary variables may be probabilistically dependent, so may the

levels (even when the differences are not), and differencing the data eliminates the information about this relationship between the levels. Nonstationary variables that display probabilistic dependence in levels are said to be *cointegrated*. If the nonstationary random walk is sometimes illustrated by the path of drunk stumbling aimlessly as he leaves the bar, then cointegration is the situation in which the drunk has a faithful friend who follows at a discreet distance to make sure that he comes to no harm.<sup>17</sup>

Sober’s counterexample “works” only because he trades on our implicitly judging probabilistic dependence against a probability model in which casual measures of association have a natural interpretation. But even at a casual level, it is obvious that a stationary probability model is not a good characterization of the data. And in any non-stationary model, the sample association of bread prices and sea levels is both natural and not indicative of probabilistic dependence. The situation is exactly like the association between time kept on two watches: rising bread prices in England give some indication of sea levels in Venice, but the distribution of sea levels is the same whether the current realization of bread prices is a rise or a fall.

The point is not that any particular non-stationary model fits Sober’s counterexample. Rather it is that we must establish the probability model before we can make any judgment of probabilistic dependence. Sober may acknowledge the two-step process, but he fails to do the work – either statistically by testing the data or hypothetically by establishing the true distribution in his thought experiment – necessary to move from the first to the second step. What Reiss thinks of as data preparation is integral to establishing the probability model from which probabilistic dependence is ascertained. On the one hand, the probability model must be appropriate to the data; on

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<sup>17</sup> See Hoover (2003), sections 4 and 5, for a more technical exposition.

the other hand, the probability model helps to guide the meaningful preparation of the data. This is crucial work for statistics or econometrics, though it is typically neglected in discussions of the Principle of the Common Cause.

In contrast to my analysis of Sober’s counterexample, which is not so much a defense of the Principle of the Common Cause as a demonstration that it does not fail for Sober’s particular reasons, Reiss offers a defense of the principle – a defense which falls into the category of “destroying-the-village-in-order-to-save-it.” Reiss agrees with Cartwright and Hacking that probabilities (i.e., frequencies that are correctly described within the canons of an axiomatization of the behavior of random variables) arise only in well-constructed chance setups. At the same time, he objects to the two-step inferential process: defining (as I do) the Principle of the Common Cause as connecting causal structure directly to facts of probability and only indirectly to facts of sample frequencies “deprives the principle of much of its inferential power and to some extent betrays the motivation behind it” (Reiss 2007, p. 185). Reiss defends the principle as a sometimes useful heuristic, providing what he refers to as an “epistemological reading” as opposed to the “metaphysical reading” implicit in the two-step inferential process.

The contrast between an epistemological and a metaphysical reading is spurious. The two-step process is about inference (that is, classically epistemological) and says nothing about what causation actually is. Reiss correctly sees that the English word “probable” is broader than probability considered as the formal theory of random variables (cf. Hacking 1965, pp. 10-11). Yet, it is hard to see that grounding the principle in such a theory is a betrayal of the motivation behind the Principle of the Common Cause. Reichenbach (1956) illustrates his principle with some homely and informal



examples of token- (as opposed to type-) causation. Yet the whole discussion is embedded in a formal, frequentist, and axiomatized account of probability. It is possible that there are inferential principles that rely on common causes in which probability is not understood stochastically (see Aviezer Tucker 2007, who argues similarly to Reiss that the principle is both heuristic and invokes a wider sense of probability). Since econometrics, graph-theoretic search algorithms, and the central literature on the Principle of the Common Cause appeal only to the stochastic interpretation, we will not consider any such wider principle here. And despite Reichenbach’s illustrations, we leave consideration of token causation for another day (although see Hoover 2001a, ch. 4 for some thoughts).

Reiss’s strategy is explicitly analogous to Patrick Suppes (1970) well-known probabilistic analysis of causation. Suppes begins by defining *prima facie* cause as the case in which  $P(A|B) > P(A)$ . He then tries to catalogue the cases in which *prima facie* causes fails to correspond to actual cause and to suggest appropriate corrections. In parallel, Reiss takes the Principle of the Common Cause as providing a rule for inferring *prima facie* cause, and then catalogues a (partial) list of exceptions to the rule. Reiss’s heuristic rule is not stated as a relationship of causal structure to probabilistic dependence, but as a relationship of sample association (or frequency) to causal structure, thus short-circuiting the first step of the two-step inferential process:

**Principle of the Common Cause (Reiss):** “The proposition  $e =$  ‘Random variables  $X$  and  $Y$  are (sample or empirically) correlated’ is *prima facie* evidence for the hypothesis  $h =$  ‘ $X$  and  $Y$  are casually connected.’ If all alternative hypotheses  $h_i^a$  (e.g., ‘the correlation is due to sampling error,’ ‘the correlation is due to the data-generating process for  $X$  and  $Y$  being non-stationary,’ ‘ $X$  and  $Y$  are logically, conceptually, or mathematically related’) can be ruled out, the  $e$  is genuine evidence for  $h$ .” [Reiss 2007, p. 193]

It is worth noting that, unlike Reiss, Suppes’ notion of *prima facie* cause is grounded in the assumption that an empirically supported probability model characterizes the data. Reiss will have none of it because he believes that informal probability claims are legitimately made without reference to such models or to the chance setups that would support them. Unfortunately, since the list of exceptions is open-ended, animated by no principle that would assure us that the exceptions all fall into a manageable number of classes, we can never know that  $e$  supports  $h$  on Reiss’s version of the principle.

It is instructive to see how the two-step inferential process handles Reiss’s exceptions. We have already discussed the case of non-stationary data. Consider some others:

- *Colliders*. Reiss (2007, pp. 187-188) takes the causal configuration  $A \rightarrow C \leftarrow B$  as an exception to the Principle of the Common Cause, because the correlation of  $A$  and  $B$  conditional on  $C$  does not indicate their causal connection (see equations (10)-(12) above for the structure of the probability model). At first this seems clearly wrong: the Principle of the Common Cause begins with the claim that  $A$  and  $B$  are unconditionally *correlated* (whereas the Principle of the Common Effect begins with them unconditionally *uncorrelated*); hence the antecedent of the Principle of the Common Cause is not fulfilled, so it fails to provide a counterexample. What Reiss has in mind, however, is that the data on  $A$  and  $B$  may be collected in such a way that, without knowing it, we observe them only conditional on  $C$ , so that they appear to be unconditionally correlated.

A real example illustrates Reiss’s concern.<sup>18</sup> Data on child molestation

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<sup>18</sup> Sher and Carey (2007).

and exposure to child pornography was collected from prisoners in jail for possession of child pornography. Eighty-five percent admitted to having molested children. Let  $A$  = viewed child pornography,  $B$  = molested children, and  $C$  = incarcerated for possession of child pornography. A concern immediately expressed by various critics of the study amounts to asserting the possibility that the variables form an unshielded collider and that  $A$  and  $B$  are correlated only because the mode of data collection implicitly conditions on  $C$ , the fear being that those who *both* view child pornography and molest children are more likely to be incarcerated for possession than those who merely view child pornography and that viewing it and child molestation may be unconditionally independent.

The intuition of the critics of the study can be interpreted with the two-step inferential process as the requirement that we get the right probability model, which means seriously entertaining the criticism and widening the scope of the data collection, so that the alternative hypothesis of an unshielded collider can be assessed. This strategy is suggested by a wider understanding of the world. But that is not an objection; there is nothing in the two-step process that suggests that a probability model is a black box for processing statistics without reference to their nature and provenance nor that only statistical criteria can be used to support a particular probability model. It is possible that sometimes we may make a mistake and not notice accidental conditioning on an unobserved variable. Reiss persistently confuses the epistemic with the practical. It is nonsense to attack an inferential principle as metaphysical and not epistemic because we sometimes make errors in practice. The right response to accidental conditioning is to try to

use all our knowledge to anticipate situations that give rise to such errors, to criticize research and to respond to criticism, and to test, test, test.

- *Mixing*. Simpson’s paradox refers generically to cases in which correlations in a sample vanish or reverse sign in subsamples. For example, professorial salaries in a university may be negatively correlated with being female and, yet, uncorrelated within every constituent department (see Hoover 2001a, pp. 19-22 and the references therein). This might arise because salaries and the distribution of the sexes may differ among departments. Reiss (2007, p. 185) lists such mixing of populations as an exception to his heuristic principle, since in such a case, the application of the Principle of the Common Cause to the whole sample would produce a misleading conclusion.

The advocates of the data-preparation strategy have suggested that the principle can be applied once one has segregated the data appropriately. Sober (2001, p. 333) rejects this approach as question-begging, suggesting that causal knowledge is invoked to effect the segregation of the data. The two-step process is sympathetic to the idea of segregating the data, but sees it in terms of the probability model. A probability model that assumes a homogeneous population is not as well supported by the data as one that accounts for the heterogeneity. The model with a homogenous population can be tested statistically against one with a heterogeneous population in the same way that Model 2 could be tested against Model 1 (see section I). Such tests do not refer in any way to the causal structure that might be inferred by applying the Principle of the Common Cause to the probability model, so that (*pace* Sober) no questions are begged.

- *Stationary Nonsense Correlation.* Following G. Udny Yule (1926) correlations that appear to be causally meaningless are referred to by statisticians and econometricians as *nonsense correlations*. The correlations between independent nonstationary variables (e.g., random walks) provide one example. Reiss (2007, p. 191) cites Clive W.J. Granger, Namwon Hyung, and Yongil Jeon (2001), who provide an example of *stationary* nonsense correlations. These can arise when time series data are highly serially correlated (i.e., revert to their means after a shock only very slowly). While differencing a pair of pure random walks, in the form of equation (13), will eliminate the nonsense correlation between them, differencing highly serially correlated series leaves it intact.

Granger *et al.* (2001, p. 903) provide the solution that suits the two-step inferential process:

Spurious relationships occur from a misspecified model, under the null. If  $X_t$  is serially correlated in Equation 1 and the true  $\beta$  [which measures the population correlation] is zero, this implies that the residuals cannot be white noise. The problem is thus that Equation 1 is a misspecified model. *It is resolved by improving the specification*; that is by adding more lagged dependent, and possibly lagged independent variables. [emphasis added]

We need to do the statistical work of establishing a well-supported probability model. The nature of the model differs from case to case (a model which accounts for stationary serial correlation is different from one that accounts for a pure random walk). A battery of statistical tools exists to determine what sort of probability model is appropriate.

- *Homoplasy.* Citing Sober (2001, pp. 335 ff.), Reiss (2007, p. 191) considers homoplasies, cases of independent evolution of organs such as wings or fins, as an examples of correlations that lack a common causal explanation (cf. Tucker

2007, p. 445).

Setting aside the possibility that some non-stochastic version of probability or some non-probabilistic version of the Principle of the Common Cause applies (a strategy that appeals to both Reiss and Tucker), we cannot reject out of court that apparent homoplasies are probabilistically dependent through some deep background variables. Analogously, bread prices in England and sea levels in Venice might be probabilistically dependent on global warming. We reject this only because Sober’s counterexample is cooked; he gets to set the rules; and ruling out such a dependency is what makes it a counterexample. Homoplasies in contrast are not cooked examples, and ruling out the possibility by assumption is simply question begging. Yet, even if there is no such deeper connection, an evolutionary sequence may well resemble another, just as the sequence of times on different watches or the graphs of bread prices and sea levels resemble each other, without the distribution of either one being different for different realizations of the other.

- *Non-statistical Sources of Nonsense Correlations.* Reiss (2007, pp. 191-192) argues that day is sample-correlated with night, that  $X$  is sample correlated with  $\log(X)$ , and that, if savings is definitionally what is not consumed out of national income, then consumption will be correlated with savings; yet, none of these relationships indicates a genuine causal relationship.<sup>19</sup>

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<sup>19</sup> The example gets its force, not from the definitional connection, but from the jointness of the consumption/savings decision in real life. The mere presence of an identity is not decisive. For example, contrary to what economists usually assume, let consumption be  $C \sim N(c, \sigma_c^2)$  and savings be  $S \sim N(s, \sigma_s^2)$  with  $\text{cov}(C, S) = 0$ . Income ( $I$ ) may still be defined as  $I \equiv C + S$ . Both  $C$  and  $S$  will be correlated with  $I$ , but they are not correlated with each other. The situation is a kind of deterministic unshielded collider.

The two-step inferential process would, as always, ask what sort of probability model is supported by the data. In each case, the best is degenerate in that it assigns probability 1 to some correlation. (The correlation between  $X$  and  $\log(X)$  would require a nonlinear model. Once we open our minds to the possibility of a nonlinear relationship, appropriate statistical tests are available.) Probability 1 is an indicator either of a deterministic relationship, which at least under some theories of causality might be causal, or of an identity (as in these cases). Any investigator would have to draw on subject-matter knowledge or conceptual analysis to decide which. But the need to decide is no threat to the Principle of the Common Cause when the principle cannot be invoked until we have an appropriate probability model.

Again, the two-step process does not require that the investigator be a cipher who, without any context, uses statistical tools to process anonymous frequencies into causal outputs. No serious investigator would intentionally model the probabilistic dependency of  $X$  on  $\log(X)$  or embed an exact identity into a regression equation. It sometimes happens inadvertently – as many students can attest – and in some of its variants even has a name (e.g., “the dummy variable trap”), but typically the statistics themselves shout out “PROBLEM!” For example, a regression with an exact dependency among dummy variables (a good model for Reiss’s day follows night correlation) simply will not compute, since one of its matrices is singular and cannot be inverted. And if the statistics do not immediately flag the problem, critics may raise the possibility, inviting statistical investigation that reveals the problem.

The likelihood of inadvertently treated identities as causally significant dependencies is greater when there is measurement error. For example, conceptually national income and national expenditure are equal at all times; but, since they are constructed from different sources of data, they may appear to be correlated. So what? The economist has to think about the conceptual nature of his variables and modeling measurement error is just another species of the genus statistical modeling, which is the work of the applied researcher.

We have exhausted Reiss’s examples of exceptions to the Principle of the Common Cause.<sup>20</sup> From the perspective of the two-step inferential process, we can see each exception as a failure to establish a well supported probability model. In rejecting the regulatory role of the probability model, Reiss is left without any guiding principle for what constitutes an exception. Instead, he ends up claiming that his heuristic version of the Principle of the Common Cause works well, . . . except when it does not. The list of exceptions is open-ended.

#### **IV. Cartwright’s Pessimism Confounded**

We have not offered a general defense of the Principle of the Common Cause. Instead, we have demonstrated that certain ways of attacking it illustrate the general proposition that we cannot neglect the need to provide a convincingly supported probability model if we wish to draw probabilistic conclusions from data. Reiss’s “defense” of the Principle of the Common Cause goes wrong because he accepts simultaneously two premises: 1) Cartwright’s view that only tightly controlled nomological (or socio-economic) machines

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<sup>20</sup> Actually, we have not addressed Reiss’s (2007, p. 185) example of laws of coexistence. But we have addressed enough to show the open-ended and heterogeneous nature of the exceptions and to illustrate the strategy that is implied by the two-step inferential process for dealing with them.



generate frequencies that can be modeled probabilistically; and 2) that something like the Principle of the Common Cause is used – as a matter of fact – in actual research.

Omitting the step of establishing a probability model, the conditions for which, Cartwright has argued, are too severe to be met in many practical contexts, is a fairly desperate move. Reiss’s defense ends with the “irony” that once his open-ended list of exceptions has been taken on board that we had better stick to experiments and eschew passive observation (Reiss 2007, p. 194). In the name of strengthening the practical applicability of the principle, Reiss kills it. His first premise is the mortal enemy of his second premise. In the end, he is left exactly where he started with Cartwright’s view that the scope of probability models is very narrow and almost entirely restricted to experimental contexts.

Cartwright’s position is deeply pessimistic with respect to economics and other sciences that rely on passive observation. Is it justified? Cartwright’s argument points to a substantial disanalogy between experiments and passive observation. The disanalogy is real enough; it is the bane of empirical economics. But there is also a strong analogy between experiments and passive observation, which was central to Haavelmo’s analysis sixty years ago.

Cartwright stresses that experiments generate probabilistically well behaved data (and, generally, display nature’s capacities) only when they are arranged “just so.” There is a danger of overstating the case. In a discussion of coin-flipping machines, Cartwright (1999, p. 166) says:

Imagine . . . that we flip the coin a number of times and record the outcomes, but that the situation of each flip is arbitrary. In this case we cannot expect any probability at all to emerge.

Approach this claim empirically: take any coin in general circulation and sit down anywhere and flip it how you will, recording heads and tails. Having done the experiment, I am sure that over the course of, say, 200 flips your data will conform – as judged by standard statistical tests – to a binomial probability model with a probability  $\frac{1}{2}$  for heads.

It matters that on a typical flip, the coin rotates at least once or twice. It matters that you do not wait to decide how to record the coin until you can see its resting position clearly. (For example, if a coin is leaning against the leg of table showing heads, and you decide after you see it that the rule for that particular flip will be to turn its visible side down before reading it, then the implicit preference for tails – if it persists in other such cases – may skew the results.) It does not matter whether you catch the coin in midair and turn it over on your wrist before reading it or let it fall to the floor or pick it out of the crack between the cushions on the sofa or fish it out from under the table. It does not matter whether the coin is new or worn or clean or dirty. The frequencies displayed by coins are very robust.

The point is not that every capacity nor every probability is similarly robust. It is quite difficult to construct a machine that will robustly deliver any probability other than zero for the frequency of a coin falling on its edge. Rather, the point is that it is a mistake to assert that a *very high* degree of control is an *a priori* requirement of frequencies conforming to stable probability models. Whether they do or do not is just something that we have to learn about the world in particular cases.

Even in controlled experiments, the range of factors that we attempt to control are frequently quite limited. Partly because we judge that certain factors are irrelevant and

partly out of ignorance, many factors are left to nature’s whims. And when we (or some other researcher) “replicates” our experiment, we cannot set every control in precisely the same way, and whimsical nature picks different values for factors that we have left uncontrolled. To paraphrase Heraclitus: you cannot perform the same experiment twice. At best each experiment is a model of its fellows. As with all models, we have to ask, is it a good or successful model? We learn from experience and from diagnostic tests whether we have successfully implemented controls of the right type and what we may neglect or ignore. Sometimes we find out later that we were wrong and that a neglected or overlooked factor is essential to the result, so that our experiment needs to be reinterpreted, redone, or set aside as uninformative.

The situation is not different in kind from what we face in formulating probability models for passively observed data. We may construct a chance setup and discover that it is reliable. Equally, we may observe the world and discover that there is a way of modeling it that reliably acts like a fabricated chance setup would. Hacking (1965, p. 1) introduces the notion of a chance setup and promptly illustrates it with a passively observed example: “the frequency of traffic accidents on foggy nights in a great city is pretty constant.” The controls (day or night, foggy or clear, in the city or not) are of the same nature as the ones that find their ways into econometric models. The controls may be represented in coarse or finely delineated categories, as may the category of traffic accidents itself. Whether we need finer controls or more controls or controls of different kind and what kind of probability model should tie them together is a matter – exactly as it is for physically controlled experiments – for experience and testing to reveal.

Passive observation is, in many respects, at a disadvantage in comparison to active experimentation. That fact poses serious challenges for empirical economics. Nevertheless, that the inferential logic of passive observation is not of a radically different kind and that statistics provides many useful tools that help us to specify and test appropriate probability models is grounds for optimism.

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