Causal Modeling and the Statistical Analysis of Causation

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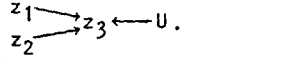
Recent studies on probabilistic causation and statistical explanation (Cartwright 1979; Salmon 1984), I believe, have opened up the possibility of a genuine unification between philosophical approaches and causal modeling (CM) in the social, behavioral and biological sciences (Wright 1934; Blalock 1964; Asher 1976). This unification rests on the statistical tools employed, the principle of common cause, the irreducibility of causation to probability or statistics, and the idea of causal process as a suitable framework for understanding causal relationships. The aim of this paper is to draw attention to these four areas of contact by focusing on the relevant aspects of CM.

1. Causal Modeling

Causal analysis in the social sciences is based on two fundamental notions: model and method. A causal model is an idealized picture of the causal relationships in the world. Method, on the other hand, refers to certain statistical techniques that are used to evaluate and test a causal model using data which consist of joint observations on the model variables.[1] Accordingly, causal modeling is the specification of the hypothesized causal relations among a number of variables as a model and its evaluation by an appropriate statistical technique.

A causal model is expressed in terms of a set of equations and a directed graph. Below is a simple linear and additive model:

(1) $z_3 = p_1 z_1 + p_2 z_2 + U$



It asserts that z_3 is caused by z_1 and z_2, which themselves are not causally related, but may be correlated with each other.

p's are called model parameters and interpreted as measuring the direct causal effects: p_1 measures the causal impact of z_1, and p_2 of z_2, on z_3. More precisely, a unit change in each explanatory variable (z_1 and z_2) produces on the average a change of p_i units in the variable to be explained (z_3). The fundamental idea behind CM is that a variable is a cause of another if and only if the corresponding parameter is significantly different from zero. If the model above is correct, for

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instance, we should find non-zero estimates for both p_1 and p_2. These parameters are, of course, unknown to researchers, and one of their most important aims is to compute reliable estimates of the parameters from sample data.

U is a random variable, called disturbance, which represents measurement errors, and more importantly, all causal factors for z_3 which are not explicitly included in the model. U is needed to have completeness over the model and required to meet certain assumptions, the most important of which is that it is uncorrelated with the explanatory variables. I will call this the U-assumption.

Violation of the U-assumption results in biased estimates for p's. Suppose that U contains a variable z_4 which is correlated with z_1, say. Then the estimate for p_1 will be misleading since, in reality, it is a combination of the influences of both z_1 and z_4 on z_3, and the model will attribute some of the influence of z_4 to z_1. Consequently, the statistical estimation procedure may yield a negative or even zero value for an actually positive p_1. Models in which omitted variables (such as z_4) both cause the variable to be explained (z_3) and are correlated with the explanatory variables (z_1 and/or z_2) are said to be misspecified. As we shall see later, the misspecification problem is Simpson's paradox.

Typically, CM employs two types of linear models: recursive (or path) and non-recursive (or structural equation) models. A model is said to be recursive if the disturbance terms across the model equations are uncorrelated with each other and there are no reciprocal causal connections between any two variables. A model is non-recursive if it is not recursive. For each type of model, there are several different techniques that can be applied. Ordinary least squares regression, path analysis, and the Simon-Blalock method (a special case of path analysis) are all appropriate for recursive models.

A desirable property of recursive models is that they are identifiable; that is, the model parameters can always be estimated. The issue of identification is ultimately linked to the problem of reduction of causation to statistics.

2. Statistical Tools

The most important tool for probabilistic causation and statistical explanation in philosophy has been the use of conditional probabilities. In his S-R model, for example, Salmon (1971) has made much use of the notion of statistical relevance, $P(A/B) \neq P(A)$, and screening-off, $P(A/BC) = P(A/B)$ while $P(A/BC) \neq P(A/C)$. He showed, among other things, that most common-cause structures exhibit the screening-off relations, as in the example of atmospheric conditions rendering irrelevant the association between the occurrence of storms and a drop in barometer reading.

Not surprisingly, the similarities between the probabilistic approach and CM start with these fundamental notions. First, while the former speaks of statistical relevance, the latter speaks of (zero-order) correlation coefficient. The absence of correlation is actually equivalent to statistical irrelevance. By definition of the correlation

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coefficient, ρ_{AB}, for dichotomous variables:

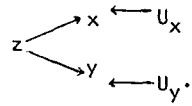
Numerator of $\rho_{AB} \equiv P(AB) - P(A)\,P(B) = 0$ iff $P(A/B) = P(A)$.

The proof easily follows from the conditional probability formula $P(i/j) = P(ij)/P(j)$.

Second, the vanishing of (first-order) partial correlation coefficient, i.e., $r_{xy \cdot z} = 0$, conveys the same idea as screening-off: The association between x and y disappears when z is controlled. In fact, this result can be deduced from a recursive common-cause model:

(2a) $x = p_1 z + U_x$

(2b) $y = p_2 z + U_y$



Multiply each equation by z and take the expected value:

$E(xz) = p_1 E(z^2) + E(zU_x)$

$E(yz) = p_2 E(z^2) + E(zU_y)$.

$E(zU_x) = E(zU_y) = 0$ because of the U-assumption. Also, since all the variables are standardized, $E(z^2) = 1$, $E(xz) = r_{xz}$ and $E(yz) = r_{yz}$. Hence,

(3) $r_{xz} = p_1$ and $r_{yz} = p_2$.

Then multiply (2a) and (2b) side by side and take the expected value as before:

(4) $r_{xy} = p_1 p_2$; or by (3),

(5) $r_{xy} = r_{xz} r_{yz}$.

Finally, by definition of the partial correlation coefficient

Numerator of $r_{xy \cdot z} \equiv r_{xy} - r_{xz} r_{yz} = 0$ by (5).

Thanks to Salmon's recent efforts, the statistical similarities between philosophical theories of causation and CM go beyond these fundamental notions. While earlier Salmon believed that statistical relevance and screening-off suffice to characterize statistical explanation, he now thinks that one actually needs to measure the relevance of each factor that figures in the explanation:

> It is important to know how each factor is relevant--whether positively or negatively, and how strongly both in the population at large and in various subgroups of the population ... .
> The moral is that we need to know not only how the various factors D_k, U_n, F_r ..., are relevant to the outcome, B_i, but how the relevance of each of them is affected by the presence or absence of the others. (1984, pp. 39-40).

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To this end, Salmon adds a new requirement to his old S-R model (or S-R basis as he calls it now), which enables him to derive such conditional probabilities as $P(B_i/AD_kE_n)$. These tell "how each factor is relevant and how strongly" within each cell of the partition of the reference class.

Salmon's new S-R basis is a further step toward making full use of statistical machinery employed by causal modeling. Although Salmon does not explicitly define a measure for weighing relevance, it can be shown that his approach yields numerically the same results as those of CM applied to dichotomous variables.[3]

The overlap between the two approaches not only brings philosophy of science closer to the sciences, but also points to the necessity and usefulness of measuring the impact of one factor upon another. It shows that the precise degree to which A influences B (the computation of which is at the heart of CM) is quite as important as the mere fact of such influence and gives a deeper insight into causal relations.

3. The Problem of Reduction

The complexity of statistical techniques employed in CM and the fact that social scientists seldom discuss the irreducibility of causal relations to statistical ones might give the impression that CM is a sophisticated reductionist program. For example, in a recent article Ellett and Ericson (1983) attribute the following reductionist rule to causal modeling:

> Rule S.B. If the correlation r_{xz} between x and z is high positive (or negative) and the partial correlation coefficient $r_{xz \cdot y}$ between x and z with y "held constant" is zero, then either (a) y is an intervening variable--the causal effect of x or z (or vice versa) operates through y; or (b) y is a common cause of x and z--the correlation between x and z is spurious. (pp. 70-71).

In other words, that $r_{xz \cdot y} = 0$ is sufficient to infer either $x \rightarrow y \rightarrow z$ or

$x \leftarrow y \leftarrow z$ or $y \swarrow x$, $y \searrow z$.

It is not difficult to show that scientists working on causal models neither used nor endorsed such a rule, for it is invalid, as Sewall Wright[4] showed as early as 1934:

Let $r_{xz} = 0.64$ and $r_{xy} = r_{yz} = 0.8$. Then numerator of $r_{xz \cdot y} \equiv r_{xz} - r_{xy} r_{yz} = 0.64 - (0.8)(0.8) = 0$.

That reduction in general is not an aim of CM can be seen from the care with which methodologists speak of their techniques. Thus, Sewall Wright writes: "It has been emphasized that the method of path

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coefficient is not intended to accomplish the impossible task of deducing causal relations from the values of the correlation coefficients. It is intended to combine the quantitative information given by the correlations with such qualitative information as may be at hand on causal relations to give a quantitative interpretation." (1934, p. 193). In a similar vein, two leading sociologists, P. M. Blau and O. D. Duncan, echo Sewall Wright: "The technique of path analysis is not a method for discovering causal laws but a procedure for giving a quantitative interpretation to the manifestations of a known or assumed causal system." (1967, p. 177).

Contrary to appearance, these quotations are not an appeal to authority. They simply express the nonreductionist character of CM. It is instructive to see why causal methodology cannot be reductionist. The scientists quoted above allude to the fact that one must have an explicit, well defined causal model in the first place so that an appropriate statistical method can be meaningfully applied. Recall the U-assumption that all models share: the disturbance term(s), U, must be uncorrelated with the explanatory variables; otherwise the statistical methods yield biased (misleading) estimates for the model parameters. But U contains all causal factors for the variable to be explained. Therefore, to see if C causes E one must make the causal assumption that all other causes of E that are not explicitly incorporated into the model are uncorrelated with C. This is one of the two reasons why CM cannot (and does not attempt to) reduce causation to statistics.

In a perceptive article, Nancy Cartwright (1979) gives a similar account as to why statistical analyses of causation (such as Patrick Suppes') have failed. She shows that most of the counterexamples against the idea that a cause C should increase (or merely change) the probability of its effect E have the same structure: there is a third factor which both causes E and is correlated with C. In the presence of such a factor, C may fail to increase (or change) the probability of E even if C causes E. This statistical phenomenon is known as Simpson's paradox. The following example by Cartwright (1979, pp. 421-422) illustrates how it functions.

Suppose that smoking causes heart disease ($s \rightarrow h$). We would expect that $P(h/s) > P(h)$. However, if exercising should happen to prevent heart disease more effectively than smoking causes it, and if smoking and exercising are themselves sufficiently correlated, we might actually observe that $P(h/s) < P(h)$. That is, if smokers also tend strongly to be exercisers, the net result may be that they are in fact less likely to have heart disease.

As cases of Simpson's paradox fail to exhibit the expected increase in the probability of the effect, so the misspecification problem in CM leads to biased estimates of the true model parameters. The reasons why CM is non-reductionist and why probabilistic theories of causation fail to reduce causal laws to laws of association are at root identical.

But there is another reason why CM cannot be reductionist. Statistical data underdetermine causal models in the sense that there may be more than one model compatible with a given body of data. Let us take the simplest case possible: a two-variable recursive model.[5]

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Model 1: $z_1 \xrightarrow{p_{21}} z_2 \leftarrow U_2$ $\qquad z_2 = p_{21}z_1 + U_2$

Model 2: $z_2 \xrightarrow{p_{12}} z_1 \leftarrow U_1$ $\qquad z_1 = p_{12}z_2 + U_1$.

Our aim is to see whether we can unequivocally distinguish between these two models solely on the basis of statistical relationships. As before, we assume that z_i represents standardized scores, and that z_1 and z_2 are uncorrelated with U_2 and U_1 respectively, so that the problem of mis-specification does not arise. By the method of expectations,

$$E(z_1z_2) = p_{21}\, E(z_1^2) + E(U_2z_1) \quad \text{for Model 1}$$

$$E(z_1z_2) = p_{12}\, E(z_2^2) + E(U_1z_2) \quad \text{for Model 2}.$$

Recalling that $E(z_iU_j) = 0$ $(i \neq j)$, $E(z_iz_j) = r_{ij}$ $(i \neq j)$ and $E(z_i^2) = 1$ $(i = j)$

since all variables are standardized, we get respectively:

$$r_{12} = p_{21} \text{ and } r_{12} = p_{12}.$$

In other words, the model parameters in both cases are given by the same quantity, namely the correlation coefficient r_{12}. Therefore, we cannot distinguish between them.

It might be argued that the time sequence of events does enable us to choose between the two models. If, for instance, z_1 occurs before z_2, we can eliminate Model 2. But the difficulty is that we do not have this kind of information for many social phenomena. Is it large budget deficits that cause higher inflation rates, or vice versa? Of course, it may be both, which suggests that we should perhaps test one non-recursive model (rather than two separate recursive models) in which both z_1 and z_2 cause each other reciprocally:

$U_1 \searrow z_1 \rightleftarrows z_2 \swarrow U_2$ $\qquad z_2 = p_{21}\, z_1 + U_2$

$\qquad\qquad\qquad\qquad\qquad\quad z_1 = p_{12}\, z_2 + U_1$.

The rationale is that if z_1 causes z_2 and not vice versa, then p_{12} must be zero while p_{21} is not. In the case of z_2 causing z_1, only p_{21} would be zero. If in reality z_1 and z_2 are mutually related as hypothesized, then neither parameter should be zero. In any case, it seems better to test this single model.

Unfortunately, we run into the problem of identification. Since U_1 influences z_2 through z_1, and similarly U_2 affects z_1 through z_2, U_1 will be correlated with z_2 and U_2 with z_1. Hence, we cannot assume that $E(z_iu_i) = 0 (i \neq j)$. This results in having one less equation than needed. The model becomes underdetermined, and neither p_{21} nor p_{12} can be computed.

In general, any model in which all possible causal paths are included will have more parameters than the number of equations from which they

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can be estimated. Consequently, all such models will be unidentifiable, and thus irreducible to statistical relations.

The recognition of the non-reductionist character of CM removes the tension between it and the recent developments in probabilistic theories of causation. As we saw, Cartwright abandons the program of reducing causal laws to laws of association and embraces the less ambitious, but perhaps more meaningful task of specifying the precise nature of the relationship between the two. CM methodology can be seen as a systematic attempt to study the statistical manifestations of underlying causal structures and hence to understand their nature.

4. The Principle of Common Cause

Causal modeling makes extensive use of the common-cause principle, advocated by Reichenbach and Salmon, which states that "when apparent coincidences occur that are too improbable to be attributed to chance, they can be explained by reference to a common causal antecedent." (Salmon 1984, p. 158). Model construction in CM begins with the identification of highly correlated variables and proceeds with postulating a direct and/or indirect causal relationship. If such a causal connection is unlikely, a common cause is hypothesized on the basis of available social theories. The principle of common cause lends CM predictive as well as explanatory power, since a common-cause model implies the vanishing of a partial correlation, which can easily be tested by sample statistical data.

Moreover, CM techniques such as path analysis employ a result known as the decomposition law[6], whose effect is to resolve a statistical association into its causal components, much in the spirit of the common cause principle. Consider the common-cause model of section 2. As we saw, $r_{xy} = p_1 p_2$. This equation (which is also derivable from the decomposition law) can be interpreted as showing how the common cause, z, produces a (spurious) correlation between x and y, because p_1 and p_2 represent the causal impact of z on x and y, respectively. The magnitude of this spurious correlation is equal to the product of p_1 and p_2.

I would like to argue further that a generalized version of the common cause principle also plays a crucial role in labeling a model causal. I claim that the causal character of any specific model is imported via (1) the U-assumption, and (2) the principle of causation according to which the correlation between any two variables in the model is either due to a (direct or indirect) causal connection or is the result of a common cause.

The argument is this. (1) and (2) together imply that the explanatory variables in the model are not causally related to U's; for otherwise U's would be correlated with them, which violates (1). But, by (2), any observed correlation between the explanatory variables and the variable-to-be explained must be produced by the causal relations between them, which are explicitly included in the model. Therefore, the model is a causal one.[7]

Needless to say, the validity of my argument depends upon (1) and (2). As I have discussed earlier, the U-assumption is necessary to avoid misspecification. Here, it ensures that the correlations in the

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model are not produced by factors outside the model. The inclusion of all other causal factors as part of the model provides a closure over the system variables, thereby preventing outside ones from interfering and producing spurious correlations in the model. The difficulty with the U-assumption is that there is no direct way of checking if it is met in a particular model. If we suspect that there are variables in U correlated with any of the explanatory variables, we must explicitly incorporate them into the model.

On the other hand, the status of the principle of causation is debatable. Although the idea behind it (namely, that statistical regularities should be accounted for in terms of causal considerations) is sound, surely there might be "accidental correlations" in the world for which no cause (common or direct) can be invoked.[8] For this reason, it seems to me that we should interpret (2) more as a necessary local condition for the applicability of the model to factual cases than as a global principle of explanation in general. I believe that, because of this local interpretation, the principle of causation escapes much of the criticism launched by van Fraassen (1982) against the principle of common cause. And, regardless of their universality, both principles undoubtedly play an important role for scientific explanations, at least outside quantum domain.

5. Causal Processes

Wesley Salmon is another philosopher who is convinced that causal factors cannot be explicated solely in terms of statistical relations. Accordingly, in the second part of his (1984), he provides a detailed characterization of causal relations in terms of physical processes. Salmon argues that, contrary to what Hume had assumed, causation is a three-term relation: the cause, the effect, and the process that connects them. *It is the processes that are basic to causation, not* events.

The distinctive feature of a physical process is its continuity in space and time (except, perhaps, in quantum theory). Of course, not all continuous processes are causal. While a car moving on a road constitutes a causal process, its shadow on the road is a paradigm case of a non-causal one. Salmon proposes a principle (which was first formulated by Hans Reichenbach) to distinguish between the two. According to the criterion of mark transmission, a causal process can transmit a mark; a non-causal process cannot. To use one of Salmon's examples, a beam of light coming from a light source qualifies as a genuine causal process because if we put a red filter through its path, this mark will be carried by the light beam and a red spot will be observed on the screen. Shadows, by contrast, are not capable of transmitting such marks.

In many cases, understanding or even accurately describing the causal process is as important as identifying the cause. I fire a cannon, the ball follows a parabolic path and hits the ground, opening up a big hole. For a physicist, the interesting problem here is not the identification of the cause (firing), but is to give a satisfactory description of the cannon ball's trajectory.

I believe that the idea of causal process provides a suitable framework both for causal modeling and for models of causal explanation--a

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framework which has been anticipated and articulated by the founders of CM, Sewall Wright and Herbert Simon.

Simon, a well known econometrician who was one of the first to apply CM to social sciences, argues that the idea of a mechanism is the basis of all causal explanations. He writes that "... in theories in the social and behavioral sciences, we frequently employ the postulate, 'if no communication, then no influence'." (1979, p. 73). A behavior is explained by identifying the stimuli that affected it as well as by showing how the stimuli were communicated. Our suspicion of ESP, remarks Simon, rests completely upon the "absence of ... mechanism or of specifications of what the character of such a mechanism might be." (p. 70).

One might think that Simon uses the word 'mechanism' metaphorically, not literally. However, he makes quite clear that a mechanism is a real entity: "In many, but not all, cases a mechanism means something which experience tells us is capable of producing the observed effect. If the mechanism is not itself visible, then there must be some detectable circumstances that tell us it is present." (p. 70). It is these mechanisms that causal models aim to capture: "scientific inquiry is concerned not only with discovering quantitative relations between variables, but also with interpreting these relations in terms of the underlying causal mechanisms that produced them." (p. 79).

The resemblance between H. Simon's and W. Salmon's (recent) views on causality is obvious. Both rely upon an intuitive notion of causality, based upon (continuous) processes, as independent and fundamental to any potential scientific explanation.

There is even a more striking similarity between Salmon's and Sewall Wright's thinking on causality. Wright's views are so novel for his time and so well articulated that it is regrettable they went unnoticed by philosophers for more than fifty years:

> It is assumed that any event always traces back continuously in time and space through successions of previous events and that, statistically, variations in events of a given sort may be traced in principle to variations in previous ones of specified sort, with varying degrees of relative importance, however difficult it may be in practice to disentangle such unidirectional sequences from the effects of common factors or of rapid reciprocal interaction. (1934, pp. 15-16)

By "traces back" Wright implied that the directionality of time was fundamental to his notion of causality. He was prepared to defend this idea with remarkable awareness of (then) recent developments in physics.

> Under the theory of relativity ... the objective world is to be thought of as a complex network of point events. Although two such events sufficiently remote from each other in space, relative to their separation in time, may have their order of succession reversed in the systems of two different observers, order in time is invariant along any strand of this network involving continuity of physical

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> action ... . Such successions of events as involved in the movement of a shadow over a surface may indeed be reversed by change of viewpoint ... but the continuity of physical action here is not along the path of the shadow ... . In principle the distinction is clear enough. Experimental intervention is possible only in the true lines of causation. (1934, p. 176).

This should sound quite familiar to those acquainted with Reichenbach's (1956) arguments, adopted by Salmon as well, on the directionality of time, the relation between time and causality, and the distinction between causal processes and pseudoprocesses by means of the mark criterion.

Wright, a population geneticist, Simon, an econometrician, and Salmon, a philosopher, have independently showed how we can make sense of causation if we turn to processes and mechanisms rather than events. That the same notion can be meaningfully appropriated for physical, biological, and social phenomena as well as in philosophy suggests that we may have finally hit upon the correct view of causation.

The similarities between causal modeling and recent developments in probabilistic causation and statistical explanation extend well beyond the technical details, to cover the fundamental intuitions and concerns behind them. We have at our disposal a powerful scientific theory and the possibility of a genuine unification between a scientific discipline and philosophy--a unification from which both sides can benefit, true to the spirit of philosophy of science.

Notes

[1] A typical assumption (which facilitates the use of statistical techniques such as regression) about these variables is that they are continuous, i.e., measured over an interval scale. We shall further assume that they are standardized, that is, measured in standard deviations from their respective means.

[2] In this paper, we will bypass the issues of model construction (the choice of relevant explanatory variables for the explanandum variable and their interrelationships) and estimation. The interested reader can refer to Blalock (1969), Duncan (1975), Hanushek and Jackson (1977).

[3] See Irzik and Meyer (forthcoming), where several superiorities of CM are also pointed out.

[4] Wright is a population geneticist, the first to develop the theory of CM and successfully apply it to a number of biological cases.

[5] It is possible to show that if reduction is not possible here, it cannot succeed for higher order systems either.

[6] See Wright (1934); Asher (1976).

[7] We should note that this argument does not tell us anything about the direction of causality, but simply reveals and ensures the existence of

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some causal connections within the model.

[8]See van Fraassen (1982) for an example from quantum mechanics.

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