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Structural equation models (SEMs) have dominated causal analysis in the social and behavioral sciences since the 1960s. Currently, many SEM practitioners are having difficulty articulating the causal content of SEM and are seeking foundational answers. Recent developments in the areas of graphical models and the logic of causality show potential for alleviating such difficulties and, thus, revitalizing structural equations as the primary language of causal modeling. This article summarizes several of these developments, including the prediction of vanishing partial correlations, model testing, model equivalence, parametric and nonparametric identifiability, control of confounding, and covariate selection. These developments clarify the causal and statistical components of SEMs and the role of SEM in the empirical sciences.

Graphs, Causality, and Structural Equation Models

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1. INTRODUCTION

1.1. CAUSALITY IN SEARCH OF A LANGUAGE

The word *cause* is not in the vocabulary of standard probability theory. It is an embarrassing yet inescapable fact that probability theory, the official mathematical language of many empirical sciences, does not permit us to express sentences such as "Mud does not

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cause rain"; all we can say is that the two events are mutually correlated, or dependent—meaning that if we find one, we can expect to encounter the other. Scientists seeking causal explanations for complex phenomena or rationales for policy decisions must therefore supplement the language of probability with a vocabulary for causality, one in which the symbolic representation for the causal relationship "Mud does not cause rain" is distinct from the symbolic representation for "Mud is independent of rain." Oddly, such distinctions have not yet been incorporated into standard scientific analysis.[1]

Two languages for causality have been proposed: path analysis or structural equation models (SEMs) (Wright 1921; Haavelmo 1943) and Neyman-Rubin's potential response model (Neyman 1923; Rubin 1974). The former has been adopted by economists and social scientists (Goldberger 1972; Duncan 1975), whereas a small group of statisticians champion the latter (Rubin 1974; Robins 1986; Holland 1988). These two languages are mathematically equivalent,[2] yet neither has become standard in causal modeling—the structural equation framework because it has been greatly misused and inadequately formalized (Freedman 1987), and the potential response framework because it has been only partially formalized and, more significant, because it rests on an esoteric and seemingly metaphysical vocabulary of counterfactual variables that bears no apparent relation to ordinary understanding of cause-effect processes.

Currently, potential response models are understood by few and used by even fewer, whereas SEMs are used by many but their causal interpretation is generally questioned or avoided. The main purpose of this article is to formulate the causal interpretation and outline the proper use of SEMs to reinstate confidence in SEM as the primary formal language for causal analysis in the social and behavioral sciences. But first, a brief analysis of the current crisis in SEM research in light of its historical development.

1.2. CAUSALITY AND STRUCTURAL MODELS

SEM was developed by geneticists (Wright 1921) and economists (Haavelmo 1943; Koopmans 1953; Koopmans, Rubin, and Leipnik 1950) so that qualitative cause-effect information could be combined with statistical data to provide quantitative assessment of cause-effect

relationships among variables of interest. Thus, to the often-asked question, "Under what conditions can we give causal interpretation to structural coefficients?" Wright and Haavelmo would have answered, "Always!" According to the founding fathers of SEM, the conditions that make the equation $y = \beta x + \varepsilon$ structural are precisely those that make the causal connection between X and Y have no other value but β, and nothing about the statistical relationship between x and ε can ever change this interpretation of β. Amazingly, this basic understanding of SEM has all but disappeared from the literature, leaving modern econometricians and social scientists in a quandary over β.

Most SEM researchers today are of the opinion that extra ingredients are necessary for structural equations to qualify as carriers of causal claims. Among social scientists, James, Mulaik, and Brett (1982:45), for example, state that a condition called self-containment is necessary for consecrating the equation $y = \beta x + \varepsilon$ with causal meaning, where self-containment stands for $\text{cov}(x, \varepsilon) = 0$. According to James, Mulaik, and Brett, whenever self-containment does not hold, "Neither the equation nor the functional relation represents a causal relation." Bollen (1989:44) reiterates the necessity of self-containment (under the rubric *isolation* or *pseudoisolation*) contrary to the understanding that structural equations attain their causal interpretation prior to, and independent of, any statistical relationships among their constituents. Since the early 1980s, it has become exceedingly rare to find an open endorsement of the original SEM logic, namely, that ε is defined in terms of β, not the other way around, and that the orthogonality condition $\text{cov}(x, \varepsilon) = 0$ is neither necessary nor sufficient for the causal interpretation of β (see Section 4.1). In fact, this condition is not necessary even for the identification of β, once β is interpreted (see the identification of α in Figures 8 and 10).

Econometricians have just as much difficulty with the causal reading of structural parameters. Leamer (1985:258) observes, "It is my surprising conclusion that economists know very well what they mean when they use the words 'exogenous,' 'structural,' and 'causal,' yet no textbook author has written adequate definitions." There has been little change since Leamer made these observations. Hendry (1995:62), for instance, amplifies the necessity of the orthogonality condition, and states: "The status of β may be unclear until the

conditions needed to estimate the postulated model are specified. For example, in the model:

$$y_t = z_t\beta + u_t \text{ where } u_t \sim \text{IN}[0, \sigma_u^2],$$

until the relationship between z_t and u_t is specified the meaning of β is uncertain since $E[z_t, u_t]$ could be either zero or non-zero on the information provided." LeRoy (1995:211) goes even further: "It is a commonplace of elementary instruction in economics that endogenous variables are not generally causally ordered, implying that the question 'What is the effect of y_1 on y_2' where y_1 and y_2 are endogenous variables is generally meaningless." According to LeRoy, causal relationships cannot be attributed to any variable whose causes have separate influence on the effect variable, a position that denies causal reading to most of the structural parameters that economists and social scientists labor to estimate.

Cartwright (1995:49), a renowned philosopher of science, addresses these difficulties by initiating a renewed attack on the tormenting question, "*Why* can we assume that we can read off causes, including causal order, from the parameters in equations whose exogenous variables are uncorrelated?" Cartwright, like SEM's founders, recognizes that causes cannot be derived from statistical or functional relationships alone and that causal assumptions are prerequisite for validating any causal conclusion. Unlike Wright and Haavelmo, however, she launches an all-out search for the assumptions that would endow the parameter β in the regression equation $y = \beta x + \varepsilon$ with a legitimate causal meaning and endeavors to prove that the assumptions she proposes are indeed sufficient. What is revealing in Cartwright's analysis is that she does not consider the answer Haavelmo would have provided, namely, that the assumptions needed for drawing causal conclusions from parameters are already encoded in the *syntax* of the equations and can be read off the associated graph as easily as a shopping list;[3] they need not be searched for elsewhere nor do they require new proofs of sufficiency. Haavelmo's answer applies to models of any size and shape, including models with correlated exogenous variables.

These examples partake of an alarming tendency among economists and social scientists to view a structural equation as an algebraic

object that carries functional and statistical assumptions but is void of causal content. This statement from one leading social scientist is typical: "It would be very healthy if more researchers abandoned thinking of and using terms such as cause and effect" (Muthen 1987:180). Perhaps the boldest expression of this tendency was recently voiced by Holland (1995:54): "I am speaking, of course, about the equation: $\{y = a + bx + \varepsilon\}$. What does it mean? The only meaning I have ever determined for such an equation is that it is a shorthand way of describing the conditional distribution of $\{y\}$ given $\{x\}$."[4]

The founders of SEM had an entirely different conception of structures and models. Wright (1923:240) declared that "prior knowledge of the causal relations is assumed as prerequisite" in the theory of path coefficients, and Haavelmo (1943) explicitly interpreted each structural equation as a statement about a hypothetical controlled experiment. Likewise, Marschak (1950) and Koopmans (1953) stated that the purpose of postulating a structure behind the probability distribution is to cope with the hypothetical changes that can be brought about by policy. One wonders, therefore, what has happened to SEM over the past 50 years, and why the basic (and still valid) teachings of Wright, Haavelmo, Marschak, and Koopmans have been forgotten.

Some economists attribute the decline in the understanding of structural equations to Lucas's (1976) critique, according to which economic agents anticipating policy interventions would tend to act contrary to the SEM's predictions, which often ignore such anticipations. However, since Lucas's critique merely shifts the model's invariants and the burden of structural modeling from the behavioral level to a deeper level, involving agents' motivations and expectations, it does not exonerate economists from defining and representing the causal content of structural equations at some level of discourse.

I believe that the causal content of SEM has gradually escaped the consciousness of SEM practitioners mainly for the following reasons:

1. SEM practitioners have sought to gain respectability for SEM by keeping causal assumptions implicit, since statisticians, the arbiters of respectability, abhor assumptions that are not directly testable.
2. The algebraic language that has dominated SEM lacks the notational facility needed to make causal assumptions, as distinct from statistical

> assumptions, explicit. By failing to equip causal relations with precise mathematical notation, the founding fathers in fact committed the causal foundations of SEM to oblivion. Their disciples today are seeking foundational answers elsewhere.

Let me elaborate on the latter point. The founders of SEM understood quite well that in structural models, the equality sign conveys the asymmetrical relation "is determined by" and, hence, behaves more like an assignment symbol (:=) in programming languages than like an algebraic equality. However, perhaps for reasons of mathematical purity (i.e., to avoid the appearance of syntax sensitivity), they refrained from introducing a symbol to represent the asymmetry. According to Epstein (1987), in the 1940s, Wright gave a seminar on path coefficients to the Cowles Commission (the breeding ground for SEM), but neither side saw particular merit in the other's methods. Why? After all, a diagram is nothing but a set of nonparametric structural equations in which, to avoid confusion, the equality signs are replaced with arrows.

My explanation is that the early econometricians were extremely careful mathematicians who thought they could keep the mathematics in purely equational-statistical form and just reason about structure in their heads. Indeed, they managed to do so surprisingly well, because they were truly remarkable individuals who *could* do it in their heads. The consequences surfaced in the early 1980s, when their disciples began to mistake the equality sign for an algebraic equality. The upshot was that suddenly the "so-called disturbance terms" did not make any sense at all (Richard 1980:3). We are living with the sad end to this tale. By failing to express their insights in mathematical notation, the founders of SEM brought about the current difficulties surrounding the interpretation of structural equations, as summarized by Holland's "What does it mean?"

1.3. GRAPHS AS A MATHEMATICAL LANGUAGE: AN EXAMPLE

Certain recent developments in graphical methods promise to bring causality back into the mainstream of scientific modeling and analysis. These developments involve an improved understanding of the relationships between graphs and probabilities on one hand and graphs

and causality on the other. But the crucial change has been the emergence of graphs as a mathematical language. This mathematical language is not simply a heuristic mnemonic device for displaying algebraic relationships, as in the writings of Blalock (1962) and Duncan (1975). Rather, graphs provide a fundamental notational system for concepts and relationships that are not easily expressed in the standard mathematical languages of algebraic equations and probability calculus. Moreover, graphical methods now provide a powerful symbolic machinery for deriving the consequences of causal assumptions when such assumptions are combined with statistical data.

A concrete example that illustrates the power of the graphical language will set the stage for the discussions in Sections 2 and 3. One of the most frustrating problems in causal analysis has been covariate selection—for instance, determining whether a variate Z can be added to a regression equation without biasing the result. More generally, whenever we try to evaluate the effect of one factor (X) on another (Y), we wonder whether we should adjust for possible variations in some other variable, Z, sometimes called a covariate, concomitant, or confounder. Adjustment amounts to partitioning the population into groups that are homogeneous relative to Z, assessing the effect of X on Y in each homogeneous group, and, finally, averaging the results.

The elusive nature of such an adjustment was recognized as early as 1899, when Pearson and Yule discovered what is now called Simpson's paradox, namely, that any statistical relationship between two variables may be reversed or negated by including additional factors in the analysis. For example, we may find that students who smoke obtain higher grades than those who do not smoke; but after we adjust for age, smokers obtain lower grades than nonsmokers in every age group; but after we further adjust for family income, smokers obtain higher grades than nonsmokers in every income-age group; and so on.[5]

Despite a century of analysis, Simpson's reversal phenomenon continues to "trap the unwary" (Dawid 1979:5), and the main question—whether an adjustment for a given covariate Z is appropriate in any given study—continues to be decided informally, case by case, with the decision resting on folklore and intuition rather than on hard mathematics. The standard statistical literature is remarkably silent on this issue. Aside from noting that one should not adjust for a covariate

that is affected by the putative cause (X),[6] it provides no guidelines as to what covariates might be admissible for adjustment and what assumptions would be needed for making such a determination formally. The reason for this silence is clear: The solution to the covariate selection problem rests on causal assumptions, as we shall see in Section 3, and such assumptions cannot be expressed formally in the standard language of statistics.

In the potential response framework, a criterion called ignorability has been advanced to address the covariate selection problem (Rosenbaum and Rubin 1983). It states that Z is an admissible covariate relative to the effect of X on Y if, for every x, the value that Y would obtain had X been x is conditionally independent of X given Z. This criterion paraphrases the problem in the language of counterfactuals without providing a working test for covariate selection. Because counterfactuals are not observable, and judgments about the conditional independence of counterfactuals are not readily made using our ordinary understanding of causal processes, ignorability has remained a theoretical construct with only a minor impact on practice. Epidemiologists, for example, well apprised of ignorability analysis via the admirable work of Robins (1986) and Greenland and Robins (1986), are still debating the meaning of "confounding" (Grayson 1987) and often adjust for the wrong sets of covariates (Weinberg 1993). Social scientists, likewise, despite the potential response analyses of Holland and Rubin (1983) and Sobel (1995), are still struggling with various manifestations of the Lord paradox (a version of Simpson's paradox) in psychometric research (Wainer 1991) and are still not distinguishing collapsibility from nonconfounding (Steyer, Gabler, and Rucai 1996).

In contrast, formulating the covariate selection problem in the language of graphs immediately yields a general solution that is both natural and formal. The investigator expresses causal knowledge (i.e., assumptions) in the familiar qualitative terminology of path diagrams, and, once the diagram is complete, a simple procedure decides whether a proposed adjustment (or regression) is appropriate relative to the quantity under evaluation. This procedure, called the backdoor criterion in Section 3 (Theorems 6 and 7), proceeds roughly as follows: To determine whether a set of variables Z should be adjusted for when we wish to evaluate the total effect of X on Y, we delete all

arrows emanating from node X and then test whether, in the resulting graph, all paths between X and Y are blocked by nodes corresponding to Z. If the direct effect is to be evaluated, then only the arrow from X to Y should be deleted before applying the test (Theorem 6). The notion blocked is defined formally in Section 2.1.2 (Definition 1).

This example is not an isolated instance of graphical methods affording clarity and understanding. In fact, the conceptual basis for SEM achieves a new level of precision through graphs. What makes a set of equations structural, what assumptions the authors of such equations should examine, what the testable implications of those assumptions are, and what policy claims a given set of structural equations advertise are some of the questions that receive simple and mathematically precise answers via graphical methods. These and related issues in SEM will be discussed in the following sections.

1.4. OUTLINE

The testable implications of structural models are explicated in Section 2. For recursive models (herein termed Markovian), we find that the statistical content of a structural model can be fully characterized by a set of vanishing partial correlations that are entailed by the model. These vanishing partial correlations can be read off the graph using a simple criterion, called d-separation, that applies to both linear and nonlinear models (Section 2.1). The application of this criterion to model testing is discussed in Section 2.2. The d-separation criterion leads to graphical tests of model equivalence that, again, apply to both linear and nonlinear models (Section 2.3).

Section 3 deals with the issue of determining the identifiability of structural parameters prior to gathering any data. In Section 3.1, simple graphical tests of identifiability are developed for linear Markovian and semi-Markovian models (i.e., acyclic diagrams with correlated errors). Extensions to nonparametric models are developed in Sections 3.2 and 3.3, and their ramifications for practical problems of covariate selection are clarified in Section 3.4.

Section 4 discusses the logical foundations of SEM and resolves a number of difficulties that were kept dormant in the past. These include operational definitions for structural equations, structural parameters, error terms, and total and direct effects.

2. GRAPHS AND MODEL TESTING

In 1919, Wright developed his method of path coefficients, which allows researchers to compute the magnitudes of cause-effect relationships from correlation measurements, as long as the path diagram represents correctly the causal processes underlying the data. Wright's method consists of writing a set of equations, one for each pair of variables (X_i, X_j), and equating the (standardized) correlation coefficient ρ_{ij} with a sum of products of path coefficients and residual correlations along the various paths connecting X_i and X_j. Whenever the resulting equations give a unique solution to some path coefficient p_{mn} that is independent of the (unobserved) residual correlations, that coefficient is said to be identifiable. If every set of correlation coefficients ρ_{ij} is compatible with some choice of path coefficients, the model is said to be untestable or unfalsifiable (also called saturated, just identified, etc.), because it is capable of perfectly fitting any data whatsoever.

Whereas Wright's method is partly graphical and partly algebraic, the theory of directed graphs permits us to analyze questions of testability and identifiability in purely graphical terms, prior to data collection, and enables us to extend these analyses from linear to nonlinear or nonparametric models. This section deals with issues of testability in linear and nonparametric models.

2.1. THE TESTABLE IMPLICATIONS OF STRUCTURAL MODELS

When we hypothesize a model of the data-generating process, that model often imposes restrictions on the statistics of the data collected. In observational studies, these restrictions provide the only view under which the hypothesized model can be tested or falsified. In many cases, such restrictions can be expressed in the form of vanishing partial correlations and, more significant, are implied by the structure of the path diagram alone, independent of the numerical values of the parameters. Blalock (1962), having recognized the importance of vanishing partial correlations that are implied by path diagrams, worked out an exhaustive list of those correlations in all path diagrams involving four variables. He also expressed doubt that the list would ever be extended to path diagrams with five (or more) variables.

Nonetheless, a method is now available for identifying vanishing partial correlations in path diagrams of any size or form. The method is based on a test called d-separation (Pearl 1986, 1988), to be discussed next.

2.1.1. Preliminary Notation

The graphs discussed in this article represent sets of structural equations of the form

$$x_i = f_i(pa_i, \varepsilon_i) \qquad i = 1, \ldots, n, \tag{1}$$

where pa_i (connoting parents) stand for the set of variables judged to be immediate causes of X_i, and ε_i represent errors due to omitted factors. Equation (1) is a nonlinear, nonparametric generalization of the standard linear equations

$$x_i = \sum_{k \neq i} \alpha_{ik} x_k + \varepsilon_i \qquad i = 1, \ldots, n, \tag{2}$$

in which pa_i correspond to those variables on the right-hand side of equation (2) that have nonzero coefficients. A set of equations in the form of equation (1) used to represent the data-generating process will be called a *causal model*.[7] The graph G obtained by drawing an arrow from every member of pa_i to X_i will be called a *causal diagram*. In addition to full arrows, a causal diagram should contain a bidirected (i.e., double-arrowed) arc between any pair of variables whose corresponding errors are dependent (as in Figure 3). A diagram may include directed cycles (e.g., $X \rightarrow Y$, $Y \rightarrow X$) representing mutual causation or feedback processes, but not self loops (e.g., $X \rightarrow X$). An edge is either an arrow or a bidirected arc, and two variables connected by an edge are called adjacent.

We make free use of the terminology of kinship (e.g., parents, children, descendants, and ancestors) to denote the relationships in a graph. These kinship relations are defined along the full arrows in the diagram, including arrows that form directed cycles but ignoring bidirected arcs. In Figure 5c, for example, Y has two parents (X and Z), three ancestors (X, Z, and W), and no children, whereas X has no parents (hence, no ancestors) and one child (Y).

Causal diagrams play the same role in nonlinear SEMs as path diagrams play in linear SEMs. Causal diagrams differ from path diagrams in that their pa_i are defined as nontrivial arguments of the function f_i rather than variables obtaining nonzero coefficients, and their bidirected arcs reflect dependency rather than correlation. It is important to emphasize that causal diagrams (as well as traditional path diagrams) should be distinguished from the wide variety of graphical models in the statistical literature whose construction rests solely on properties of the joint distribution (Cox and Wermuth 1996; Andersson et al. 1997; Lauritzen 1996). The missing links in those statistical models represent conditional independencies, whereas the missing links in causal diagrams represent absence of causal connections (see note 3 and Section 4) that may or may not imply conditional independencies in the distribution.

A causal model will be called Markovian if its graph contains no directed cycles and if its ε_is are mutually independent (i.e., no bidirected arcs). A model is semi-Markovian if its graph is acyclic and if it contains dependent errors.

Markovian models (the parallel term in the SEM literature is recursive models;[8] Bollen 1989) possess useful features, shared by both linear and nonlinear systems, that make their statistical implications transparent. One fundamental property of Markovian models is parent screening: given the state of its parents pa_i, each variable X_i is conditionally independent of all its nondescendants in the graph. This follows immediately from the independence of the errors ε_i and supports the intuition that once the direct causes of X_i are known, the probability of X_i is completely determined; no other event preceding X_i could modify this probability. As a result, the statistical parameters of Markovian models can be estimated by ordinary regression analysis.

An immediate consequence of this Markovian property is that the joint distribution of variables generated by equation (1) can be decomposed (using the chain rule of probability calculus) into the product

$$P(x_i,\ldots,x_n) = \prod_i P(x_i \mid pa_i), \tag{3}$$

where pa_i are the values of the parents of X_i in the causal graph G. For example, the model illustrated in Figure 1 induces the decomposition

$$P(x_1, x_2, x_3, x_4, x_5) = P(x_1)P(x_2 \mid x_1)P(x_3 \mid x_1)P(x_4 \mid x_2, x_3)P(x_5 \mid x_4). \quad (4)$$

This decomposition holds for any distribution of the error terms, regardless of the functional form of f_i; it depends only on the structural features of the generating model in equation (1) as captured by the graph G. The product decomposition, in turn, entails certain conditional independence relationships that hold regardless of the functional form of f_i and regardless of the error distribution. Such independencies are said to be entailed by the graph and can be read from the graph using a criterion called d-separation (the d denotes directional).

2.1.2. The d-Separation Criterion

Consider three disjoint sets of variables, X, Y, and Z, that are represented as nodes in a directed acyclic graph (DAG) G. To test whether X is independent of Y given Z in any Markovian model represented by G, we need to test whether the nodes corresponding to variables Z block all paths from nodes in X to nodes in Y. By path, we mean a sequence of consecutive edges (of any directionality) in the graph, and blocking is to be interpreted as stopping the flow of information (or the correlation) between the variables that are connected by such paths.

Definition 1: (d-separation) A path p is said to be d-separated (or blocked) by a set of nodes Z if and only if

1. p contains a chain $i \rightarrow m \rightarrow j$ or a fork $i \leftarrow m \rightarrow j$ such that the middle node m is in Z
2. p contains an inverted fork (or collider) $i \rightarrow m \leftarrow j$ such that the middle node m is not in Z and such that no descendant of m is in Z.

A set Z is said to d-separate X from Y if and only if Z blocks every path from a node in X to a node in Y.

The intuition behind d-separation is simple. In causal chains $i \rightarrow m \rightarrow j$ and causal forks $i \leftarrow m \rightarrow j$, the two extreme variables are marginally dependent but become independent of each other (i.e.,

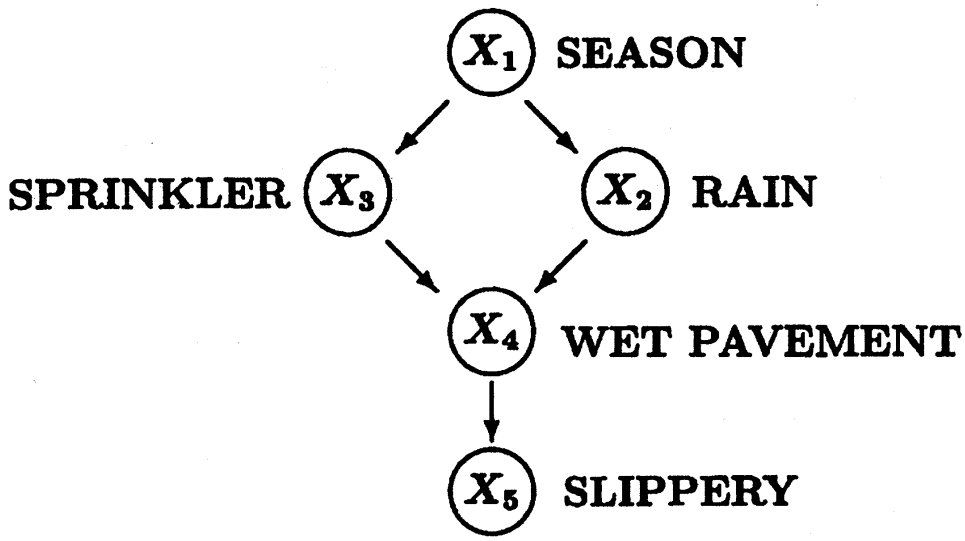

Figure 1: Graph Illustrating Causal Relationships Among Five Variables

blocked) once we condition on the middle variable. Figuratively, conditioning on m appears to block the flow of information along the path, since learning about i has no effect on the probability of j given m. Inverted forks $i \rightarrow m \leftarrow j$, representing two causes having a common effect, act the opposite way; if the two extreme variables are (marginally) independent, they will become dependent (i.e., connected through unblocked path) once we condition on the middle variable (i.e., the common effect) or any of its descendants. This can be confirmed in the context of Figure 1. Once we know the season, X_3 and X_2 are independent (assuming that sprinklers are set in advance, according to the season), but finding that the pavement is wet or slippery renders X_2 and X_3 dependent, since refuting one of these explanations increases the probability of the other.

In Figure 1, $X = \{X_2\}$ and $Y = \{X_3\}$ are d-separated by $Z = \{X_1\}$ because both paths connecting X_2 and X_3 are blocked by Z. The path $X_2 \leftarrow X_1 \rightarrow X_3$ is blocked because it is a fork in which the middle node, X_1, is in Z, whereas the path $X_2 \rightarrow X_4 \leftarrow X_3$ is blocked because it is an inverted fork in which the middle node, X_4, and all its descendants are outside Z. However, X and Y are not d-separated by the set $Z' = \{X_1, X_5\}$: the path $X_2 \rightarrow X_4 \leftarrow X_3$ (an inverted fork) is not blocked by Z', since X_5, a descendant of the middle node X_4, is in Z'. Metaphorically, learning the value of the consequence X_5 renders its causes X_2 and X_3 dependent, as if a pathway were opened along the arrows converging at X_4.

Readers might find it a bit odd that conditioning on a node not lying on a blocked path may unblock the path. However, this corresponds to a general rule about causal relationships: Observations on a common consequence of uncorrelated causes tend to render those causes correlated. This rule is known as Berkson's paradox in the statistical literature (Berkson 1946) and as the explaining away effect in artificial intelligence (Kim and Pearl 1983). For example, if the admission criteria to a certain graduate school call for either high grades as an undergraduate or special musical talents, then these two attributes will be found to be negatively correlated in the student population of that school, even if these attributes are uncorrelated in the population at large. Indeed, students with low grades are likely to be exceptionally gifted in music, which explains their admission to graduate school.

Algebraically, the partial correlations associated with $i \rightarrow m \leftarrow j$ are governed by the equation $\rho_{ij \cdot m} = (\rho_{ij} - \rho_{im}\rho_{jm})/(1-p_{im}^2)^{\frac{1}{2}}(1-\rho_{jm}^2)^{\frac{1}{2}}$, which renders $\rho_{ij \cdot m} \neq 0$ when $\rho_{ij} = 0$. The same applies to the partial correlation $\rho_{ij \cdot m'}$, where m' is any descendant of m.

Theorem 1: If sets X and Y are d-separated by Z in a DAG G, then X is independent of Y conditional on Z in every Markovian model structured according to G. Conversely, if X and Y are not d-separated by Z in a DAG G, then X and Y are dependent conditional on Z in almost all Markovian models structured according to G (Verma and Pearl 1990a; Geiger, Verma, and Pearl 1990).

Because conditional independence implies zero partial correlation, Theorem 1 translates into a graphical test for identifying those partial correlations that must vanish in the model.

Corollary 1: In any Markovian model structured according to a DAG G, the partial correlation $\rho_{XY \cdot Z}$ vanishes whenever the nodes corresponding to the variables in Z d-separate node X from node Y in G, regardless of the model's parameters. Moreover, no other partial correlation would vanish for all the model's parameters.

Unrestricted semi-Markovian models can always be emulated by Markovian models that include latent variables, with the latter accounting for all dependencies among error terms. Consequently, the

d-separation criterion remains valid in such models if we interpret bidirected arcs as emanating from latent common parents. This is not always possible in linear semi-Markovian models if each latent variable is restricted to influence at most two observed variables (Spirtes et al. 1996). However, it has been shown that the d-separation criterion remains valid in such restricted systems (Spirtes et al. 1996) and, moreover, that the validity is preserved when the network contains cycles (Koster 1998; Spirtes et al. 1998 [this issue]). These results are summarized in the next theorem.

Theorem 2: For any linear model structured according to diagram D, which may include cycles and bidirected arcs, the partial correlation $\rho_{XY \cdot Z}$ vanishes if the nodes corresponding to the set of variables Z d-separate node X from node Y in D, where each bidirected arc $i \leftarrow - - \rightarrow j$ is interpreted as a latent common parent $i \leftarrow L \rightarrow j$.

For linear SEMs (see equation (2)), Theorem 2 implies that those (and only those) partial correlations identified by the d-separation test are guaranteed to vanish independent of the model parameters α_{ik} and independent of the error variances. This suggests a simple and direct method for testing models: Rather than going through the standard exercise of finding a maximum likelihood estimate for the model's parameters and scoring those estimates for fit to the data, we can directly test for each zero partial correlation implied by the free model. The advantages of using such tests were noted by Shipley (1997), who also devised implementations of these tests.

However, the question arises whether it is feasible to test for the vast number of vanishing partial correlations entailed by a given model. Fortunately, these partial correlations are not independent of each other but can be derived from a relatively small number of partial correlations that constitutes a basis for the entire set (Pearl and Verma 1987).

Definition 2: (basis) Let S be a set of partial correlations. A basis B for S is a set of zero partial correlations that implies (using the laws of probability) the vanishing of every element of S, and no proper subset of B sustains such implication.

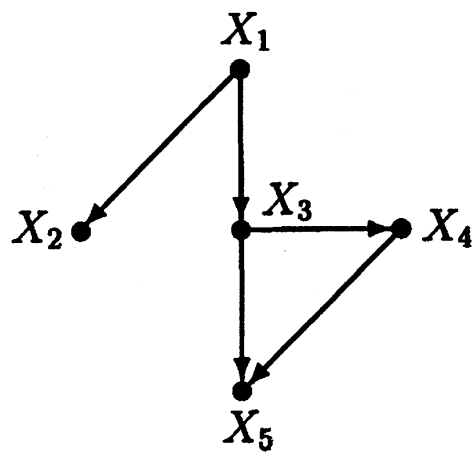

Figure 2: Model Testable With Two Regressors for Each Missing Link (Equation 5)

An obvious choice of a basis for the zero partial correlations entailed by a DAG D is the set of equalities $B = \{\rho_{ij \cdot pa_i} = 0 \mid i > j\}$, where i ranges over all nodes in D and j ranges over all predecessors of i in any order that agrees with the arrows of D. This set of equalities reflects in fact the "parent screening" property of Markovian models, which is the source of all the probabilistic information encoded in a DAG. Testing for these equalities is sufficient, therefore, for testing all the statistical claims of a linear Markovian model. Moreover, when the parent sets pa_i are large, it may be possible to select a more economical basis, as shown in the next theorem (Pearl and Meshkat 1998).[9]

Theorem 3: (graphical basis) Let (i,j) be a pair of nonadjacent nodes in a DAG D, and Z_{ij} any set of nodes that are closer to i than j is to i, and such that Z_{ij} d-separates i from j. The set of zero partial correlations $B = \{\rho_{ij \cdot Z_{ij}} = 0 \mid i > j\}$ consisting of one element per nonadjacent pair constitutes a basis for the set of all vanishing partial correlations entailed by D.

Theorem 3 states that each of several sets of zero partial correlations can encapsulate the entire statistical information conveyed by a linear Markovian model. Examining Figure 2, we see that each of following two sets forms a basis for the model in the figure:

$$B_1 = \{\rho_{32 \cdot 1} = 0, \rho_{41 \cdot 3} = 0, \rho_{42 \cdot 3} = 0, \rho_{51 \cdot 43} = 0, \rho_{52 \cdot 43} = 0\} \tag{5}$$
$$B_2 = \{\rho_{32 \cdot 1} = 0, \rho_{41 \cdot 3} = 0, \rho_{42 \cdot 1} = 0, \rho_{51 \cdot 3} = 0, \rho_{52 \cdot 1} = 0\}.$$

The basis B_1 employs the parent set pa_i for separating i from j, $i > j$. Basis B_2, on the other hand, employs smaller separating sets, thus leading to tests involving fewer regressors. Note that each member of a basis corresponds to a missing arrow in the DAG; therefore, the number of tests required to validate a DAG is equal to the number of missing arrows it contains. The sparser the graph, the more it constrains the covariance matrix, and more tests are required to verify those constraints.

2.2. *TESTING THE TESTABLE*

In linear SEMs, the hypothesized causal relationships between variables can be expressed in the form of a directed graph annotated with coefficients, some fixed a priori (usually to zero) and some free to vary. The conventional method for testing such a model against the data involves two stages. First, the free parameters are estimated by iteratively maximizing a fitness measure such as the maximum likelihood function. Second, the covariance matrix implied by the estimated parameters is compared to the sample covariances and a statistical test is applied to decide whether the latter could originate from the former (Bollen 1989; Chou and Bentler 1995).

There are two major weaknesses to this approach:

1. If some parameters are not identifiable, the first phase may fail to reach stable estimates for the parameters, and the investigator must simply abandon the test.
2. If the model fails to pass the data fitness test, the investigator receives very little guidance about which modeling assumptions are wrong.

For example, Figure 3 shows a path model in which the parameter α is not identifiable if $\text{cov}(\varepsilon_1, \varepsilon_2)$ is assumed unknown, which means that the maximum likelihood method may fail to find a suitable estimate for α, thus precluding the second phase of the test. Still, this model is no less testable than the one in which $\text{cov}(\varepsilon_1, \varepsilon_2) = 0$, α is identifiable, and the test can proceed. These models impose the same restrictions on the covariance matrix, namely, that the partial correlation $\rho_{XZ \cdot Y}$ should vanish (i.e., $\rho_{XZ} = \rho_{XY}\, \rho_{YZ}$), yet the model with

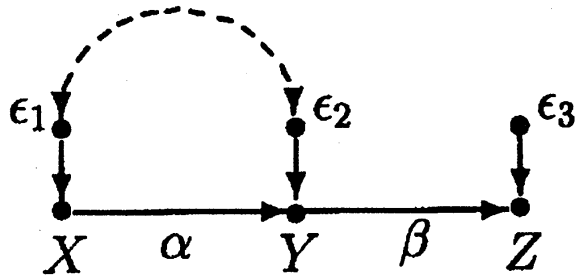

Figure 3: A Testable Model Containing Unidentified Parameter (α)

free cov(ε_1, ε_2), by virtue of α being nonidentifiable, cannot be tested for this restriction.

Figure 4 illustrates the weakness associated with model diagnosis. Suppose the true data-generating model has a direct causal connection between *X* and *W*, as shown in Figure 4a, whereas the hypothesized model (Figure 4b) has no such connection. Statistically, the two models differ in the term $\rho_{XW \cdot Z}$, which should vanish according to Figure 4b and is left free according to Figure 4a.

Once the nature of the discrepancy is clear, the investigator must decide whether substantive knowledge justifies alteration of the model, namely, adding either a link or a curved arc between *X* and *W*. However, because the effect of the discrepancy will be spread over several covariance terms, global fitness tests will not be able to isolate the discrepancy easily. Even multiple fitness tests on various local modifications of the model (such tests are provided by LISREL) may not help much, since the results may be skewed by other discrepancies in different parts of the model, such as the subgraph rooted at *Y*. Thus, testing for global fitness is often of only minor use in model debugging.

Local fitness testing is an attractive alternative to global fitness testing. This involves listing the restrictions implied by the model and testing them one by one. Local testing may help isolate the discrepancy and can be performed more reliably than testing the overall model as one unit. A restriction such as $\rho_{XW \cdot Z} = 0$, for example, can be tested locally without measuring *Y* or any of its descendants, thus keeping errors associated with those measurements from interfering with the test for $\rho_{XW \cdot Z} = 0$, which is the real source of the lack of fit. More generally, typical SEM models are often close to being "saturated," claiming but a few restrictions, in the form of a few edges missing from large, otherwise unrestrictive diagrams. Local and direct tests for those

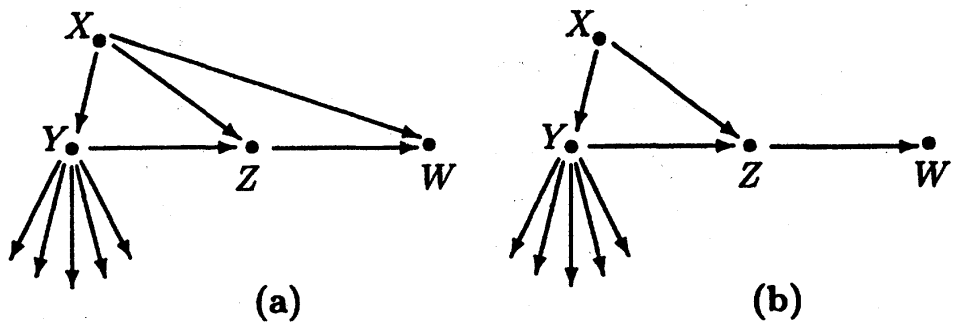

Figure 4: Models Differing in One Local Test, $\rho_{XW \cdot Z} = 0$

restrictions are more reliable than global tests, since they involve fewer degrees of freedom and are not contaminated with irrelevant measurement errors. The missing edges approach described in Section 2.1 provides a systematic way of detecting and enumerating the local tests needed for testing a given model.

2.3. MODEL EQUIVALENCE

An important criterion for determining whether two given causal models are observationally equivalent follows from the d-separation test.

Definition 3: (observational equivalence) Two SEMs are said to be observationally equivalent if every probability distribution that is generated by one of the models can also be generated by the other.

Theorem 4: Two Markovian models are observationally equivalent if and only if they entail the same sets of conditional independencies. Moreover, two such models are observationally equivalent if and only if their corresponding graphs have the same sets of edges and the same sets of v-structures (two converging arrows whose tails are not connected by an arrow) (Verma and Pearl 1990b).

In standard SEM, models are assumed linear and data are characterized by covariance matrices. Thus, two such models are observationally indistinguishable if they are covariance equivalent, that is, if every covariance matrix generated by one model (through some choice of parameters) can also be generated by the other. It can be easily verified that Theorem 4 extends to covariance equivalence.

Theorem 5: Two Markovian linear normal models are covariance equivalent if and only if they entail the same sets of zero partial correlations. Moreover, two such models are covariance equivalent if and only if their corresponding graphs have the same sets of edges and the same sets of ν-structures.

In Theorems 4 and 5, the first part defines the testable implications of any Markovian SEM. These theorems state that, in nonmanipulative studies, Markovian SEMs cannot be tested for any feature other than those zero partial correlations that the d-separation test reveals. They provide as well a simple test for equivalence that requires, instead of the checking of all d-separation conditions, merely a comparison of corresponding edges and their directionalities.

For example, reversing the direction of the arrow between X_1 and X_2 in Figure 1 does not introduce any new ν-structure. Therefore, this reversal yields an observationally equivalent network, and the directionality of the link $X_1 \rightarrow X_2$ cannot be determined from statistical information. The arrows $X_2 \rightarrow X_4$ and $X_4 \rightarrow X_5$ are of a different nature, however; their directionality cannot be reversed without creating a new ν-structure. Thus, we see that some arrows retain their directionality in all models equivalent to a given model and, hence, that this directionality is testable whenever the equivalence class (of models) is testable. Algorithms for automatically identifying such arrows in the graph have been devised by Chickering (1995), Meek (1995), and Andersson et al. (1997). We further see that some kinds of statistical data (such as those generated by the model in Figure 1), unaccompanied by temporal information, can reveal the directionality of some arrows and, hence, the directionality of the causal relationships among the corresponding variables. This feature is used in various discovery algorithms that elicit causal relationships from complex patterns of statistical associations (e.g., Pearl and Verma 1991; Spirtes, Glymour, and Scheines 1993), but discussion of such algorithms lies beyond the scope of this article.

In semi-Markovian models (DAGs with correlated errors), the d-separation criterion is still valid for testing independencies (see Theorem 2), but independence equivalence no longer implies observational equivalence.[10] Two models that entail the same set of zero partial correlations among the observed variables may yet impose

different inequality constraints on the covariance matrix. Nevertheless, Theorems 2 and 4 still provide necessary conditions for testing equivalence.

2.3.1. Generating Equivalent Models

By permitting arrows to be reversed as long as no ν-structures are destroyed or created, we can use Theorems 4 and 5 to generate equivalent alternatives to any Markovian model. Meek (1995) and Chickering (1995) have shown that $X \rightarrow Y$ can be replaced by $X \leftarrow Y$ if and only if all parents of X are also parents of Y, and, moreover, that for any two equivalent models, there is always some sequence of such edge reversals that takes one model into the other. This simple rule for edge reversal coincides with those proposed by Stelzl (1986) and Lee and Hershberger (1990).

In semi-Markovian models, the rules for generating equivalent models are more complicated. Nevertheless, Theorems 4 and 5 yield convenient graphical principles for testing the correctness of edge-replacement rules.

The basic principle is that if we regard each bidirected arc $X \leftarrow\!-\!-\!\rightarrow Y$ as representing a latent common cause $X \leftarrow L \rightarrow Y$, then the "if" part of Theorem 4 remains valid; that is, any edge replacement transformation that does not destroy or create a ν-structure is allowed. Thus, for example, an edge $X \rightarrow Y$ can be replaced by a bidirected arc $X \leftarrow\!-\!-\!\rightarrow Y$ whenever X and Y have no other parents, latent or observed. Likewise, an edge $X \rightarrow Y$ can be replaced by a bidirected arc $X \leftarrow\!-\!-\!\rightarrow Y$ whenever (1) X and Y have no latent parents and (2) every parent of X or Y is a parent of both. Such replacements do not introduce new ν-structures. Because ν-structures may now involve latent variables, however, we can tolerate the creation or destruction of some ν-structures as long as this does not affect partial correlations among the observed variables. Figure 5a demonstrates that the creation of certain ν-structures can be tolerated. If we reverse the arrow $X \rightarrow Y$, we create two converging arrows $Z \rightarrow X \leftarrow Y$ whose tails are connected, not directly, but through a latent common cause. This is tolerated because, although the new convergence at X blocks the path (Z, X, Y), the connection between Z and Y (through the arc $Z \leftarrow\!-\!-\!\rightarrow Y$)

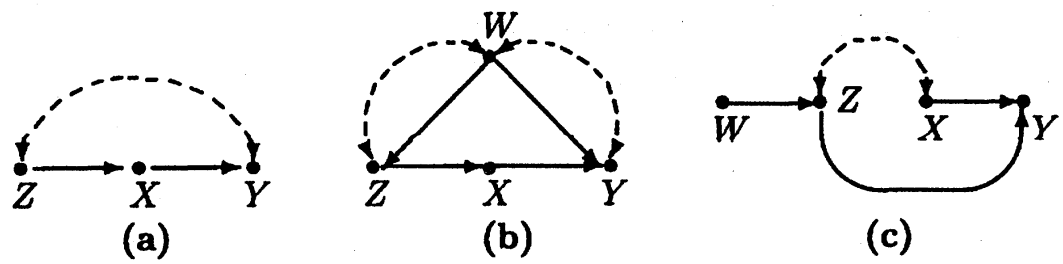

Figure 5: Models Permitting ([a] and [b]) and Forbidding (c) the Reversal of $X \rightarrow Y$

remains unblocked and, in fact, cannot be blocked by any set of observed variables.

We can carry this principle further by generalizing the concept of v-structure. Whereas in Markovian models, a v-structure is defined as two converging arrows whose tails are not connected by a link, we now define v-structure as any two converging arrowheads whose tails are separable. By separable, we mean that there exists a conditioning set S capable of d-separating the two tails. Clearly, the two tails will not be separable if they are connected by an arrow or by a bidirected arc. But a pair of nodes in a semi-Markovian model can be inseparable even when not connected by an edge (Verma and Pearl 1990b). With this generalization in mind, we can state necessary conditions for edge replacement:

Rule 1: An arrow $X \rightarrow Y$ is interchangeable with $X \leftarrow - - \rightarrow Y$ only if every neighbor or parent of X is inseparable from Y (by neighbor, we mean a node connected—to X—through a bidirected arc).

Rule 2: An arrow $X \rightarrow Y$ can be reversed into $X \leftarrow Y$ only if, before reversal, (i) every neighbor or parent of Y (excluding X) is inseparable from X and (ii) every neighbor or parent of X is inseparable from Y.

For example, consider the model $Z \leftarrow - - \rightarrow X \rightarrow Y$. The arrow $X \rightarrow Y$ cannot be replaced with a bidirected arc $X \leftarrow - - \rightarrow Y$ because Z (a neighbor of X) is separable from Y by the set $S = \{X\}$. Indeed, the new v-structure created at X would render X and Y marginally independent, contrary to the original model.

As another example, consider the graph in Figure 5a. Here, it is legitimate to replace $X \rightarrow Y$ with $X \leftarrow - - \rightarrow Y$ or a reversed arrow $X \leftarrow Y$ because X has no neighbors and Z, the only parent of X, is

inseparable from Y. The same considerations apply to Figure 5b; variables Z and Y, although nonadjacent, are inseparable, because the paths going from Z to Y through W cannot be blocked.

A more complicated example, one that demonstrates that the rules above are not sufficient to ensure the legitimacy of a transformation, is shown in Figure 5c. Here, it appears that replacing $X \rightarrow Y$ with $X \leftarrow - - \rightarrow Y$ would be legitimate because the (latent) v-structure at X is shunted by the arrow $Z \rightarrow Y$. However, the original model shows the path from W to Y to be d-connected given Z, whereas the postreplacement model shows the same path d-separated given Z. Consequently, the partial correlation $\rho_{WY \cdot Z}$ vanishes in the postreplacement model but not in the prereplacement model. A similar disparity also occurs relative to the partial correlation $\rho_{WY \cdot ZX}$. The original model shows that the path from W to Y is blocked given $\{Z, X\}$, whereas the postreplacement model shows that path d-connected given $\{Z, X\}$. Consequently, the partial correlation $\rho_{WY \cdot ZX}$ vanishes in the prereplacement model but is unconstrained in the postreplacement model.[11] Evidently, it is not enough to impose rules on the parents and neighbors of X; remote ancestors (e.g., W) should be considered too.

These rules are just a few of the implications of the d-separation criterion when applied to semi-Markovian models. A necessary and sufficient criterion for testing the d-separation equivalence of two semi-Markovian models has been devised by Spirtes and Verma (1992). Spirtes and Richardson (1996) have extended that criterion to include models with selection bias. We should keep in mind, however, that because two semi-Markovian models can be zero partial correlation equivalent and yet not covariance equivalent, criteria based on d-separation can provide merely the necessary conditions for model equivalence.

2.3.2. The Significance of Equivalent Models

Theorem 4 is methodologically significant because it clarifies what it means to claim that structural models are "testable" (Bollen 1989:78).[12] It asserts that we never test a model but, rather, a whole class of observationally equivalent models from which the hypothesized model cannot be distinguished by any statistical means. It asserts

as well that this equivalence class can be constructed by inspection, from the graph, which thus provides the investigator with a vivid representation of competing alternatives for consideration. Graphs representing all models in a given equivalence class have been given by Verma and Pearl (1990b), Spirtes et al. (1993), and Andersson et al. (1997). Richardson (1996) discusses the representation of equivalence classes of models with cycles.

Although it is true that (overidentified) SEMs have testable implications, those implications are but a small part of what the model represents, namely, a set of claims, assumptions, and implications. Failure to distinguish among causal assumptions, statistical implications, and policy claims has been one of the main reasons for the suspicion and confusion surrounding quantitative methods in the social sciences (Freedman 1987:112; Goldberger 1992; Wermuth 1992). However, because they make the distinctions among these components vivid and crisp, graphical methods promise to make SEM more acceptable to researchers from a wide variety of disciplines.

By and large, the SEM literature has ignored the explicit analysis of equivalent models. Breckler (1990), for example, found that out of 72 articles in the areas of social and personality psychology, only 1 acknowledged the existence of an equivalent model. The general attitude has been that the combination of data fitness and model overidentification is sufficient to confirm the hypothesized model. Recently, however, the existence of multiple equivalent models seems to have jangled the nerves of some SEM researchers. MacCallum et al. (1993:198) conclude that "the phenomenon of equivalent models represents a serious problem for empirical researchers using CSM" and "a threat to the validity of interpretation of CSM results." Breckler (1990:262) reckons that "if one model is supported, so too are all of its equivalent models," and consequently ventures that "the term *causal modeling* is a misnomer."

Such extremes are not justifiable. The existence of equivalent models is logically inevitable if we accept the fact that causal relations cannot be inferred from statistical data alone; as Wright (1921) stated, "Prior knowledge of the causal relations is assumed as prerequisite" in SEM. But this does not make SEM useless as a tool for causal modeling. The move from the qualitative causal premises represented

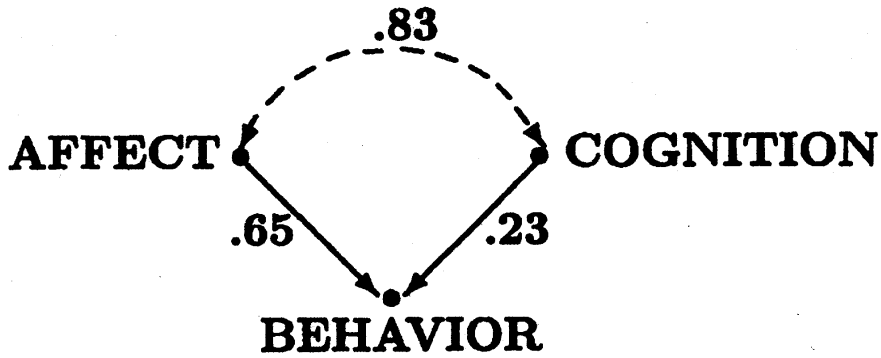

Figure 6: Untestable Model Displaying Quantitative Causal Information Derived From Qualitative Substantive Assumptions

by the structure of a path diagram (see note 3) to the quantitative causal conclusions advertised by the coefficients in the diagram is neither useless nor trivial. Consider, for example, the model depicted in Figure 6, which Bagozzi and Burnkrant (1979) use to illustrate problems associated with equivalent models. Although this model is saturated (i.e., just identified), and although it has (at least) 27 semi-Markovian equivalent models, finding that the influence of AFFECT on BEHAVIOR is almost three times stronger (on a standardized scale) than the influence of COGNITION on BEHAVIOR is still very illuminating—it tells us about the relative effectiveness of different behavior modification policies if some are known to influence AFFECT and others COGNITION. The significance of this quantitative analysis on policy analysis may be more dramatic when a path coefficient turns negative while the corresponding correlation coefficient measures positive. Learning that such a reversal is logically implied by the qualitative causal premises embedded in the diagram may have a profound impact on policy decisions.

In summary, social scientists need not abandon SEM altogether; they need only abandon the notion that SEM is a method of *testing* causal models. SEM is a method of testing a tiny fraction of the premises that make up a causal model, and, in cases where that fraction is found to be compatible with the data, SEM elucidates the necessary quantitative consequences of both the premises and the data. It follows, then, that users of SEM should concentrate on examining the implicit theoretical premises that enter into a model. As we will see in Section 4, graphical methods make these premises vivid and precise.

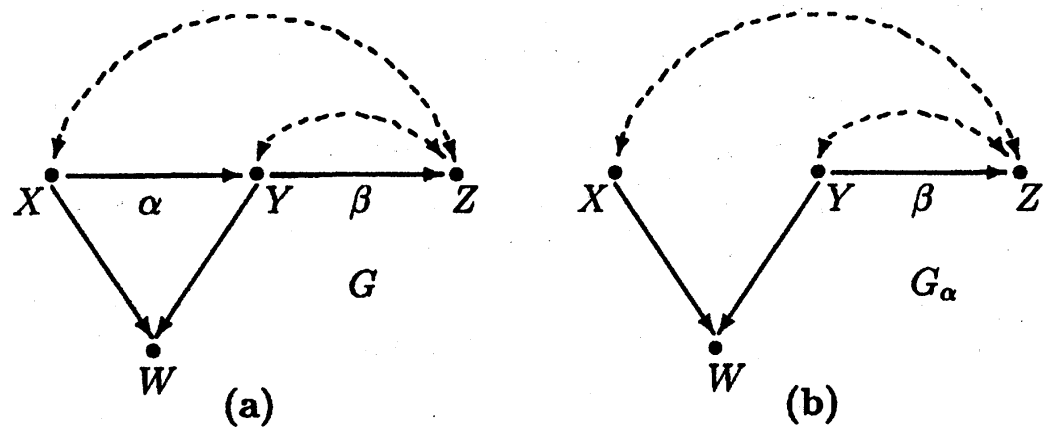

Figure 7: Testing Whether Structural Parameter α Can Be Equated With Regression Coefficient r_{YX}

3. GRAPHS AND IDENTIFIABILITY

3.1. PARAMETER IDENTIFICATION IN LINEAR MODELS

Consider a directed edge $X \rightarrow Y$ embedded in a path diagram G, and let α stand for the path coefficient associated with that edge. It is well known that the regression coefficient $r_{YX} = \rho_{XY}\sigma_Y/\sigma_X$ can be decomposed into the sum

$$r_{YX} = \alpha + I_{YX},$$

where I_{YX} is not a function of α, since it is computed (e.g., using Wright's rules) from other paths connecting X and Y excluding the edge $X \rightarrow Y$ (such paths traverse both unidirected and bidirected arcs). Thus, if we remove the edge $X \rightarrow Y$ from the path diagram and find that the resulting subgraph entails zero correlation between X and Y, then we know that $I_{YX} = 0$ and $\alpha = r_{YX}$; hence, α is identified. Such entailment can be established graphically by testing whether X is d-separated from Y (by the empty set $Z = \{\varnothing\}$) in the subgraph. Figure 7 illustrates this simple test for identification: All paths between X and Y in the subgraph G_α are blocked by converging arrows, and α can immediately be equated with r_{YX}.

We can extend this basic idea to cases in which I_{YX} is not zero but can be made zero by adjusting for a set of variables $Z = \{Z_1, Z_2, \ldots, Z_k\}$ that lie on various d-connected paths between X and Y. Consider

the partial regression coefficient $r_{YX \cdot Z} = \rho_{YX \cdot Z}\, \sigma_{Y \cdot Z} / \sigma_{X \cdot Z}$, which represents the residual correlation between Y and X after Z is "partialed out." If Z contains no descendant of Y, then again we can write[13]

$$r_{YX \cdot Z} = \alpha + I_{YX \cdot Z},$$

where $I_{YX \cdot Z}$ represents the partial correlation between X and Y resulting from setting α to zero, that is, the partial correlation in a model whose graph, G_α, lacks the edge $X \rightarrow Y$ but is otherwise identical to G. If Z d-separates X from Y in G_α, then $I_{YX \cdot Z}$ would indeed be zero in such a model, and we can conclude that in our original model, α is identified and is equal to $\alpha = r_{YX \cdot Z}$. Moreover, since $r_{YX \cdot Z}$ is given by the coefficient of x in the regression of Y on X and Z, α can be estimated using the regression

$$y = \alpha x + \beta_1 z_1 + \ldots + \beta_k z_k + \varepsilon.$$

This result provides a simple graphical answer to the questions, alluded to in Section 1.3, of what constitutes an adequate set of regressors and when a regression coefficient provides a consistent estimate of a path coefficient. The answers are summarized in the following theorem.[14]

Theorem 6: (single-link criterion) Let G be any path diagram in which α is the path coefficient associated with link $X \rightarrow Y$, and let G_α denote the diagram that results when $X \rightarrow Y$ is deleted from G. The coefficient α is identifiable if there exists a set of variables Z such that Z contains no descendant of Y, and Z d-separates X from Y in G_α. If Z satisfies these two conditions, then α is equal to the regression coefficient $r_{YX \cdot Z}$. Conversely, if Z does not satisfy these conditions, then $r_{YX \cdot Z}$ is not a consistent estimator of α, except in rare instances of measure zero.

The use of Theorem 6 can be illustrated as follows. Consider the graphs G and G_α in Figure 8. The only path connecting X and Y in G_α is the one traversing Z, and since that path is d-separated (blocked) by Z, α is identifiable and is given by $\alpha = r_{YX \cdot Z}$. The coefficient β is identifiable, of course, since Z is d-separated from X in G_β (by the empty set $\{\emptyset\}$), and thus $\beta = r_{XZ}$.

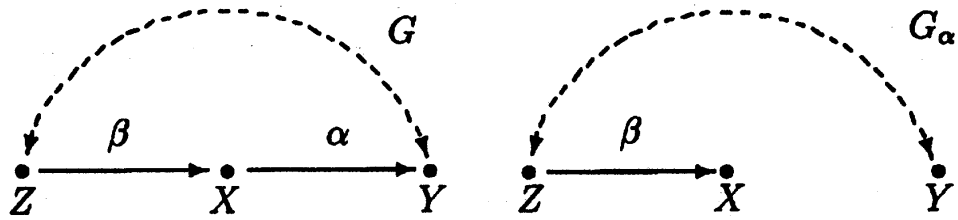

Figure 8: Illustrating the Identification of α (Theorem 6)

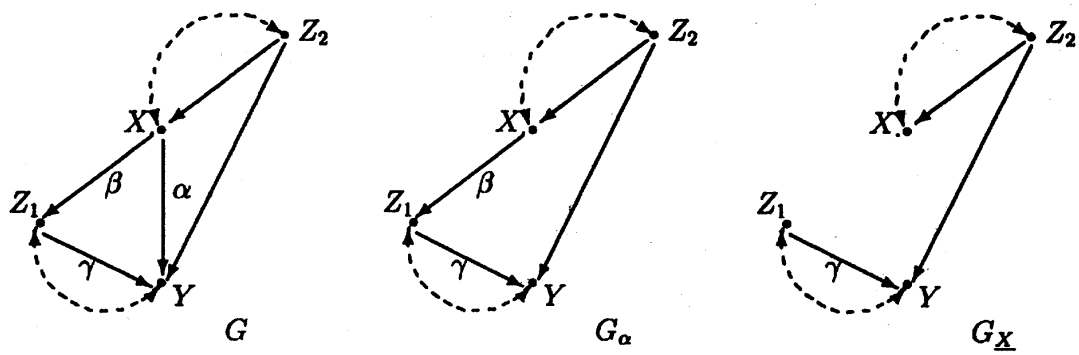

Figure 9: Graphical Identification of the Total Effect of X on Y, $\alpha + \beta\gamma = r_{YX \cdot Z_2}$

We now extend the use of d-separation to facilitate the identification of total, rather than direct, effects. Consider the graph G in Figure 9. If we form the graph G_α (by removing the link $X \rightarrow Y$), we observe that there is no set Z of nodes that d-separates all paths from X to Y. If Z contains Z_1, then the path $X \rightarrow Z_1 \leftarrow - - \rightarrow Y$ will be unblocked through the converging arrows at Z_1. If Z does not contain Z_1, the path $X \rightarrow Z_1 \rightarrow Y$ is unblocked. Thus, we conclude that α cannot be identified using our previous method. However, suppose we are interested in the total effect of X on Y given by $\alpha + \beta\gamma$. For this sum to be identified by r_{YX}, there should be no contribution to r_{YX} from paths other than those leading from X to Y. However, we see that two such paths, called confounding or backdoor paths, exist in the graph, namely, $X \leftarrow Z_2 \rightarrow Y$ and $X \leftarrow - - \rightarrow Z_2 \rightarrow Y$. Fortunately, these paths are blocked by Z_2, and we conclude that adjusting for Z_2 would render $\alpha + \beta\gamma$ identifiable and given by

$$\alpha + \beta\gamma = r_{YX \cdot Z_2}.$$

This line of reasoning leads to a general test for the identifiability of total effects, called the backdoor criterion (Pearl 1993a; Pearl 1995).

Theorem 7: (backdoor criterion) For any two variables X and Y in a causal diagram G, the total effect of X on Y is identifiable if there exists a set of measurements Z such that

1. no member of Z is a descendant of X
2. Z d-separates X from Y in the subgraph $G_{\underline{X}}$ formed by deleting from G all arrows emanating from X.

Moreover, if the two conditions are satisfied, then the total effect of X on Y is given by $r_{YX \cdot Z}$.

The two conditions of Theorem 7, as we will see in the next subsection, are also valid in nonlinear non-Gaussian models, as well as in models with discrete variables. It is for this reason that the backdoor criterion can serve as a general test for covariate selection, as described in Section 1.3. The reason for deleting the arrows in step 2 is to ensure that only confounding (i.e., backdoor) paths participate in the d-separation test. The test ensures that, after adjustment for Z, X and Y are not associated through confounding paths, which means that the partial correlation $r_{YX \cdot Z}$ is equal to the total effect. In fact, we can view Theorems 6 and 7 as special cases of a more general scheme: To identify any partial effect, as defined by a select bundle of causal paths from X to Y, we ought to find a set Z of measured variables that block all nonselected paths between X and Y. The partial effect will then equal the regression coefficient $r_{YX \cdot Z}$.

Figure 9 demonstrates that some total effects can be determined directly from the graphs, without having to identify their individual components. Standard SEM methods (Bollen 1989; Chou and Bentler 1995), which focus on the identification and estimation of individual parameters, may miss the identification and estimation of effects such as the one in Figure 9, which can be estimated reliably even though some of the constituents remain unidentified.

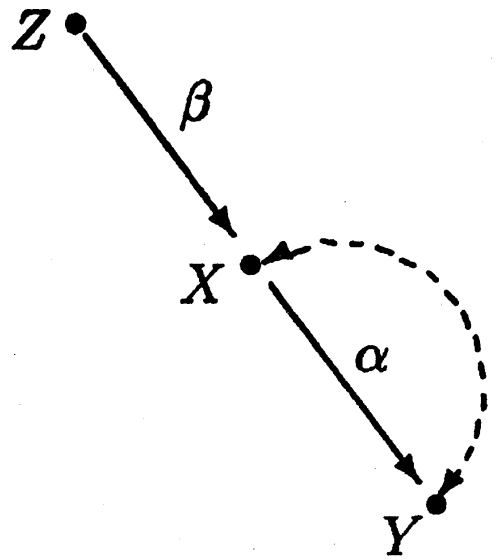

Figure 10: Graphical Identification of α Using Instrumental Variable Z

Some total effects cannot be determined directly, as a unit, but require the determination of each component separately. In Figure 8, for example, the effect of Z on $Y(= \alpha\beta)$ does not meet the backdoor criterion, yet this effect can be determined from its constituents α and β, which meet the backdoor criterion individually and evaluate to

$$\beta = r_{XZ} \qquad \alpha = r_{YX \cdot Z}.$$

There is still a third kind of causal parameter that cannot be determined directly or through its constituents but requires the evaluation of a broader causal effect of which it is a part. The structure shown in Figure 10 represents an example of this case. The parameter α cannot be identified directly, yet it can be determined from $\alpha\beta$ and β, which represent the effect of Z on Y and that of Z on X, respectively. These two effects can be identified directly, since there are no backdoor paths from Z to either Y or X, giving $\alpha\beta = r_{YZ}$ and $\beta = r_{XZ}$. Thus,

$$\alpha = r_{YZ} / r_{XZ},$$

which is familiar to us as the instrumental variable formula (Bowden and Turkington 1984).

The example shown in Figure 11 combines all three methods considered thus far. The total effect of X on Y is given by $\alpha\beta + \gamma\delta$, which is not identifiable because it does not meet the backdoor

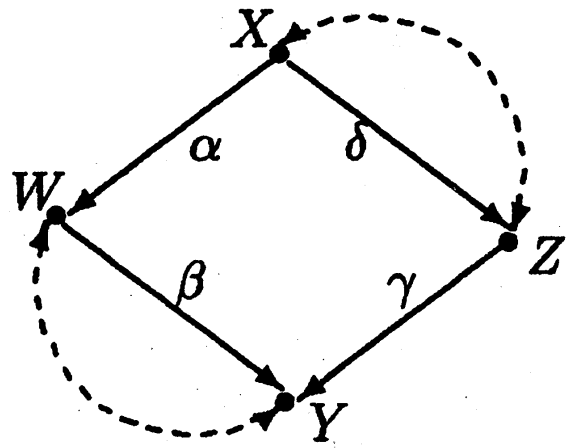

Figure 11: Graphical Identification of α, β and γ

criterion and is not part of another identifiable structure. However, suppose we wish to estimate β. By conditioning on Z, we block all paths going through Z and obtain $\alpha\beta = r_{YX \cdot Z}$, which is the effect of X on Y mediated by W. Because there are no backdoor paths from X to W, α itself evaluates directly to $\alpha = r_{WX}$. We therefore obtain

$$\beta = r_{YX \cdot Z} / r_{WX}.$$

In contrast, γ can be evaluated directly by conditioning on X (thus blocking the backdoor path from Z to Y through X), which gives

$$\gamma = r_{YZ \cdot X}.$$

The methods we have been using suggest a systematic procedure for recognizing identifiable coefficients in a graph.

1. Start by searching for identifiable causal effects among pairs of variables in the graph using the backdoor criterion and Theorem 6. These can be either direct effects, total effects, or partial effects, that is, effects mediated by specific sets of variables.
2. For any such identified effect, collect the path coefficients involved and put them in a bucket.
3. Begin labeling the coefficients in the buckets according to the following procedure: If a bucket is a singleton, label its coefficient I (denoting identifiable); if a bucket is not a singleton but contains only a single unlabeled element, label that element I.
4. Repeat this process until no new labeling is possible.
5. List all labeled coefficients; these are identifiable.

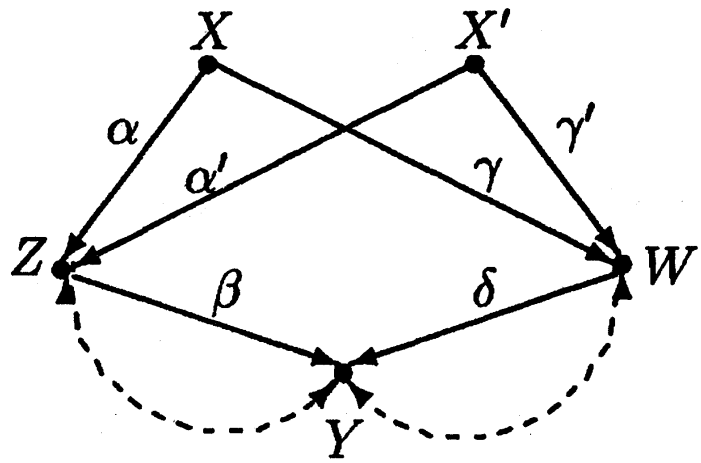

Figure 12: Identifying β and δ Using Two Instrumental Variables

The process described above is not complete, since our insistence on labeling coefficients one at a time may cause us to miss certain opportunities. This is shown in Figure 12.

Starting with the pairs (X, Z), (X, W), (X', Z), and (X', W), we discover that α, γ, α', and γ' are identifiable. Going to (X, Y), we find that $\alpha\beta + \delta\gamma$ is identifiable, and, from (X', Y), that $\alpha'\beta + \gamma'\delta$ is identifiable. This does not enable us to label β or δ yet, but we can solve two equations for the unknowns β and δ as long as the determinant $\begin{vmatrix} \alpha & \gamma \\ \alpha' & \gamma' \end{vmatrix}$ is nonzero. Because we are interested in identifiability not at a point but rather "almost everywhere" (Koopmans et al. 1950; Simon 1953), we need not compute this determinant. We merely inspect the symbolic form of the determinant's rows to make sure that the equations are nonredundant; each imposes a new constraint on the unlabeled coefficients for at least one value of the labeled coefficients.

With a facility to detect redundancies, we can increase the power of our procedure by adding the following provision to Step 3:

> If there are k nonredundant buckets that contain at most k unlabeled coefficients, label these coefficients, and continue.

Another way to increase the power of our procedure is to list not just identifiable effects, but also expressions involving correlations due to bidirected arcs, in accordance with Wright's rules. Finally, one

can endeavor to list effects of several variables jointly. A modified backdoor criterion for evaluating joint effects has been reported by Pearl and Robins (1995). However, such enrichments tend to make the procedure more complex and might compromise our main objective of providing investigators with a way of immediately recognizing the identified coefficients in a given model and immediately understanding those features in the model that influence the identifiability of the target quantity. We now address the problem of identification in nonparametric models, where the machinery of linear algebra can be of little help and where graph theoretical techniques have led to significant progress.

3.2. IDENTIFICATION IN NONPARAMETRIC MODELS

Nonparametric models are SEMs in which both the functional forms of the equations and the probability distributions of the disturbances remain unspecified. We consider nonparametric models for both practical and conceptual reasons. On the practical side, investigators often find it hard to defend the assumptions of linearity and normality, or other functional-distributional assumptions, especially when categorical variables are involved. Nonparametric results are valid for nonlinear functions and for any distribution of errors. Moreover, having such results allows us to gauge how sensitive standard techniques are to assumptions of linearity and normality. On the conceptual side, nonparametric models, which are stripped of algebraic connotations, illuminate the distinctions between structural and algebraic equations. The search for alternatives to path coefficients (which are nonexistent in nonparametric models) and to the standard definitions of direct and total causal effects (which are normally defined in terms of path coefficients) forces explication of what path coefficients really mean and where their empirical content comes from.

3.2.1. Parametric Versus Nonparametric Models: An Example

Consider the set of structural equations

$$x = f_1(u, \varepsilon_1) \tag{6}$$

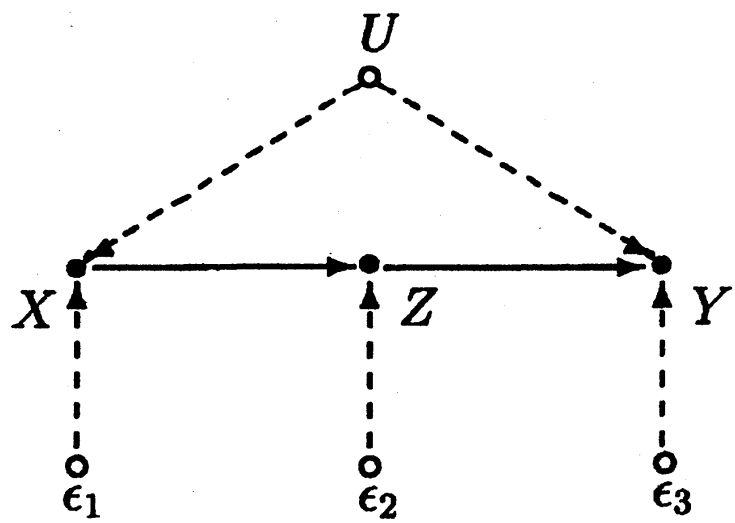

Figure 13: Path Diagram Corresponding to Equations (6)-(8), Where {X, Y, Z} Are Observed and {U, ε1, ε2, ε3} Are Unobserved

$$z = f_2(x, \varepsilon_2) \tag{7}$$

$$y = f_3(z, u, \varepsilon_3), \tag{8}$$

where X, Y, and Z are observed variables; f_1, f_2, and f_3 are unknown arbitrary functions; and U, ε_1, ε_2, and ε_3 are unobservables that we can regard either as latent variables or as disturbances. For the sake of this discussion, we will assume that U, ε_1, ε_2, and ε_3 are mutually independent and arbitrarily distributed. Graphically, these influences can be represented by the path diagram of Figure 13.

The problem is as follows: We have drawn a long stream of independent samples of the process defined by equations (6) through (8) and have recorded the values of the observed variables X, Z, and Y, and we now wish to estimate the unspecified quantities of the model to the greatest extent possible.

To clarify the scope of the problem, let us consider its linear version, which is given by

$$x = u + \varepsilon_1 \tag{9}$$

$$z = \alpha x + \varepsilon_2 \tag{10}$$

$$y = \beta z + \gamma u + \varepsilon_3, \tag{11}$$

where U, ε_1, ε_2, and ε_3 are uncorrelated, zero-mean disturbances.[15] It is not hard to show that parameters α, β, and γ can be determined uniquely from the correlations among the observed quantities X, Z, and Y. This identification was demonstrated already in the example of Figure 8, where the backdoor criterion yielded

$$\beta = r_{YZ \cdot X} \qquad \alpha = r_{ZX}, \tag{12}$$

and hence

$$\gamma = r_{YX} - \alpha\beta. \tag{13}$$

Thus, returning to the nonparametric version of the model, it is tempting to generalize that for the model to be identifiable, the functions $\{f_1, f_2, f_3\}$ must be determined uniquely from the data. However, the prospect of this happening is unlikely because the mapping between functions and distributions is known to be many to one. In other words, given any nonparametric model M, if there exists one set of functions $\{f_1, f_2, f_3\}$ compatible with a given distribution $P(x, y, z)$, then there are infinitely many such functions. Thus, it seems that nothing useful can be inferred from loosely specified models such as the one given by equations (6) through (8).

Identification is not an end in itself, however, even in linear models. Rather, it serves to answer practical questions of prediction and control. At issue is not whether the data permit us to identify the form of the equations but rather whether the data permit us to provide unambiguous answers to questions of the kind traditionally answered by parametric models.

When the model given by equations (6) through (8) is used strictly for prediction (i.e., to determine the probabilities of some variables given a set of observations on other variables), the structural content of the parameters becomes irrelevant; the predictions can be estimated directly from either the covariance matrices or the sample estimates of those covariances. If dimensionality reduction is needed (e.g., to improve estimation accuracy), the covariance matrix can be encoded in a variety of simultaneous equation models, all of the same dimensionality. For example, the correlations among X, Y, and Z in the linear model M of equations (9) through (11) might well be represented by the model M' (Figure 14):

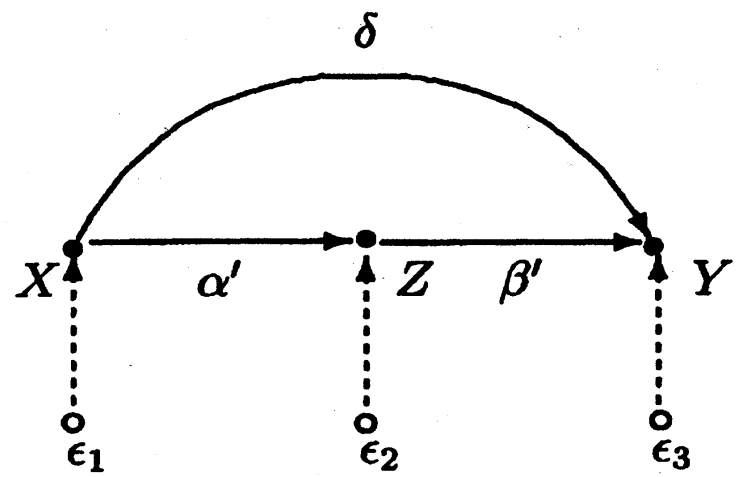

Figure 14: Diagram Representing Model M' of Equations (14) Through (16)

$$x = \varepsilon_1 \tag{14}$$

$$z = \alpha x + \varepsilon_2 \tag{15}$$

$$y = \beta z + \delta x + \varepsilon_3, \tag{16}$$

which is as compact as equations (9) through (11) and is covariance equivalent to M with respect to the observed variables X, Y, and Z. Upon setting $\alpha' = \alpha$, $\beta' = \beta$, and $\delta = \gamma$, model M' will yield the same probabilistic predictions as those of the model of equations (9) through (11). Still, when viewed as data-generating mechanisms, the two models are not equivalent; each tells a different story about the processes generating X, Y, and Z, and, naturally, their predictions about the changes that would result from subjecting these processes to external interventions differ.

3.3. CAUSAL EFFECTS: THE INTERVENTIONAL INTERPRETATION OF SEMs

The differences between models M and M' illustrate precisely where the structural reading of simultaneous equation models comes into play. Model M', defined by equations (14) through (16), regards X as a direct participant in the process that determines the value of Y, whereas model M, defined by equations (9) through (11), views X as an indirect factor whose effect on Y is mediated by Z. This difference

is manifested not in the data but in the way the data would change in response to outside interventions. For example, suppose we wish to predict the expectation of Y after we intervene and fix the value of X to some constant x, denoted $E(Y \mid \mathrm{do}(X=x))$.[16] After $X=x$ is substituted into equations (15) and (16), model M' yields

$$E[Y \mid \mathrm{do}(X=x)] = E[\beta'\alpha' x + \beta'\varepsilon_2 + \delta x + \varepsilon_3] \tag{17}$$

$$= (\beta'\alpha' + \delta)x, \tag{18}$$

whereas model M yields

$$E[Y \mid \mathrm{do}(X=x)] = E[\beta\alpha x + \beta\varepsilon_2 + \gamma u + \varepsilon_3] \tag{19}$$

$$= \beta\alpha x. \tag{20}$$

Upon setting $\alpha' = \alpha$, $\beta' = \beta$, and $\delta = \gamma$ (as required for covariance equivalence; see equations (12) and (13)), we see clearly that the two models assign different magnitudes to the (total) causal effect of X on Y; model M predicts that a unit change in x will change $E(Y)$ by the amount $\beta\alpha$, whereas model M' puts this amount at $\beta\alpha + \gamma$.

At this point, it is tempting to ask whether we should substitute $x - \varepsilon_1$ for u in equation (11) prior to taking expectations in equation (19). If we permit the substitution of equation (10) into equation (11), as we did in deriving equation (19), why not permit the substitution of equation (9) into equation (11) as well? After all, so the argument goes, there is no harm in upholding a mathematical equality, $u = x - \varepsilon_1$, that the modeler deems valid. This argument is fallacious, however. Structural equations are not meant to be treated as immutable mathematical equalities. Rather, they are introduced into the model to describe a state of equilibrium, and they are violated when that equilibrium is perturbed by outside interventions. In fact, the power of SEMs is that they encode not only the initial equilibrium state but also the information necessary for determining which equations must be violated to account for a new state of equilibrium. For example, if the intervention merely consists of holding X constant at x, then the equation $x = u + \varepsilon_1$, which represents the preintervention process determining X, should be overruled and replaced with the equation

$X = x$. The solution to the new set of equations then represents the new equilibrium. Thus, the essential characteristic of structural equations that sets them apart from ordinary mathematical equations is that they stand not for one but for many sets of equations, each corresponding to a subset of equations taken from the original model. Every such subset represents some hypothetical physical reality that would prevail under a given intervention.

If we take the stand that the value of structural equations lies not in summarizing distribution functions but in encoding causal information for predicting the effects of policies (Haavelmo 1943; Marschak 1950; Koopmans 1953), it is natural to view such predictions as the proper generalization of structural coefficients. For example, the proper generalization of the coefficient β in the linear model M would be the answer to the control query, "What would be the change in the expected value of Y if we were to intervene and change the value of Z from z to $z + 1$?" which is different, of course, from the observational query, "What would be the difference in the expected value of Y if we were to *find* Z at level $z + 1$ instead of level z?" Observational queries can be answered directly from the joint distribution $P(x, y, z)$, whereas control queries require causal information as well. Structural equations encode this causal information in their syntax by treating the variable on the left-hand side of the equality sign as the effect and those on the right-hand side as causes. To distinguish between the two types of queries, we will use the symbol $do(\cdot)$ to indicate externally controlled quantities. For example, we write

$$E(Y \mid do(x)) \triangleq E[Y \mid do(X{=}x)] \tag{21}$$

for the controlled expectation and

$$E(Y \mid x) \triangleq E(Y \mid X{=}x) \tag{22}$$

for the standard conditional or observational expectation. That $E(Y \mid do(x)) \neq E(Y \mid x)$ can easily be seen in the model of equations (9) through (11), where $E(Y \mid do(x)) = \alpha\beta x$ but $E(Y \mid x) = r_{YX}x = (\alpha\beta + \gamma)x$. Indeed, the passive observation $X = x$ should not violate any of the equations, and this is the justification for substituting both equations (9) and (10) into equation (11) before taking the expectation.

In linear models, the answers to questions of direct control are encoded in the so-called path coefficients or structural coefficients, and these can be used to derive the total effect of any variable on another. For example, the value of $E(Y \mid do(x))$ in the model defined by equations (9) through (11) is $\alpha\beta x$, namely, x times the product of the path coefficients along the path $X \rightarrow Z \rightarrow Y$. In the nonparametric case, computation of $E(Y \mid do(x))$ would be more complicated, even when we know the functions f_1, f_2, and f_3. Nevertheless, this computation is well defined and requires the solution (for the expectation of Y) of a modified set of equations in which f_1 is "wiped out" and X is replaced by the constant x:

$$z = f_2(x, \varepsilon_2) \tag{23}$$

$$y = f_3(z, u, \varepsilon_3). \tag{24}$$

Thus, computation of $E(Y \mid do(x))$ requires evaluation of

$$E(Y \mid do(x)) = E\{f_3[f_2(x, \varepsilon_2), u, \varepsilon_3]\},$$

where the expectation is taken over U, ε_2, and ε_3. Graphical methods for performing this computation are discussed in Section 3.4.

What, then, is an appropriate definition of identifiability for nonparametric models? One reasonable definition is that answers to interventional queries are unique. Accordingly, we call a model identifiable if there exists a consistent estimate for every query of the type "Find $P(r \mid do(s)) \triangleq P[R = r \mid do(S = s)]$," where R and S are subsets of observables and r and s are any realizations of these variables. The set of probabilities $P(r \mid do(s))$ is called the causal effect of S on R, since it describes how the distribution of R varies when S is changed by external control.[17] Naturally, we need to allow for instances in which some queries are identifiable while the system as a whole is not. Hence, we say that $P(r \mid do(s))$ is identifiable in model M if every choice of the model's parameters (i.e., functional forms and distributions) compatible with the observed distribution P yields the same value for $P(r \mid do(s))$.

Remarkably, many aspects of nonparametric identification, including tests for deciding whether a given interventional query is identi-

fiable, as well as formulas for estimating such queries, can be determined graphically, almost by inspection, from the diagrams that accompany the equations.

3.4. IDENTIFICATION OF CAUSAL EFFECTS

Definition 4: (causal effect) Given a causal model M (as in equation (1)) and two disjoint sets of variables, X and Y, the causal effect of the set X on the set Y, denoted $P_M(y \mid do(x))$, is the probability of $Y = y$ induced by deleting from the model all equations corresponding to variables in X and substituting $X = x$ in the remaining equations.[18]

Clearly, the graph corresponding to the reduced set of equations is an edge subgraph of G from which all arrows to X have been pruned (Spirtes et al. 1993).

Readers accustomed to thinking of causal effects in terms of randomized experiments may interpret $P(y \mid do(x))$ as the conditional probability $P_{\text{exp}}(Y = y \mid X = x)$ corresponding to a controlled experiment in which X is randomized. An equivalent interpretation can be formulated using the potential response notation (Rubin 1974) to read

$$P(y \mid do(x)) = P(Y_x = y),$$

where Y_x is the value that Y would obtain under the hypothetical control (or treatment) $do(X = x)$. Rubin's definition of causal effect, $E(Y_{x'}) - E(Y_{x''})$, where x' and x'' are two levels of a treatment variable X, corresponds to the difference $E(y \mid do(x')) - E(Y \mid do(x''))$ and can always be obtained from the generic distribution $P(y \mid do(x))$. Definition 4 forms the bridge between SEMs and the potential response framework. It provides a precise model-theoretic definition for the counterfactual variable Y_x, which in the potential response framework is taken as a hypothetical mental construct. The SEM equivalent of Y_x is the solution for Y, after deleting from the model all equations corresponding to variables in the set X, and substituting $X = x$ in the remaining equations.

Definition 5: (causal effect identifiability) The causal effect of X on Y is said to be identifiable in a class C of models if the quantity

$P(y \mid do(x))$ can be computed uniquely from the probabilities of the observed variables V; that is, if for every pair of models M_1 and M_2 in C for which $P_{M_1}(\mathrm{v}) = P_{M_2}(\mathrm{v})$, we have $P_{M_1}(y \mid do(x)) = P_{M_2}(y \mid do(x))$.

Our analysis of identifiability will focus on a class C of models that have the following characteristics in common: (1) they share the same causal graph G and (2) they induce positive distributions on the observed variables; that is, $P_M(\mathrm{v}) > 0$.

Analysis of causal effects becomes particularly simple when dealing with Markovian models. In such models, all causal effect queries are identifiable; that is, they can be computed directly from the conditional probabilities $P(x_i \mid pa_i)$, even when the functional forms of the functions f_i and the distributions of the disturbances are not specified (Pearl 1993b; Spirtes et al. 1993). This is seen immediately from the following observations. On one hand, the distribution induced by any Markovian model M is given by the product in equation (3),

$$P_M(x_1,\ldots, x_n) = \prod_i P(x_i \mid pa_i), \tag{25}$$

where pa_i are (values of) the parents of X_i in the diagram representing M. On the other hand, the submodel $M_{x_j'}$, representing the action $do(X_j=x_j')$, is also Markovian; hence, it also induces a product-like distribution

$$P_{M_{x_j'}}(x_1,\ldots, x_n) = \begin{cases} \prod_{ij} P(x_i \mid pa_i) = \dfrac{P(x_1,\ldots, x_n)}{P(x_j \mid pa_j)} & \text{if} \quad x_j = x_j' \\ 0 & \text{if} \quad x_j \neq x_j' \end{cases} \tag{26}$$

where the partial product reflects the removal of the equation $x_j = f_j(pa_j, \varepsilon_j)$ from the model. Thus, we see that both the preaction and the postaction distributions depend only on observed conditional probabilities, but they are independent of the particular functional forms of $\{f_i\}$ and of the error distributions that generated those probabilities.

It is possible to show that certain, although not all, causal effects are identifiable in semi-Markovian nonparametric models (Pearl

1995). An important result in this direction has been the extension of the backdoor criterion (Theorem 7) to nonparametric models.

Theorem 8: (nonparametric backdoor criterion) For any disjoint sets of variables X and Y in a causal diagram G, if the two conditions of Theorem 7 are satisfied, then the causal effect of X on Y is identified and is given by

$$P(y \mid do(x)) = \sum_z P(y \mid x,z)P(z). \tag{27}$$

This theorem provides a formal definition for the concepts of exogeneity and confounding in econometrics (Engle, Hendry, and Richard 1983) and epidemiology (Greenland, Pearl, and Robins 1998), respectively. A variable X is said to be exogenous (unconfounded) relative to Y if $P(y \mid do(x)) = P(y \mid x)$; that is, if the conditions of the backdoor criterion hold when Z is the empty set. Alternatively, X is said to be conditionally exogenous (unconfounded) relative to Y given measurements on set Z if equation (27) holds; that is, if the conditions of the backdoor criterion hold for Z. Section 3.1 proposes an explanation why the definitions of these two basic concepts have encountered difficulties in econometrics and epidemiology (see also Pearl 1998).

Pearl (1995) introduces a symbolic calculus for the $do(\cdot)$ operator, which facilitates the identification of additional causal effects in nonparametric models. Using this calculus, Galles and Pearl (1995) have devised graphical criteria for identifying causal effects in any semi-Markovian model. Finally, if the objective of a study is to evaluate the direct, rather than the total, causal effect of X on Y, as was the case with the Berkeley graduate admissions study (see note 5), then some other graphical criteria that determine identifiability are available (Pearl and Robins 1995).

In light of these results, the reader might want to know whether the model defined by equations (6) through (8) is identifiable. The answer is yes; this model permits the identification of all interventional queries. For example, from inspection of the graph in Figure 13, we can conclude immediately that

1. $P(x \mid do(y), do(z)) = P(x)$, consistent with the intuition that consequences can have no effect on their causes;
2. $P(z \mid do(x)) = P(z \mid x)$, because ε_2 is independent of X, and hence Z is not confounded by X (alternatively, and hence all backdoor paths between Z and X are blocked);
3. $P(y \mid do(z)) = \sum_x P(y \mid z,x)P(x)$, because the backdoor criterion qualifies X as an appropriate covariate for adjustment;
4. $P(y \mid do(x)) = \sum_z P(z \mid x) \sum_{x'} P(y \mid x',z)P(x')$, which results from chaining $P(z \mid do(x))$ with $P(y \mid do(z))$, as is shown formally in Pearl (1995).

4. SOME CONCEPTUAL UNDERPINNINGS

4.1. WHAT DO STRUCTURAL PARAMETERS REALLY MEAN?

Every student of SEM has stumbled on the following paradox at some point in his or her career. If we interpret the coefficient β in the equation

$$y = \beta x + \varepsilon$$

as the change in $E(Y)$ per unit change of X, then after rewriting the equation as

$$x = (y - \varepsilon)/\beta,$$

we ought to interpret $1/\beta$ as the change in $E(X)$ per unit change of Y. But this conflicts both with intuition and with the prediction of the model: The change in $E(X)$ per unit change of Y ought to be zero if Y does not appear as an independent variable in the equation for X.

Teachers of SEM generally evade this dilemma via one of two escape routes. One route involves denying that β has any causal reading and settling for a purely statistical interpretation in which β measures the reduction in the variance of Y explained by X (e.g., Muthen 1987). The other route permits causal reading of only those coefficients that meet the so-called isolation restriction (Bollen 1989;

James et al. 1982); namely, the explanatory variable must be uncorrelated with the error in the equation. Because ε cannot be uncorrelated with both X and Y, so the argument goes, β and $1/\beta$ cannot both have causal meaning, and the paradox dissolves.

The first route is self-consistent, but it compromises the founders' intent that SEM function as an aid to policy making and clashes with the intuition of most SEM users. The second is vulnerable to attack logically. It is well known that every pair of bivariate normal variables, X and Y, can be expressed in two equivalent ways:

$$y = \beta x + \varepsilon_1$$

and

$$x = \alpha y + \varepsilon_2,$$

where $\text{cov}(X, \varepsilon_1) = \text{cov}(Y, \varepsilon_2) = 0$, and $\alpha = r_{XY} = \beta\sigma_X^2/\sigma_Y^2$. Thus, if the condition $\text{cov}(X, \varepsilon_1) = 0$ endows β with causal meaning, then $\text{cov}(Y, \varepsilon_2) = 0$ ought to endow α with causal meaning as well. But this, too, conflicts with both intuition and the intentions behind SEM; the change in $E(X)$ per unit change of Y ought to be zero, not r_{XY}, if there is no arrow from Y to X.

What then is the meaning of a structural coefficient? Or a structural equation? Or an error term? The interventional interpretation of causal effects, when coupled with the $do(x)$ notation introduced in Section 3.3, provides simple answers to these questions. The answers explicate the operational meaning of structural equations and, it is hoped, end an era of controversy and confusion regarding these entities.

4.1.1. Structural Equations: Operational Definition

Definition 6: (structural equations) An equation $y = \beta x + \varepsilon$ is said to be structural if it is to be interpreted as follows. In an ideal experiment where we control X to x and any other set Z of variables (not containing X or Y) to z, the value y of Y would be independent of z and is given by $\beta x + \varepsilon$.

This definition is operational because all quantities are observable, albeit under conditions of controlled manipulation. That manipulations cannot be performed in most observational studies does not

negate the operationality of the definition, in much the same way that our inability to observe bacteria with the naked eye does not negate their observability under a microscope. The challenge of SEM is to extract the maximum information on what we wish to observe from the little we can observe.

Note that the operational reading given above makes no claim about how X (or any other variable) will behave when we control Y. This asymmetry makes the equality signs in structural equations different from algebraic equality signs; the former act symmetrically in relating observations on X and Y (e.g., observing $Y = 0$ implies $\beta x = -\varepsilon$), but they act asymmetrically when it comes to interventions (e.g., setting Y to zero tells us nothing about the relation between x and ε). The arrows in path diagrams make this dual role explicit, and this may account for the insight and inferential power gained through the use of diagrams.

The strongest empirical claim of the equation $y = \beta x + \varepsilon$ is made by excluding other variables from the right-hand side of the equation, thus proclaiming X the *only* immediate cause of Y. This translates into a testable claim of invariance: The statistics of Y under condition $do(x)$ should remain invariant to the manipulation of any other variable in the model (Pearl 1996; Galles and Pearl 1998).[19] This can be written symbolically as

$$P(y \mid do(x), do(z)) = P(y \mid do(x)) \tag{28}$$

for all Z disjoint of $\{X \cup Y\}$.

Note that this invariance holds relative to manipulations, not observations, of Z. The statistics of Y under condition $do(x)$, given the measurement $Z = z$, written $P(y \mid do(x), z)$, would certainly depend on z if the measurement was taken on a consequence (i.e., descendant) of Y. Note also that the ordinary conditional probability $P(y \mid x)$ does not enjoy such a strong property of invariance, since $P(y \mid x)$ is generally sensitive to manipulations of variables other than X in the model, unless X and ε are independent. Equation (28), in contrast, remains valid regardless of the statistical relationship between ε and X.

Generalized to a set of several structural equations, equation (28) explicates the assumptions underlying a given causal diagram. If G is the graph associated with a set of structural equations, then the

assumptions are embodied in G as follows: (1) every missing arrow, say between X and Y, represents the assumption that X has no causal effect on Y once we intervene and hold the parents of Y fixed; and (2) every missing bidirected link between X and Y represents the assumption that the omitted factors that influence Y are uncorrelated with those that influence X. Sections 4.1.3 and 4.1.4 discuss the operational meaning of the latter assumption.

4.1.2. The Structural Parameters: Operational Definition

The interpretation of a structural equation as a statement about the behavior of Y under a hypothetical intervention yields a simple definition for the structural parameters. The meaning of β in the equation $y = \beta x + \varepsilon$ is simply

$$\beta = \frac{\partial}{\partial x} E[Y \mid do(x)], \tag{29}$$

namely, the rate of change (relative to x) of the expectation of Y in an experiment where X is held at x by external control. This interpretation holds regardless of whether ε and X are correlated in nonexperimental studies (e.g., via another equation $x = \alpha y + \delta$).

4.1.3. The Mystical Error Term: Operational Definition

The interpretations given in Sections 4.1.1 and 4.1.2 provide an operational definition for that mystical error term

$$\varepsilon = y - E[Y \mid do(x)], \tag{30}$$

which, despite being unobserved in nonmanipulative studies, is far from being metaphysical or definitional as suggested by some researchers (e.g., Richard 1980; Holland 1988:460; Hendry 1995:62). Unlike errors in regression equations, ε measures the deviation of Y from its controlled expectation $E[Y \mid do(x)]$ and not from its conditional expectation $E[Y \mid x]$. The statistics of ε can therefore be measured from observations on Y once X is controlled. Alternatively, since β remains the same, regardless of whether X is manipulated or ob-

served, the statistics of $\varepsilon = y - \beta x$ can be measured in observational studies, if we know β.

Likewise, correlations among errors can be estimated empirically. For any two nonadjacent variables in the model, X and Y, equation (30) yields

$$E[\varepsilon_Y \varepsilon_X] = E[YX \mid do(pa_Y, pa_X)] - E[Y \mid do(pa_Y)]E[X \mid do(pa_X)]. \quad (31)$$

Once we determine the structural coefficients, the controlled expectations $E[Y \mid do(pa_Y)]$, $E[X \mid do(pa_X)]$, and $E[YX \mid do(pa_Y, pa_X)]$ become known linear functions of the observed variables pa_Y and pa_X; hence, the expectations on the right-hand side of equation (31) can be estimated in observational studies. Alternatively, if the coefficients are not determined, the expression can be assessed directly in interventional studies by holding pa_X and pa_Y fixed (assuming X and Y are not in parent-child relationship) and estimating the covariance of X and Y from data obtained under such conditions.

Finally, we are often interested not in assessing the numerical value of $E[\varepsilon_Y, \varepsilon_X]$ but rather in determining whether ε_Y and ε_X can be assumed uncorrelated. For this determination, it suffices to test whether the equality

$$E[Y \mid x, do(s_{XY})] = E[Y \mid do(x), do(s_{XY})] \quad (32)$$

holds true, where s_{XY} stands for (any setting of) all variables in the model excluding X and Y. This test can be applied to any two variables in the model, except when Y is a parent of X, in which case the symmetrical equation is applicable, with X and Y interchanged.

4.1.4. The Mystical Error Term: Conceptual Interpretation

The authors of SEM textbooks usually interpret error terms as representing the influence of omitted factors. Many SEM researchers are reluctant to accept this interpretation, however, partly because unspecified omitted factors open the door to metaphysical speculations, and partly because arguments based on such factors were improperly used as a generic, substance-free license to omit bidirected arcs from path diagrams (McDonald 1997). Such concerns are an-

swered by the operational interpretation of error terms, equation (30), since it prescribes how errors are measured, not how they originate.

It is important to note, however, that the operational definition is no substitute for the omitted-factors conception when it comes to deciding whether pairs of error terms can be assumed to be uncorrelated. Because such decisions are needed at a stage when the model's parameters are still "free," they cannot be made on the basis of numerical assessments of correlations but must rest instead on qualitative structural knowledge about how mechanisms are tied together and how variables affect each other. Such judgmental decisions are hardly aided by the operational criterion of equation (31), since that criterion instructs the investigator to assess whether two deviations, taken on two different variables under complex experimental conditions, would be correlated or uncorrelated. Such assessments are cognitively unfeasible.

In contrast, the omitted-factors conception instructs the investigator to judge whether there could be factors that simultaneously influence several observed variables. Such judgments are cognitively manageable, since they are qualitative and rest on purely structural knowledge—the only knowledge available during this phase of modeling.

Another source of error correlation is selection bias. If two uncorrelated unobserved factors have a common effect that is omitted from the analysis but influences the selection of samples for the study, then the corresponding error terms will be correlated in the sampled population; that is, the expectation in equation (31) will not vanish when taken over the sampled population (see discussion of Berkson's paradox following Definition 1).

We should emphasize, however, that the arcs *missing* from the diagram, not those *in* the diagram, demand the most attention and careful substantive justification. Adding an extra bidirected arc can only compromise the identifiability of parameters, but deleting an existing bidirected arc may produce erroneous conclusions and a false sense of model testability. Thus, bidirected arcs should be assumed to exist, by default, between any two nodes in the diagram. They should be deleted only by well-motivated justifications, such as the unlikely existence of a common cause for the two variables and the unlikely existence of selection bias. Although we can never be cognizant of all

the factors that may affect our variables, substantive knowledge sometimes permits us to state that if a common factor exists, its influence is not likely to be significant.

Thus, as often happens in the sciences, the way we measure physical entities does not offer the best way of thinking about them. The omitted-factors conception of errors, because it rests on structural knowledge, is a more useful guide than the operational definition when building, evaluating, and thinking about causal models.

4.2. INTERVENTIONAL INTERPRETATION OF EFFECT DECOMPOSITION

In this section, we show that the interventional interpretation of structural equations provides simple, unambiguous definitions of effect decomposition for both parametric and nonparametric models.

We start with the general notion of a causal effect $P(y \mid do(x))$ from Definition 4, which applies to arbitrary sets of variables X and Y. This interpretation of causal effect can be specialized to define total and direct causal effects, as follows.

Definition 7: (total effect) The total effect of X on Y is given by $P(y \mid do(x))$; namely, the distribution of Y while X is held constant at x and all other variables are permitted to run their natural course.

Definition 8: (direct effect) The direct effect of X on Y is given by $P(y \mid do(x), do(s_{XY}))$, where S_{XY} is the set of all observed variables except X and Y in the system.

In linear analysis, Definitions 7 and 8 yield, after differentiation with respect to x, the path coefficients that are normally associated with direct and indirect effects, yet they differ from conventional definitions in several important aspects. First, direct effects are defined in terms of hypothetical experiments in which intermediate variables are held constant by physical intervention, not by statistical adjustment (which is often disguised under the misleading phrase "control for"). Figure 11 depicts a simple example in which adjusting for the intermediate variables (Z and W) would not give the correct value (= 0) for the direct effect of X on Y, whereas $\partial/\partial x E(Y \mid do(x, z,$

w)) does yield the correct value: $\partial/\partial x(\beta w + \gamma z) = 0$. Second, there is no need to limit control to only intermediate variables; all variables in the system may be held constant (excluding X and Y). Thus, the measurement of direct effects is ascribed to an ideal laboratory; the scientist controls for all possible conditions S_{XY}, and measurements may commence without knowing the structure of the diagram. Finally, our definitions differ from convention by interpreting total and direct effects independently of each other, as outcomes of two different experiments. Textbook definitions (e.g., Bollen 1989:376) usually equate the total effect with a power series of path coefficient matrices. This algebraic definition coincides with the operational definition (Definition 7) in recursive (semi-Markovian) systems but yields erroneous expressions in models with feedback. For instance, given the pair of equations $\{y = \beta x + \varepsilon, x = \alpha y + \delta\}$, the total effect of X on Y is simply β, not $\beta(1 - \alpha\beta)^{-1}$ as stated in Bollen (1989:379). The latter has no operational significance worthy of the phrase "effect of X."[20]

Note that when the structure of the causal diagram is known, there is no need to actually hold all other variables constant; holding constant the direct parents of Y (excluding X) would have the same effect on Y as holding all variables except X and Y constant. Thus, we obtain the following equivalent definition of a direct effect.

Corollary 2: The direct effect of X on Y is given by $P(y \mid do(x), do(pa_{Y\setminus X}))$, where $pa_{Y\setminus X}$ stands for any realization of the variables appearing in the equation for Y, excluding X.

Readers versed in linear analysis may find it a bit strange that the direct effect of X on Y involves variables other than X and Y. However, given that we are dealing with nonlinear interactions, the effect of X on Y should indeed depend on the levels at which we hold the other parents of Y. If we wish to average over these values and take the expectation of Y, we obtain the expression

$$\Delta_{x,x'}(Y) = \sum_{pa_{Y/X}} [E(Y \mid do(x'), do(pa_{Y\setminus X})) - E(Y(do(x), do(pa_{Y\setminus X}))]P(pa_{Y\setminus X} \mid do(x)),$$

where $\Delta_{x,x'}(Y)$ stands for the average change in $E(Y)$ induced by changing X from x to x' while keeping the other parents of Y constant

at whatever value they obtain under $do(x)$. This expression explicates what we actually wish to measure in race or sex discrimination cases, where we are instructed to assess the effect of one factor (X) while keeping "all other factors constant." If X is an exogenous variable, as "gender" is in Berkeley's sex discrimination case (see note 5), then $do(x)$ can be replaced with x. However, we are not at liberty to replace $do(pa_{YX})$ with pa_{YX} unless we can safely assume that the factors represented by pa_{YX} (e.g., student's qualifications and the choice of department) are not confounded with Y (as defined in Theorem 8). In general, we see that the average direct effect $\Delta_{x,x'}(Y)$ is identifiable whenever both $E[Y \mid do(x), do(pa_{YX})]$ and $P(pa_{YX} \mid do(x))$ are identifiable. Note that if X does not appear in the equation for Y, then $P(y \mid do(x), do(pa_{YX}))$ defines a constant distribution on Y, independent of x, which matches our understanding of "having no direct effect." Note as well that, in linear models, $\Delta_{x,x'}(Y)$ reduces to $x' - x$ times the path coefficient between X and Y.

In standard linear analysis, an indirect effect may be defined as the difference between the total and the direct effects (Bollen 1989). In nonlinear analysis, differences lose their significance, and one must isolate the contribution of mediating paths in some other way. Expressions of the form $P(y \mid do(x), do(z))$ cannot be used to isolate such contributions, however, because there is no physical means of selectively disabling a direct causal link from X to Y by holding some variables constant. This suggests that the notion of indirect effect has no intrinsic operational meaning apart from providing a comparison between the direct and the total effects. In other words, a policy maker who asks for that part of the total effect transmitted by a particular intermediate variable or by a group Z of such variables is really asking for a comparison of the effects of two policies, one where Z is held constant, the other where it is not. The expressions corresponding to these policies are $P(y \mid do(x), do(z))$ and $P(y \mid do(x))$, and this pair of distributions should be taken as the most general representation of indirect effects. Similar conclusions have been expressed by Robins (1986) and Robins and Greenland (1992).

5. CONCLUSION

Currently, the enterprise known as structural equation modeling is increasingly under fire. The founding fathers have retired, their teachings are forgotten, and practitioners, teachers, and researchers currently find the methodology they inherited difficult to either defend or supplant. Modern SEM textbooks are preoccupied with parameter estimation and rarely explicate the role that those parameters play in causal explanations or in policy analysis; examples dealing with the effects of interventions are conspicuously absent, for instance. Research in SEM now focuses almost exclusively on model fitting, while foundational issues pertaining to the meaning of SEM's models are subjects of confusion and controversy.

The contemporary crisis in SEM originates, I am thoroughly convinced, in the lack of a mathematical language that can handle the causal information embedded in structural equations. Recently, graphical models have provided such a language. They have thus helped us answer many of the unsettled foundational questions that drive the current crisis, including:

- Under what conditions can we give causal interpretation to structural coefficients?
- What are the causal assumptions underlying a given SEM?
- What are the statistical implications of any given SEM?
- What is the operational meaning of a given structural coefficient?
- What are the policy-making claims of any given SEM?
- When is an equation not structural?

In this article, I have summarized the conceptual developments that now resolve such foundational questions. In addition, by way of illustrating the soundness of the approach, I have presented several tools to be used in answering questions of practical importance. Questions of this type include:

- When are two SEMs observationally indistinguishable?
- When do regression coefficients represent path coefficients?
- When would the addition of a regressor introduce bias?
- How can we tell, prior to taking any data, which path coefficients can be identified?

- When can we dispose of the linearity-normality assumption and still extract causal information from the data?

I am hopeful that researchers will recognize the benefits of these concepts and tools and use them to revitalize causal analysis in the social and behavioral sciences.

NOTES

1. A summary of attempts by philosophers to reduce causality to probabilities is given in Pearl (1996:396-405).

2. The equivalence of the potential response and structural equation frameworks, anticipated by Holland (1986), Pratt and Schlaifer (1988), Pearl (1995), and Robins (1995), is proven formally in Galles and Pearl (1998).

3. These assumptions are explicated and operationalized in Section 4. Briefly, if G is the graph associated with a causal model that renders a certain parameter identifiable, then two assumptions are sufficient for authenticating the causal reading of that parameter; namely, (1) every missing arrow, say between X and Y, represents the assumption that X has no effect on Y once we intervene and hold certain other variables fixed, and (2) every missing bidirected arc $X \leftarrow - - \rightarrow Y$ represents the assumption that all omitted factors that affect Y are uncorrelated with those that affect X. Each of these assumptions is testable in experimental settings, where interventions are feasible (Section 4.1.3).

4. Holland's interpretation is at variance with the structural reading of the equation (Haavelmo 1943): "In an ideal experiment where we control X to x and any other set Z of variables (not containing X or Y) to z, Y is independent of z and is given by $a + bx + \varepsilon$" (see section 4.1.1).

5. The classic case demonstrating Simpson's reversal is the study of Berkeley's alleged sex bias in graduate admission (Bickel, Hammel, and O'Connell 1975), where data showed a higher rate of admission for male applicants overall but, when broken down by departments, yielded a slight bias toward female applicants.

6. This advice, which rests on the causal relationship "not affected by," is, to the best of my knowledge, the only causal notion that has found a place in statistics textbooks. The advice is necessary, but it is not sufficient. The other common guideline, that X should not precede Z (Shafer 1996:326), is neither necessary nor sufficient, as will become clear in Section 3.

7. Causal models, structural equations, and error terms will be defined in terms of response to interventions in Section 4. Formal treatments of these notions are given in Galles and Pearl (1997, 1998).

8. The term *recursive* is ambiguous; some authors exclude correlated errors, but others do not.

9. The possibility that linear models may possess more economical bases came to my awareness during a conversation with Rod McDonald.

10. Verma and Pearl (1990b) present an example using a nonparametric model, and Richardson has devised an example using linear models with correlated errors (Spirtes and Richardson 1996).

11. This example was brought to my attention by Jin Tian, and a similar one by two anonymous reviewers.

12. In response to an allegation that "path analysis does not derive the causal theory from the data, or test any major part of it against the data" (Freedman 1987:112), Bollen (1989:78) states, "We can test and reject structural models. . . . Thus the assertion that these models cannot be falsified has little basis."

13. This can be seen when the relation between Y and its parents, $Y = \alpha x + \sum_i \beta_i w_i + \varepsilon$ is substituted into the expression for $r_{YX \cdot Z}$, which yields α plus an expression $I_{YX \cdot Z}$ involving partial correlations among the variables $\{X, W_1, \ldots, W_k, Z, \varepsilon\}$. Because Y is assumed not to be an ancestor of any of these variables, their joint density is unaffected by the equation for Y; hence, $I_{YX \cdot Z}$ is independent of α.

14. This result is also presented in Spirtes et al. (1998).

15. An equivalent version of this model is obtained by eliminating U from the equations and allowing ε_1 and ε_3 to be correlated.

16. Pearl (1993b, 1995) used the notation $set(X = x)$. Currently, however, the $do(X = x)$ notation (taken from Goldszmidt and Pearl 1992) seems to be winning broader popular support.

17. Technically, the adjective *causal* is redundant. It serves to emphasize, however, that the changes in S are enforced by external control and do not represent stochastic variations in the observed value of S.

18. Explicit translation of interventions to "wiping out" equations from the model was first proposed by Strotz and Wold (1960) and has since been used by Fisher (1970) and Sobel (1990). Graphical ramifications of this translation were explicated by Spirtes, Glymour, and Scheines (1993) and Pearl (1993b) A related mathematical model using event trees has been introduced by Robins (1986:1422-25).

19. This statistical claim is, in fact, only part of the message conveyed by the equation; the other part consists of a counterfactual claim (Lewis 1973; Rubin 1990; Galles and Pearl 1998): If we were to control X to x' instead of x, then Y would attain the value $\beta x' + \varepsilon$. In other words, plotting the value of Y under various hypothetical controls of X, and under the same external conditions (ε), should result in a straight line with slope β. Such claims can be tested only under the assumption that ε, representing external conditions or properties of experimental units, remains unaltered as we switch from x to x' (Pearl 1996:404).

20. This error was noted by Sobel (1990), but, perhaps because constancy of path coefficients was presented as new and extraneous assumptions, Sobel's correction has not brought about a shift in practice or philosophy.

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