The role that causality can play in social network analysis is unclear. The author provides a broad characterization of social network analysis before considering the nature of causality. He distinguishes four types of causality: system causality, statistical causality, mechanism causality, and algorithmic causality. Their potential places in network analysis are discussed. Understanding generative mechanisms—be they system, mechanism, or algorithmic—seems the most promising way to proceed. The role of statistical causality is a source of potential data analytic tools that can be mobilized within analyses conducted in the spirit of the other three types of causality.

Causality in Social Network Analysis

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The title *Causality in Social Network Analysis* is both precise and ambiguous. The ambiguity stems from the terms *causality* and *social network analysis*. I start with causality as a primitive term and then outline four avenues by which causality can be approached: system causality, statistical causality, (social) mechanism causality, and algorithmic causality. The potential roles each can play in social network analysis are then discussed.

1. SOCIAL NETWORK ANALYSIS

1.1. SOCIAL NETWORKS

The most straightforward definition of a social network is $G = (V, R)$, where V is a set of social actors and R is a social relation defined over the elements of V. Each element of R is a pair of elements from V with $R \subseteq V \times V$, the Cartesian product of V with itself. Put differently,

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for $i, j \varepsilon V$, iRj means $(i, j) \varepsilon R$. The relational ties can be binary (0, 1) or have magnitude.

Instead of labeling a relation as R, we can list the elements of R and call them *lines*. The network can be represented as $N = (V, L)$, where L is the set of lines in the relation R. For a symmetric relation, $iRj \Leftrightarrow jRi$. Such lines are undirected and are termed *edges*. For directed ties, the lines are termed *arcs*. Networks having both arcs and edges can be written $N = (V, A, E)$, with A the set of arcs and E the set of edges. A less simple definition of a network is $N = (V, R_1, R_2, \ldots R_K)$, where the social actors have multiple (K) social relations defined over them.

Another type of network structure involving two kinds of social actors is a membership or affiliation network. Letting G and H be the two sets of actors and λ the membership/affiliation relation, if $g \in G$ and $h \in H$, then $g \lambda h$ means that $(g, h) \in \lambda$. The data structure for this type of relation (defined in terms of two sets of social actors) has been termed "two mode," in contrast to "one mode," having only one kind of social actor. In principle, there can be a specific data set. We could consider three kinds of social actors (see Fararo and Doreian 1984). Batchelder, Kumbasar, and Boyd (1997) provided an alternate treatment of three-mode data. In a specific analysis of a network, we can include multiple types of actors and multiple-mode relations. All that is required for this discussion is that there can be multiple relations for multiple types of actors in the definition of a social network.

Networks may be disjoint. They may overlap to differing extents, and they may be nested within hierarchies of networks. In principle, any structure that can be expressed in terms of social actors and social relations can be represented in social network terms. This means that empirical social networks can range from the very simple (e.g., an isolated dyad) to the extremely complex (e.g., a nation-state). It may well be the case that representing these social structures faithfully is extremely difficult.

1.2. THE SOCIAL NETWORK ANALYTIC PERSPECTIVE

Social network analysts are committed, in a fundamental way, to incorporating social network characteristics in their work. This leads them to define their central concerns as the creation, maintenance, transformation, and dissolution of social structures. (For Fararo

1989:62, these are the main problems of general theoretical sociology.) As Doreian and Stokman (1997) claimed, "network *processes* are *series of events* that create, sustain and dissolve social structures" (p. 3, italics added).

This commitment is paradigmatic. For network analysts, there is a sharp distinction between information about the social actors and information concerning the social structures within which these actors are located. Without the latter, there are no network analyses. Wellman (1988) distilled the central features of this paradigm, and the following narrative focuses exclusively on his characterization.

First, "behavior is *interpreted* in terms of structural constraints on activity *rather than* in terms of inner forces within [actors]" (Wellman 1988:20, italics added). For some analysts (me included), the "rather than" can be replaced by "in addition to." Second, "analyses focus on the relations between [social actors]" (p. 20). The third feature is "A central consideration is how the patterned relationships among multiple actors *jointly affect* [italics added] network members' behavior" (p. 20). Here, the language has shaded into terms incorporating causality in some sense. The fourth feature is expressed as follows: "Structure is treated as a network of networks that may or may not be partitioned into discrete groups" (p. 20). Finally, "analytic methods deal directly with the patterned, relational nature of social structure" (p. 20).

Items 2, 4, and 5 are simply descriptions of what network analysts do. In contrast, Items 1 and 3 are different: A primacy is placed on the benefits of working within the network analytic paradigm. The language Wellman (1988) used became more forceful when he outlined the structural alternative.

Again, Wellman (1988) used five claims. In restating these claims, emphasis has been placed on those words that seem relevant for discussing causality. "Structured social relationships are a *more powerful* source of sociological *explanation* than person attributes of system members" (p. 31, italics added). Whatever sociological explanation is, it fares better, in Wellman's view, when the (relevant) structural information is incorporated into the explanation.

"Norms *emerge* [italics added] from location in structured systems of social relationships" (Wellman 1988:33). This, from a nonnetwork perspective, is radical. There is much more here than an emphasis on

structural characteristics. Often, in conventional actor-attribute–oriented accounts, people's behaviors are explained by their adherence to norms (or values or other cultural prescriptions and proscriptions). The structuralist argument claims that even if such accounts have merit, the norms and so forth used in these explanations are created structurally. The ultimate explanation, it seems, is found in social structures.

"Social structures *determine* [italics added] the operation of dyadic relationships" (Wellman 1988:35). The simplest network structure is a dyad. At face value, a dyad involves two actors, their attributes, and the relation(s) between them. It is possible that actor attributes have some impact on the formation, maintenance, and dissolution of their social ties. In a completely isolated dyad, this may be the case. However, Wellman (1988) argued that dyads are embedded in larger structures and these structures determine the operation and structure of dyadic ties.

"The world is composed of networks, not groups" (Wellman 1988:37). We can make this (trivially) true by use of the affiliation type of relational tie. However, as I read it, this was not Wellman's (1988) intent. If social actors are located in multiple networks, it is this set of structural locations that comes first in trying to understand behavior. Viewing the world as networks of networks—and not social aggregates as social classes and age groups—is more fruitful than struggling with such ill-defined aggregates in seeking to account for behavior.

"Structural methods supplement and *supplant* [italics added] individualistic methods" (Wellman 1988:38). There is no ambiguity here. When network analysts express preferences about the units of analysis, about using social relational information and constructing structural accounts, it is possible to think of structuralist methods supplementing whatever is used in sociological explanation. But the claim to "supplant" other approaches is unequivocal and seems to suggest that social scientists should use network analysis.

Consistent with this is Berkowitz's (1982) claim that "structural analysis does not represent a simple extension of existing social science paradigms, but, signals instead the beginning of a scientific revolution" (p. 158). The "ought" claim is now explicit. Rogers (1987) drew attention to "how radically different network analysis is from

conventional social science" (p. 288). Doreian (1995) suggested that it was far too early to declare the arrival of a (structural) scientific revolution. Thinking about causality reinforces that argument.

1.3. ANALYSIS OF NETWORK AND OTHER TYPES OF DATA

Before discussing issues of causality, some attention to data and data analyses is useful. Conventional data sets are viewed in terms of units of analysis and variables. They reflect a variable-centered approach as described by Abell (1987). Such attribute data can be analyzed with a wide variety of statistical procedures including regression, structural equation modeling, and log-linear models. This can be characterized as a statistics-only approach.

Based on the prospect of revolutionizing social science via networks—or just from the need to analyze network structures—many special social network tools have been created to analyze network data. Wasserman and Faust (1994) provided a compendium of such procedures together with an informed interpretive narrative. A strict network orthodoxy would insist that only data and analyses of this sort are pertinent. This is a networks-only approach. Of course, statistical tools have been used to analyze network data for network concerns—see, for example, Wasserman and Pattison (1996)—and some of the network tools have been used on conventional attribute data. Even if this distinction between statistical and network analyses is too simplistic, it is useful here.

It seems most flexible and fruitful to work with both network and attribute data. Such uses can range from simply interpreting the outcomes of one in terms of the other to the joint integrated use of tools to analyze both types of data. Wasserman and Pattison's (1996) adaptation of logistic regression for use with network data, models of network autocorrelation (Doreian 1989), and Snijders's (1997) innovative development of a class of statistical models for examining network change provide examples. Together, they are parts of a joint statistics and networks approach.

But useful for what? This signals Causality that it is time to come to center stage. However, data analysis will not slink off the stage because all discussions of causality—as an empirical matter—turn on what we do with regard to data, our assumptions about the tools we

use, the nature of the world, and our assumptions about what we think we know.

2. CAUSALITY

As libraries can be filled with the articles and books devoted to this topic, the consideration here is woefully incomplete. The approach taken here starts within an engineering perspective, one that provides a conceptually straightforward approach to characterizing causality by means of the idea of a system. I call this system causality. The System Causality section is followed by a section on statistical causality that looks at the use of statistics to discern causality. Both of these sections are formulated within the variable-centered approach. Our discussion will then shift to consider social mechanism causality and event sequences before a concluding subsection of algorithmic causality. The roles these types of causality play in network analysis is considered in Section 3.

2.1. SYSTEM CAUSALITY

My point of departure is alien from the structuralist perspective as described above. A (social) system can be described by a set of variables and one or more (social) processes. In a crude fashion, the operation of such a system transforms inputs at one point in time into outputs at a subsequent point in time. This is rather glib, as instantaneous change is possible and to make such statements requires knowledge of the time scales of the processes modeled.

> The *state* (of the system) is some compact representation of the past activity of the system complete enough to allow us to predict, on the basis of the inputs, exactly what the outputs will be, and also update the state itself. (Padulo and Arbib 1974:21)

Let x represent a set of, say, m variables that characterize a system, and let $\mathrm{x}(t)$ represent the state of the system at time t.

Suppose the system exists at time t_0 and receives inputs, represented by z. If the state of the system at t_0 is $\mathrm{x}(t_0)$, then the new state of the sys-

tem is given by $x(t_1) = \phi\ (t_0, t_1, x(t_0), z)$ for some well-defined function ϕ.

> This function ϕ is called the *state transition map* and it tells us that if we specify two times t_1 and t_0, a state $\tilde{x}$, and an admissible input function z, then, if we start the system in state $\tilde{x}$ at time t_0 and apply the input function z, the system will end up at state $\phi\ (t_0, t_1, \tilde{x}, z)$ at time t_1. (Padulo and Arbib 1974:26)

The output of the operation of the system is given by some function $y(t_1) = \eta\ (t_1, t_0, x(t_1), z)$. This characterization is for a deterministic system. If, no matter how carefully the state of the system and the inputs are specified, the subsequent state of the system and the output are not predicted exactly, then the system is said to be stochastic.

If we focus on $x(t)$, putting $y(t)$ to one side, we can examine how these variables might change. The $x(t)$ values are not free to take on any values. The state of the system constrains the possible future states. Doreian and Hummon (1976) wrote of structural control models where the core statement of change is:

$$\Delta x(t) = \gamma(x^*(t) - x(t))\Delta t, \tag{1}$$

where $\Delta x(t)$ is an increment of change of the x values in an increment of time Δt. In this formulation, $x^*(t)$ represents the control variables for the process and γ is a parameter that represents the responsiveness of the system to departures of $x(t)$ from the values of the control variables, $x^*(t)$. These departures are $(x^*(t) - x(t))$, and the specifications are those of an equilibration model. The variables in $x^*(t)$ are taken to be functions of some of the other variables in $x(t)$ that represent the state of the system. Dividing by Δt and taking limits as Δt tends to 0,

$$\dot{x}(t) = \gamma(x^*(t) - x(t)), \tag{2}$$

where $\dot{x}(t)$ represents the derivatives of the system variables with respect to time. If, for example, $x^*(t) = Ax + b$, where A is a matrix of parameters (with zeros on the main diagonal) and b is a vector of parameters, the equation system representing change is

$$\dot{x}(t) = \gamma(Ax(t) + b - x(t)). \tag{3}$$

Equation (3) is particularly important as it expresses the coupling of the $x_k(t)$ processes generating each of the x_k. More generally, the A and b may be time-varying parameters; even more generally, x*(t) can be a set of nonlinear functions of the x(t).

Another source of model-building complexity is the incorporation of exogenous impacts on the system (or inputs to the system). Denoting these by z(t), they can be incorporated in the control variable specification $x^*(t) = Ax + B + \Gamma z(t)$ where Γ contains further parameters.

Each of these differential equation systems can be represented simply as

$$\dot{x}(t) = f(x(t), z(t), c), \tag{4}$$

where c is a collection of parameters (or products of parameters) drawn from γ, A, b, and Γ, and f is a well-defined function. In general, such a differential equation system can be solved to yield

$$x(t) = g(x(t), z(t), c), \tag{5}$$

where g is another well-defined function. If there are no exogenous inputs, then equations (4) and (5) are written as $\dot{x}(t) = f(x(t), c)$ and $x(t) = g(x(t), c)$, respectively. Notationally and conceptually, these two equations are those used by Fararo (1989:74-5) to represent a general dynamical system.

Equations (4) and (5) (or their variants without exogenous inputs) are particularly important as they describe the generators of the processes. If the parameters, the initial conditions (at t_0) and the exogenous inputs, z(t), are all known, these equations can be used to generate the states of the system and its outputs at each point in time. Fararo (1989) used the term "recursive generation" to label such processes.

By way of a partial summary, system causality is given by the operation of the generating equations, examples of which are equations (4) and (5). With the parameters, the initial conditions, and the equations all known, the trajectories of the system are generated. In this sense, the idea of variables causing variables has a clear and simple meaning.

Approaching causality in this fashion permits important questions. First, our attention is directed to the idea of initial conditions, the $x(t_0)$. Knowledge of these conditions is needed to understand a trajectory

(and potential trajectories). The basic (ordinary or partial) differential equations can be viewed as providing a set of transition rules that govern the operation of the system, and knowledge of these rules also is needed to understand the behavior generated by the system. With the processes represented in this fashion, equilibria (if they exist) are characterized by $\dot{x}(t) = 0$. This implies $f(x(t), c) = 0$ which can be used to locate the equilibrium state(s). If there are equilibria, the formal machinery permits the determination of whether the equilibria are stable or unstable.

For these systems, there is a set of possible parameter values and a set of possible system states. Together, these generate all conceptually possible outcomes as results of the operation of system causality. Actually using this idea of system causality implies two types of tasks. One is theoretical, where the task is to explore the implications of different sets of parameter values. If the dynamic behavior of such systems is different for different sets of parameter values, understanding how differences in parameters produce different behaviors and system states is important theoretical knowledge. If the trajectories of the system depend on the initial conditions, knowing the nature of this dependency is also important theoretical knowledge.

The second type of task is empirical. Given an empirical system, the task is to locate appropriate process models and estimate the values of the parameters of such models. (See Tuma and Hannan 1984 for a discussion of such methods.) The operation of a specific empirical system can be understood. To do this, the theoretical tasks become particularly important. In general, a specific set of parameter values can generate many trajectories depending on where the process is activated. Empirically, we observe a very small number of instantiations (or realizations) of a generative process, and it is useful to locate these trajectories in the space of possible trajectories.

2.2. STATISTICAL CAUSALITY

> A causal law is a statement or proposition in a theory which says there exist environments in which change in the value of one variable is associated with a change in the value of another variable and can produce this change without change of other variables in the environment. (Stinchcombe 1968:31)

I think Stinchcombe (1968), in a very influential document, is writing about "propagated effects" in the sense of Doreian and Hummon (1976) and can be viewed, in a limited way, as consistent with the operation of system causality. Yet, the book was influential also in the causal modeling literature.

2.2.1. Structural Equation Modeling

Two of the most used tools in sociological research are (linear) regression and structural equation modeling. In this context, there is some sense that an effect is obtained from a cause with the expression of some model with parametric values. For regression, the most general statement of a model is

$$y = X\beta + \epsilon, \tag{6}$$

where ϵ is independently distributed as $N(0, \sigma^2 I)$. The variables in X are used to predict y through the estimation of the parameters (β and σ) in the model. Estimating the parameters of this type of model is more straightforward than estimating parameters in systemic causality models—which may account for their popularity. Some asymmetry is imposed by specifying the predictor variables and the response variable(s). For structural equation modeling (SEM), the observed variables are partitioned into those that are exogenous (whose variation is taken as a given) and the endogenous variables (whose variation is to be explained). The structural model can be stated as

$$\eta = \beta\eta + \Gamma\xi + \zeta, \tag{7}$$

where η is a vector of unobserved endogenous variable, ξ is a vector of unobserved exogenous variables, ζ is a vector of disturbance terms, and β and Γ are matrices of parameters to be estimated. There is also a measurement model given by

$$y = \Lambda_y\eta + \epsilon \text{ and } x = \Lambda_x\xi + \upsilon, \tag{8}$$

where ε and υ are vectors of white noise terms. The two Λ matrices specify which unobserved variables have which observed indicators.

The immense appeal here is the prospect of estimating equations with linked latent variables. Together, the structural model and measurement model, with specific numerical values, can be used to construct a covariance matrix for the observed variables, $\hat{\Sigma}$. This may or may not be close to the observed covariance matrix, S. If $\hat{\Sigma}$ is sufficiently close to *S*, the specific model (with its parameter values) is said to fit the data. And, if the correspondence between $\hat{\Sigma}$ and S is poor, then the model with its parameter values does not fit. The empirical task is to specify a substantively based model, estimate it, and assess its fit. (See Bollen and Long 1993 for a collection of methods for assessing the fit of an estimated SEM.) Models that do not fit tend to be respecified, reestimated, and tested again, an iterative process that ends with a fitted model (or no model at all). In the following, it is assumed that for a given covariance structure, there is at least one model that fits. It is in this sense that I use the term *statistical causality.*

2.2.2. Causal Modeling

For my purposes here, causal modeling is the use of statistical machinery to determine or locate causal relationships between variables. This includes sifting partial correlations, uses of regressions, path analysis, and the use of SEM. Although there are many ways of approaching this topic, I will focus on three.

From correlations to causality. Morgan (1997) provided a constructive history of the sifting of economic statistics in the form of time series. She described changes in the collective mindsets of those who sifted this kind of evidence. Initially, there was a concern to understand whole series of one (or more) variables with an emphasis on examining variation (as do causal modelers today.) Morgan argued that these (early) practitioners understood that "causes were generally treated as being historically contingent" (p. 61). There was a recognition also of a need to disentangle multiple causes and a recognition that different causes can operate in different periods.

Over time, it seemed that the mind-set shifted so that if a causal relation was assumed, the "correlation (could be) interpreted in terms of the strength of an atemporal cause-effect or functional relationship" (Morgan 1997:72). Morgan (1997) used the (delightful) term "causal

stories" for substantive tales woven around particular correlational analyses and concluded that "different causal stories could still be advanced and supported by appeal to the same set of data" (p. 73).

Yet another change in the mind-set came in the 1930s and 1940s with the recognition that correlations were rather limited and that multiple equations were needed. "The general causal mechanism was now understood to be embedded in the relationships *between* [italics added] the individual equations of the model" (Morgan 1997:75-6). This line of thought is reflected in the formulation and estimation of SEM models as described above. Although it may not be clear that the formulation of a set of equations, even when estimated simultaneously, captures the nature of the causality, the idea is reasonable. Morgan (1997) went on to observe that "such structural models provided the basis for a *causal story* in terms of causal processes which *defined* the time order and interaction of the multiple causes at work" (p. 76, italics added). There are two key ideas here: (1) the time order is defined (or assumed) and (2) a model is specified and estimated and then a causal story is told. I have no quarrel with this procedure when our collective ambition is modest. The claim to be able to detect causes through sifting covariance structures speaks to a much greater ambition.

Regression Equations. Clogg and Haritou (1997) focus attention on the use of regression equations in the enterprise of detecting casual connections and the problems that occur in such efforts. Problems arise when we look at data that are generated non-experimentally with the ambition of substituting statistical control—via incorporating variables—for randomization. As they noted, arguments that apply for one equation can be extended to the use of many equations in a multiple equation system.

Consider the equation $y = \beta X + \varepsilon$ as a simple bivariate regression equation (taken as an unconditional form with centered variables). The unconditional expectation is $E[y] = \beta E[X] + E[\varepsilon]$, which becomes $E[y] = \beta E[X]$ with the assumption $E[\varepsilon] = 0$. If the bivariate regression equation is multiplied by X and expectations taken, we have $\sigma_{yx} = \beta\sigma_x^2 + \sigma_{x\varepsilon}$, where σ_{yx} is the covariance between X and y, σ_x^2 is the variance of x, and $\sigma_{x\varepsilon}$ is the covariance of X and ε. Although both σ_{yx} and σ^2 are

available from the data, this equation cannot be solved unless $\sigma_{x\varepsilon}$ is known. If we assume (by convention) that $\sigma_{x\varepsilon} = E[X\varepsilon] = 0$, then $\hat{\beta} = \sigma_{yx}/\sigma^2$. Clogg and Haritou (1997) called $E[X\varepsilon] = 0$ a causal assumption. If we start with equation (6) as the regression equation in matrix form for a set of regressors, multiply by X′ and take expectations, we reach $E(X'y) = \beta E(X'X) + E(X'\varepsilon)$. Again, if we assume $E(X'\varepsilon) = 0$, obtaining β is possible. The specification $E(X'\varepsilon) = 0$ becomes a set of causal assumptions. Following the statement of the regression model (equation (6) in matrix form), the OLS regression estimator of β is given by $\hat{\beta} = (X'X)^{-1}(X'y)$ where, along the way, the assumptions $E(X'\varepsilon) = 0$ have been made. These assumptions can never be checked because the OLS procedure produces residuals, $\hat{\varepsilon}$, uncorrelated with all of the variables in X. The drawback with using more control variables, Z, is that they too must be specified as being uncorrelated with ε: $E[Z'\varepsilon] = 0$. Put differently, even if the concern is with the causal impact of X on y, the number of causal assumptions goes up with the inclusion of Z.

In an analysis for the parameter estimates, Clogg and Haritou (1997) showed that the numerical values of the $\hat{\beta}$ also turn on the correlations between X and ε (which are usually assumed to be zero.) They "conclude that nothing can be learned about the causal effect of X on y from the correlation(s) between X and y unless something is really known about the correlation between X and ε" (pp. 95-6). They observed that it would be more useful to think of plausible nonzero values for these correlations than to cling blindly to the notion that they are all zero.

Clogg and Haritou's (1997) conclusions are bleak in their implications for causal modeling: (1) "Causal inferences are driven more by assumptions than by data" (p. 101); (2) "It is impossible from the data to know which causal assumptions are true" (p. 103); and (3) "Finding the 'best' statistical (conditional) regression has no bearing whatsoever on the problem of making a causal inference about the effect of *X* on *y*" (p. 104). Their analysis extends to multiple equation systems: "It is difficult to distinguish between what is assumed and what is inferred from complex 'supermodels'" (p. 106). This makes it hard to keep the faith in making sound causal inferences from statistical procedures applied to conventional data.

Automated causal detection systems. Spirtes, Glymour, and Scheines (1993) have proposed imaginative methods for detecting causal models through the sifting of data in a linear world. They combine three fundamental ideas: (1) the use of directed acyclic graphs (DAGs) (which describe a causal structure), (2) probability theory (with a focus on conditional independence), and (3) the idea of causality. Although some SEM devotees may chafe at the apparent restriction of attention to recursive systems where the β in equation (7) is lower triangular, this does not seem a pressing problem. Given a causal model (as a DAG), a set of probability distributions for the predictor variables and the disturbance terms, the joint probability distribution of all of the included variables can be generated (as is the case for SEM). In particular, conditional independence configurations implied in the model can be assessed. At this level, different causal diagrams can be drawn and examined. For each, implied (conditional) independencies can be checked: Some of the causal diagrams will have patterns that are consistent with the empirical evidence whereas others will be inconsistent with the evidence.

There seems to be serious disagreement about the intent of so-called automated causal detection systems—whether in the form of old style causal modeling (sifting partial correlations and playing with path coefficients) or the newer, far more sophisticated methods. A simple purpose is to specify a specific causal model and check it out. This can be extended to having some causal models that are checked. At some point, there will be a practical and conceptual limit as to how many such models can be checked. There is an obvious question: Would it not be grand if we did not have to specify these models and yet still check them all automatically? An automated causal model detector would do just that and the use of TETRAD (Spirtes et al. 1993) can be seen as an artful way of accomplishing this task. But somewhere, a boundary between assessing a small number of (substantively based) models and checking them en masse has been crossed.

Freedman (1997) is among the critics who have expressed grave doubts about the methods developed by Spirtes et al. (1993). The spirited exchange between Freedman and Spirtes and Scheines (1997) is delightful and instructive for both what is written and what is not.

Freedman's point of departure, like that of Clogg and Haritou (1997), is regression. "Many treatments of regression seem to take for granted that the investigator knows the relevant variables, their causal order, and the functional form of the relationships among them; measurements of the independent variables are measured without error" (Freedman 1997:113). Clearly, Freedman sees many problems here. However, if all of these claims (or assumptions) are appropriate, then there is little problem in using regression and related techniques. Nor is the assumption of measuring independent variables without error a major problem, and this objection, in principle, is met through the use of measurement models, in the form of equation (8), specified with SEM. Of course, the arguments of Clogg and Haritou will give one pause regarding the claims of knowing the causal order of all of the relevant variables.

Freedman (1997) went further when he wrote, "I see no cases in which regression equations, let alone the more complex methods, have succeeded as engines for discovering causal relations" (p. 114). In part, this rests on the nature of the tools used and our assumptions concerning the empirical world. In the model-building and model-fitting realm, the tasks are mathematical and statistical. However, as Freedman noted, statements of cause are statements about the empirical world. The use of a specific DAG as a representation of some phenomenon appears to bring cause into the model by fiat. By itself, this is not a problem if the model is being tested. But, claims about the detection of causal models go beyond this. Freedman provided examples of nonsense models "detected" by using TETRAD. At face value, this is damaging to the case for the causal detection program. But, from a substantive view, this is not a serious problem as nonsense models can be dismissed. More problematic are models that are plausible but also incorrect. Although simulation studies can provide comfort that causal models can be detected when the linear causal model "generating" the data is known, the deeper problem is that empirically, the true model is unknown. Suddenly, the problems of detecting causal models begin to rival those of estimating parameters in a system causality model.

However, there is a sharp difference between these kinds of models. One tries to capture the generative mechanisms of social phenomena

while the other seeks a numerical summary in the form of a set of linked equations and their estimated parameters. If there is a concern with prediction, it seems that the system causality models have a better chance of success relative to the statistical causality models. Of course, this holds only if they can be estimated. There is one way to couple system causality ideas and the tools used in statistical causality modeling. When equations like equation (4) are solved to yield equations like equation (5), these estimation equations could be estimated via SEM procedures. I write "could" because it will be necessary to assume equilibrium conditions. Otherwise, the parameters in the regression or SEM equations are functions of time and other parameters. Including such transients greatly complicates estimation.

2.3. MECHANISM CAUSALITY AND SEQUENCES OF EVENTS

Looking at social mechanisms and sequences of events provides another approach to the issues raised by using causality in providing explanations of social organization. The link between statistical causality and this discussion is provided by N. Cartwright (1997). Her point of departure is the Spirtes et al. (1993:45) definition of a causal structure as

> an ordered pair, $< V, E >$, where V is a set of variables, and E is a set of ordered pairs of V where $< V, E >$ is in E if and only if X is a direct cause of Y relative to V.

Cartwright wrote, "alternatively, V can be a set of events. But we should not be misled into thinking we are talking about *specific events occurring at particular times and places* [italics added]" (p. 343).

A focus on specific events draws us into the arena of social mechanisms. As is the case with causality, *social mechanism* is not defined uniquely. But, the general idea as well as the theory goals are clear. For Hedström and Swedberg (1998), the theory goal is "to explicate the social mechanisms that generate and explain observed associations with events" (p. 1). They added that an approach "should not be confused with a purely descriptive approach that seeks to account for the unique chain of events that lead from one situation or event to another" (p. 1). This is a very subtle distinction: We observe sequences in the

empirical world and seek to account for them in terms of generalized mechanisms that are also sequences of some kinds of events. Hedström and Swedberg insisted that the distinction be made as they went on to observe that their vision for explanatory sociology includes the creation of "an ensemble of such fundamental mechanisms that can be used for explanatory purposes" (p. 2). For them, a simple description of a sequence of events is not an explanatory account.

This is consistent with Stinchcombe's (1998) characterization of a mechanism as

> bits of "sometimes true theory" or "model" that represent a causal process, that have some actual or possible empirical support separate from the larger theory in which it is a mechanism, and that generate increased precision, power or elegance in the large-scale theories. (p. 267)

He offered the example of monopoly power and competitive environments as mechanisms (that can also be coupled and help generate economic structures). Another example (Chase 1982; Fararo, Skvoretz, and Kosaka 1994) has fighting and bystander effects as two mechanisms generating dominance hierarchies among primates. Schelling's (1998) definition of an institution is close to that of Stinchcombe (1998): "A social mechanism is a plausible hypothesis, or set of hypotheses, that could be the explanation of some social phenomenon, the explanation being in terms of interactions between individuals and other individuals, or between individuals and some social aggregate" (pp. 32-3). (Schelling's formulation has obvious implications for network analysts.)

Another definition of a mechanism comes from Elster (1998): "Mechanisms are frequently occurring and easily recognizable casual patterns that are triggered under generally unknown conditions or with indeterminate consequences" (p. 44). A major substantive task for Elster is to establish mechanisms (compare Hedström and Swedberg 1998) and the conditions under which they are triggered.

In an earlier analysis, Elster (1983) asserted that the antonym of a mechanism is a black box. This is an image that social mechanism theorists appear to like: $i_1 \rightarrow [M_1] \rightarrow o_1$, where i_1 and o_1 are inputs and outputs, respectively, and M_1 is a black box. In causal modeling, i and o are variables, and the black box is present as a magical object

accepted by causal modelers. An emphasis on mechanisms takes us inside the black box and helps explain phenomena (and not variables—or, rather, covariances of variables).

The attack on the black box aspect of variable-oriented causal modeling is rather obvious. According to Elster (1998), commenting on the use of statistics, it is difficult to discern causality from correlations (something the causal modelers would not deny), and he claimed this is another reason why statistical explanations are weak and vulnerable. He is joined by Sørensen (1998) in an extended critique of the (blind) use of regression analysis where linear equations are confused for theory. However, Sørensen had a different definition of social mechanism, one that I think Elster would disavow: "It is an account of how a change in some *variables* [italics added] is brought about" (p. 240). Even so, because Sørensen's specific examples are much closer in spirit to those of the social mechanism theorists than to those of the causal modelers, I suspect that the primary feature of a social mechanism has little to do with the presence or absence of variables. Mechanisms as bits of theory or understandings are pieces of substantive knowledge. A commitment to social mechanisms does not exclude the idea of variables nor analyses of data constructed in terms of variables. If this is the case, then much of the attack on causal modeling, regression, and SEM for involving variables is misplaced. Also, I suspect that once inside the black box [M_1], we will encounter $i_2 \rightarrow [M_2] \rightarrow o_2$. This implies that the sting in the argument of how bad it is to not go inside the black box when using regression and SEM is rather mild.

2.4. ALGORITHMIC CAUSALITY

Discussions of social mechanisms suggest the relevance of rules as elements that govern social action and interaction. Hummon and Fararo (1995) described the emergence of computation as a crucial part of a scientific triad whose elements are "theory," "empirical," and "computation." Theory and computation are coupled to form simulations: Theoretical ideas inform the construction of simulated social actors that embody rules for action. When these rules are invoked, the simulation generates outcomes implied by the theory built into the rules of the simulation. These rules—deterministic or stochastic—are

built into the code of the simulation, hence the term *algorithmic causality.* Notions of object-oriented programming and parallel processing are used to generate rich processes. The rules can represent activities whose precise order is not determined in the simulation, yet the global outcomes depend on the order in which activities occur. If theories are expressed in parametric form, the behavior generated by differing combinations of the parameters can be explored. In short, the parametric space is explored to learn the implications of theory. See, for example, Flache and Macy (1997) or Fararo (1978).

Hummon and Fararo (1995) described the link between computation and empirical as "data analysis." This can be split into two parts. One comprises the conventional data analyses used to estimate parameters, make inferences, and the like. The second part is the computation done to generate simulation outcomes into which specific instantiations can be located. In the empirical world, we study a limited number of instantiations and, in general, cannot really locate those from the one empirical world in the set of all of the potential empirical worlds governed by the same process rules. The key notions here are twofold: (1) We are better able to look at our empirical world if we know more about how a single process operates under different (parametric or algorithmic) conditions and (2) our simulations (and theory expressed within them) improve when we examine their outcomes in relation to what empirical evidence we have.

3. CAUSALITY IN SOCIAL NETWORK ANALYSIS

The distinctions between the four types of causality are not truly tight. At some level, all are concerned with prediction and a broad term of predictive causality covers them. Although the algorithmic rules could be consistent with system causality in a broad view, the rules themselves might not be embodied in generating equations. Mechanism causality shades into algorithmic causality if the algorithmic rules capture the operation of the social mechanisms. Both system causality and statistical causality provide explicit foundations for precise predictions. To the extent that a dynamic system is specified correctly, the foundations for prediction seem more secure. But, in talking about the future and trying to predict what would happen if

variables and a small number of parameters (usually one) change, they are both vulnerable. Using counterfactuals—see, for example, Cox and Wermuth (1996)—carries great risk if other parameters change in a hypothetical future.

In general, experiments where variables are controlled and/or randomization is used are not very relevant in this discussion. True, the communication experiments of Leavitt (1951) and Bavalas (1948) have near-legendary status among network analysts for providing evidence that network structures of groups have relevance for collective outcomes and the location of actors in a network has relevance for actor outcomes. And, exchange theory driven experiments—for example, those discussed in the special issue of *Social Networks* (1992, Vol. 14, Nos. 3 and 4)—have provided great insight into studies of power and bargaining. However, the brutal truth is that most empirical studies of social networks are done in uncontrolled situations. For this realm of empirical work, experimental evidence provides, at most, suggestive insights.

3.1. STATISTICAL CAUSALITY AND NETWORK ANALYSIS

Attempts to study network behavior require the use of both network and attribute data. Even when a great emphasis is placed on the network within which the actor is embedded, it seems overly narrow to exclude actor attributes. If large organizations behave differently than small organizations (in some contexts), excluding size from the items used to understand organizational conduct seems risky. Of course, if we do have a very long sequence of data, we may be able to model the processes by which organizations change in size and learn that the distribution of size is fundamentally a network process. After all, as network analysts believe that location in a network can facilitate or restrict access to resources, this is not unreasonable. Similar arguments can be put forward for the operation of children's networks. At very young ages, there are many between-gender ties. These diminish as children get older—almost to the point of friendship choice's being completely segregated by gender in networks. As they get a little older, between-gender ties increase. This description is a kind of causal story where gender plays a role.

For networks of actors with attributes, there are two kinds of explanatory accounts. The ultimate network explanation is that everything—for example, gender (of people) and size (of organizations)—is generated through network processes. To empirically test this, we will need very, very long time series of observations. The second kind of explanation concerns phenomena as we study them without having to go back to primordial times. Large organizations do act differently than small organizations with regard to both intraorganizational structural processes and interorganizational structural processes. To explain the behavior of General Motors now with regard to say, industrialization and the global movement of capital, going back to the origins of the automobile industry at the end of the 19th century and the beginning of the 20th century is, at best, a distraction. It seems that men and women do behave differently—for whatever reason—in many social contexts. Gender is thought to have an impact on these processes and conditions them. The inescapable point is that social network analysts will have to deal with both attribute and network data.

Network analytic life might be more perverse. (Some) network analysts construct measures of centrality, prominence, status, or standing from network data for use as . . . variables to help characterize actors! These variables have been known to go into statements such as "The greater the centrality of an actor, the greater (or lesser). . . ." Does statistical causality help us determine causal relations? Probably not: All of the reasons that are cited for not being able to determine statistical causality unambiguously extend to the use of variables constructed from network data.

The conventional regression model $y = X\beta + \epsilon$ can be modified to incorporate structural (relational) information. This is exactly what network autocorrelation models (Doreian 1980, 1989) are designed to do. Given a network, some matrix W can be constructed to capture the relevant interdependency of actors. An effects model uses $y = \rho W + X\beta + \epsilon$ where ρ is a parameter to be estimated. The disturbance term, ϵ, is specified in the same way as for a regression model. A disturbances model is one where the regression model is unchanged but the disturbance is specified as $\epsilon = \rho W\epsilon + \upsilon$,with υ representing the white noise disturbance terms. A combined effects and disturbances model can be constructed as well as models with multiple autocorrelation regimes.

Regardless, network information can be brought into statistical equations in the narrow sense used here. But, is this useful for providing causal explanations?

Again, the answer is "probably not." Here the argument is stronger. Suppose W expresses in some fashion structural or regular equivalence and the estimate of ρ is significant. It would suggest that structurally or regularly equivalent actors exhibit similarity. If the data points are nations and W is defined in terms of trade, the causal story is obscure. If the colonies of England (or of France) are similar, understanding comes with incorporating mechanisms. If the tie between colonies and the colonial powers is one where the latter exploit the former, the mechanism provides the explanation of why colonies do not fare well. And, if the colonies of France and England exhibit the same properties, invoking arguments of regular equivalence seems an incomplete account. Again, colonial mechanisms seem more fruitful as an explanatory account than some appeal to equivalence with regard to structural location.

Statistical methods are used for network analysis in a variety of other ways. A rich tradition starting with the Holland and Leinhardt (1981) family of p_1 models, leading to Feinberg and Wasserman's (1981) application of log linear modeling tools, to Wasserman and Pattison's (1996) use of logit modeling methods to estimate p^* models provide clear evidence of the fruitful use of statistical procedures to analyze social networks.

Given the uses of statistical methods, it seems that statistical causality has obvious relevance for network analysis. The downside of this observation is that all of the limitations of statistical causality with regard to establishing cause apply. It will be extremely difficult to establish causality through the use of statistical tools when network data are analyzed. One interesting approach is that of Leenders (1997), who disentangles processes of selection (actors choosing actors like themselves) and contagion (where actors become similar to the actors to whom they are linked). An even stronger argument can be made. Within the "covariance approach" there is a systematic examination of causal diagrams (Pearl 1995) as ways of integrating statistical and substantive ideas in an attempt to discern causal relations. The focus is on reasoning with conditional probabilities (see, e.g., Pearl, Geiger, and Verma 1990) in a philosophical context where relations

between variables—represented in a DAG—are paramount. At a minimum, network analysts will need to construct—or adapt— such a framework to examine statistical causality for networks. Cox and Wermuth (1996) provided a helpful discussion of issues involved in the context of conventional variables.

3.2. SYSTEM CAUSALITY AND NETWORK ANALYSIS

System causality was cast in terms of generative rules or prediction equations for variables. One fragment of studying this concerns the relational ties between social actors. For two actors, i and j, let one of the ties between them be a_{ij}. If we abandon a static approach to describing networks, the ties between i and j are better written as $a_{ij}(t)$ to allow that these ties can change through time. Thus, if the relational tie concerns liking, $a_{ij}(t)$ represents the strength of i's liking of j at time t. Or, for the nations and the relation exports, $a_{ij}(t)$ is the volume of exports from i to j at time t. Expressed in these terms, the ties themselves are variables, and the language of both system causality and statistical causality apply to the relational items. Certainly, there will be greater difficulty in trying to establish formal models in explicit system terms and estimating them. Although Doreian (1979) provided a discussion of structural control models applied to the ties between actors in a small group, there was no discussion of estimating such models.

The general image is one of a system of n actors that are characterized by $\{x_r(t), y_s(t)\}$ as variables that change through time. If there is a set of relations $\{R_m(t)\}$ whose elements are also time varying, we have a system where both the relational ties and actor attributes can vary through time. The state space is characterized by variables measuring attributes and structures, all of which are to be modeled.

Stepping back from the differential equation imagery, Fararo (1989:80) described four choice sets: The state space variables can be continuous or discrete, the parameter space can be continuous or discrete, the time domain can be continuous or discrete, and the generator can be deterministic or stochastic. All combinations of these items can be used to describe dynamic systems. In principle, while these can be modeled, there are very few attempts to do so empirically.

3.3. EVENT SEQUENCES AND SOCIAL NETWORK MECHANISMS

It is possible to describe event sequences of both network and attribute data. A simple example is found in institutionalist theories of interorganizational relations and organizational forms. Consider the dyad of a mental health office (MHO), a large major funding organization for mental health care and a residential care provider (RHOME). Suppose further that the RHOME is critically dependent on the MHO for funds and clients. In general, there will be an interorganizational network containing multiple large funding organizations and many residential care facilities. Suppose we have data that show the residential facilities are structured in very similar ways. Consider the following hypothesis, one that fits well with statistical causality: "The greater the dependence of an organization on another organization, the more similar it will become to that organization in structure, climate, and behavioral focus" (DiMaggio and Powell 1991:74). Substantively, this is a nonstarter as it would be absurd for a small residential facility to structure itself as if it were a funding organization. A more compelling hypothesis would claim, "The greater the dependence of an organization on another organization, the more similar it will become in structure, climate and behavioral focus to yet other organizations that are dependent—in the same fashion—on the same (focal) organization."

We could construct an argument cast in terms of social mechanisms. Representatives of the focal organization (the MHO) could impose criteria—minimum bed sizes, small numbers of patients in a room, cleanliness standards, accounting procedures, and so forth—that the RHOME must satisfy if it is to receive clients and funding. There could be a sequence of negotiations (interactions) as a set of working arrangements is hammered out. Institutional theorists would recognize this as an example of a coercion mechanism. Standards are set and an organization must conform to them if it wants to receive further funding. If the RHOME falls out of compliance, further interactions are triggered. The actual sequences of interactions are empirical descriptions. Although they are of narrative interest, the explanation comes from seeing them as an integral part of a generalized (coercive) mechanism. Is this a causal explanation? It takes the form of identifying an empirical event sequence as an instantiation of

the coercion mechanism together with a specification of how this mechanism works. If there is causality involved, it comes from the general claim as to how the mechanism operates.

Is it possible to specify events that trigger the mechanism? Usually, it is tied to a (predictable, routinized) budgetary cycle, but it can be triggered by an unpredictable event (e.g., a patient dies or leaves the residential home without permission). Once invoked, it is assumed that the mechanism operates in a certain (causal) way, and a causal story can be told. But, is it truly causal? If the mechanism operates in the same fashion every time and always produces the same organizational structure, there is some basis for accepting a causal argument. Causality has been brought into the presumed operation of the coercive mechanism. This seems appropriate, but if the operation of the coercive mechanism does not lead to the same outcome every time, belief about the operation of a causal mechanism becomes fragile. The director of the RHOME could seek alternative sources of funding and not comply with the demands of the MHO. No doubt, a second-order mechanism could be constructed, but the analysis verges on being another just-so story.

Using social mechanisms as part of a causal explanation becomes much more difficult if there are multiple social mechanisms that could be operating. If the substantive issue is to understand how organizations (in a specific domain) are so alike, the operation of an institutionalized coercive mechanism provides one account. Consider a school of social work (SSW) that is coming up for accreditation from some licensing authority. The timetable for these events is fixed, and the accrediting rituals occur at predictable times. Part of the process is coercive: If the SSW does not have certain courses, does not have enough faculty with certain types of degrees, does not have a coherent field placement system, and so forth, it will not be accredited fully. But, is this a sufficient account for isomorphic structures among places such as the SSW? No, because there are rival mechanisms. There is a mimetic mechanism whereby the practices of successful organizations are copied by other organizations wanting to be successful (or wanting just to survive), and there are normative mechanisms whereby norms are followed because they specify the appropriate behaviors. This could be a normative mechanism—both with regard to the creation of norms (compare Wellman 1988, above) and

their role in constraining behavior. Put differently, a set of mechanisms is an element in a profession mechanism that governs behavior of professionals. In the SSW example, all three mechanisms could be operating. The explanatory task becomes one on which the mechanisms are disentangled in their operation and combined in their (joint) impact.

Abell (1987, 1993) advocated the use of narrative methods as a route toward the construction of social theory. He assumed "a social world to be comprised of a web of interconnected human actions and forbearances" (Abell 1993:94). With a focus on action, he noted that action "is characteristically situated in at least four senses": (1) Action is a/the consequence—usually in part—of prior actions, (2) action may be contingent on a set of circumstances, (3) beliefs and values of an actor inform the actions of an actor, and (4) actors can involve strategic calculation. He argued that laying out a sequence (or sequences) of actions and interactions constitutes a narrative. At this level, such a narrative is just an empirical sequence and, in Hedström and Swedberg's (1998) terms, cannot be viewed as a social mechanism. However, Abell (1987) sought to compare narratives by homomorphically mapping them to a set of well-defined and known structures. He talked of successive levels of abstraction that may bring his analyses into the realm of social network mechanisms. Where this is located in a sequence of homomorphic mappings from a very concrete empirical narrative to successive higher levels of abstracted structures is not clear. However, Abell (1987) constructed tools for establishing a set of homomorphic reductions of complexity into abstracted structures that can be formally compared. There is a remarkable parallel between Abell's diagrams connecting events and the diagrams of Cox and Wermuth (1996) connecting variables. This seems a parallel worthy of exploration.

3.4. ALGORITHMIC CAUSALITY AND NETWORK ANALYSIS

We can step further back and consider an algorithm as any well-defined computational procedure that takes some value (or a set of values) as input and produces a value (or a set of values) as output (Cormen, Lieserson, and Rivest 1990). This output can be viewed as including the new state of the system. Put differently, an algorithm is a sequence

of computational steps that transforms inputs to outputs and generates new system states. The transition rules considered in Section 2.1. are equational. However, a well-defined sequence of computational steps can capture process rules for many processes without having explicit equational generators. Indeed, the whole domain of theory-driven simulations (see, e.g., Heise 1995, Fararo and Hummon 1994) can be viewed as examples where dynamic generating processes transform systems. Whether equations are used is secondary. What truly counts is the set of process rules, the study of which helps us understand the operation of social processes. The research agenda of Fararo and Skvoretz (1986) on E-state structuralism is an attempt to use expectation states theory to unify a set of structuralist theories via a set of coherent rules governing structural action.

One successful area of structuralist theory is structural balance. When Heider (1946) laid out the foundations for balance theory, it was clear that empirical domain for the structural balance dynamics were in actors' heads. The generalization proposed by D. Cartwright and Harary (1956) was a brilliant example of formalizing theory. But, it came with a cost: The location of the dynamics was taken out of the minds of individuals and located at the level of a social group. Their basic structural theorem stated that if a group structure was balanced, the group members belong to two subgroups (later called plus-sets) where all of the positive ties were within subgroups and all of the negative ties were between subgroups. Davis (1967) generalized balance by defining the all-negative triad as balanced and established a second structure theorem: If a group is balanced, there are two or more plus-sets such that all positive ties are within plus-sets and all of the negative ties are between plus-sets. Doreian and Mrvar (1996) proposed a method for locating partitions of signed networks that are as close as possible to an ideal partition based on structural balance. They used the line index of imbalance as a general measure to be minimized in locating the set of best fitting partitions.

Hummon and Doreian (2001) developed a simulation based on structural balance theory and the set of partitions that are closest to a balanced configuration. All actors in the network have images of the overall signed network in which they are located. The simulation runs on two levels. One is inside the minds of the actors and a balance mechanism is specified as a set of rules whereby actors move toward

balance by changing one line (either its sign or deleting it) and reporting the change to the group (as a second level). The actor making the change can report to the group in a variety of ways: to the person involved in the specific changed tie, to actors connected via a positive tie (friends), to actors connected by any tie (positive or negative), or to the whole group (broadcasting). The group "signed structure" is constructed from the reports of tie changes. The relevant point for this discussion is that the group network can be different to the actor images of the network, and the actor images can differ from one another.

The partitions of the group network, obtained from a group-level process, are returned to the actors as information to be incorporated by them as they reflect on their image of the signed network. In general, an actor has more than one option in trying to move toward balance. This ambiguity is resolved by having the actors select one option randomly. For this discussion, there are two important implications.

First, the balance mechanism is specified and operates: All individual images as well as the group structure do move toward balance. This is not surprising nor is there much learned from the existence of change in a given direction. What is a surprise is the sheer number of terminal states: One process with fixed algorithmic rules can generate many outcomes even when the same process operates. Furthermore, the process can generate different outcomes from the same starting point (as initial conditions). This has serious implications in the theory-empirical-computation triad of Hummon and Fararo (1995) and especially for the empirical-theory link. (It has implications also for system causality and statistical causality.) In the empirical world, we observe a relatively small number of instantiations of an assumed process. Certainly, the number of observed outcomes will be tiny compared to the number of outcomes that could be generated by the same process. In the simulation study, one outcome was a single positive plus-set whereas another had many plus-sets and a large measure of imbalance. Yet another comprised a small number of plus-sets that were mutually hostile. Some of these outcomes were balanced, but others were not. The same process rules generated all of them. Is this analysis causal? The same arguments hold as in Section 2. Causality comes from mapping a presumed mechanism generating outcomes to an empirical network. Causality is presumed in the operation of the network mechanism and not detected. In short, a causal story can be

constructed to go with the presumed operation of a mechanism with a very large number of potential outcomes.

The second implication concerns the vexing problem in balance theory with regard to its major empirical hypothesis that signed structures move toward balance. Often, the groups did not reach exact balance. Some of the simulation outcomes are networks where each individual's image of the structure was balanced but the group network was not balanced. This is substantively appealing as it is an account of how the balance mechanism can operate without leading to a balanced group network. The simulation is driven by an operationalized social network mechanism in the form of algorithmic rules. The large number of potential outcomes suggests a stochastic mechanism rather than a deterministic one. The specific change rule once a tie is selected for a change is deterministic—for the single change of a single tie by one actor. But, the changes can be executed in different sequences in different runs. The sequencing is important: Different individual and group trajectories are generated. Staying within the realm of a simulation, explorations of theories are possible and appealing. But, does it establish cause empirically? Not really: If an empirical outcome matches one simulation outcome, it does not follow that the empirical outcome was generated by the kind of process built into the simulation. Nor does an empirical outcome that differs from the known simulation outcomes mean it was not generated by that process.

4. CONCLUSIONS

To get at causes requires a temporal framework. Some sort of process has to be defined and seen to be operating to generate a sequence of outcomes. This suggests that some combinations of system causality, mechanism causality, or algorithmic causality have the most promise. Yet, this promise seems fragile—and our grasp of it tenuous. System causality requires that we get all of the process rules right and can estimate all (or enough) of the relevant parameters. Mechanism causality is the most seductive as it seems to permit a simultaneous description of the form of networks together with events that generate both actor and network outcomes. This line of analyses builds the causes into the functioning of the mechanisms. To the extent that

mechanisms are bits of sometime true theory and represent our current state of knowledge, this has considerable appeal. If the empirical realm is mapped cleanly—in the sense that the mechanism is relevant and veridical for the process—"causes" for the outcomes can be discerned. The obvious drawback is that there is much circularity in this kind of argument. Algorithmic causality is very flexible as it uses a set of rules and the operation of these rules generates specific outcomes. But, there is a huge amount of indeterminacy as the operation of the algorithmic rules can generate many different trajectories—and many of these will never be observed.

Our basic goal is to look at the empirical world and construct some understanding as to how it works. Within the social network framework, social network analysts can continue to examine network structures to discern the consequences for actors located in networks and outcomes for the whole network. We will continue to describe networks and attempt to link these descriptions to network outcomes. Asking if these analyses are therefore causal in the sense of determining causes of either actor or network outcomes may be an impossibly broad question. The basic problem is that causal explanations are constructed on the basis of empirical evidence expressed in descriptions. They can take the form of equations, mechanisms, or rules—or combinations of these. But, they remain specific descriptions. For all of the types of causality discussed here, moving from a formulated causal machine to generated data that reflect the generative mechanisms is easy. Traveling in the reverse direction is much harder with the difficulty verging on the impossible. This may help explain why simulation is a popular approach for exploring the implications of a theory. The intrinsic empirical difficulties can be ignored safely, and many of the insights from simulations are appealing and oh so plausible.

A narrative as a straightforward description of a sequence of events has considerable appeal. Most network analysts who study empirical phenomena use narrative. In part, it is window dressing, but it has more than surface interest. The risk is that the narrative becomes yet another just-so story with events following each other in time under convenient stage management. Once it is recognized that the only real connection between the described events is merely temporal, the causal enterprise is shaken. If a different event could follow a given

event—which happens—the coupling of the events in a narrative is loosened. And, if there could be other outcomes between two hitherto sequential events that appear in a set of narratives, the tight coupling between events is lost again. Here, Abell's (1987) tools have potential at some homomorphic level to provide rigor for constructing narratives.

The most hard-nosed assessment is that truly establishing causality in network analysis is impossible—just as it is in the realm of statistical causality. Perhaps this answer is premature, and the question is ill posed. There is a far more constructive conclusion: There needs to be a very tight coupling of theory, mechanisms, and credible empirical information before we can delineate the actual operation of causes in the empirical world before we can tell causal stories. Understanding generative mechanisms—be they system, mechanism, or algorithmic—seems the most promising way to proceed. The role of statistical causality is a source of potential data analytic tools that can be mobilized within analyses conducted in the spirit of the other three types of causality. The rigor of thought in the form of causal diagrams will be another very useful import from the domain of statistical causality. Two further points follow: (1) Scorning such a fruitful source is foolish and (2) until we do make significant progress with the incorporation of causality into network analysis, it seems best not to crow about the alleged supremacy of network analytic accounts of how the world works.

REFERENCES

Abell, P. 1987. *The Syntax of Social Life*. Oxford, UK: Clarendon.

Abell, P. 1993. "Some Aspects of Narrative Method." *Journal of Mathematical Sociology* 18:93-134.

Batchelder, W. H., E. Kumbasar, and J. Boyd. 1997. "Consensus Analysis of Three-Way Social Network Data." *Journal of Mathematical Sociology* 22:29-57.

Bavalas, A. 1948. "A Mathematical Model of Group Structure." *Human Organization* 7:16-30.

Berkowitz, S. D. 1982. *An Introduction to Structural Analysis: The Network Approach to Social Research*. Toronto, Canada: Butterworths.

Bollen, K. A., and J. S. Long. 1993. *Testing Structural Equation Models*. Newbury Park, CA: Sage.

Cartwright, D., and F. Harary. 1956. "Structural Balance: A Generalization of Heider's Theory." *Psychological Review* 63:277-92.

Cartwright, N. 1997. "What Is Causal Structure?" Pp. 343-57 in *Causality in Crisis: Statistical Methods and the Search for Causal Knowledge in the Social Sciences*, edited by V. R. McKim and S. Turner. Notre Dame, IN: University of Notre Dame Press.

Chase, I. 1982. "Dynamics of Hierarchy Formation: The Sequential Development of Dominance Relationships." *Behavior* 80:218-40.

Clogg, C. C., and A. Haritou. 1997. "The Regression Method of Causal Inference and a Dilemma Confronting This Method." Pp. 83-112 in *Causality in Crisis: Statistical Methods and the Search for Causal Knowledge in the Social Sciences*, edited by V. R. McKim and S. Turner. Notre Dame, IN: University of Notre Dame Press.

Cormen, T. H., C. E. Lieserson, and R. L. Rivest. 1990. *Introduction to Algorithms*. Cambridge, MA: MIT Press.

Cox, D. R., and N. Wermuth. 1996. *Multivariate Dependencies: Models, Analysis and Interpretation*. London: Chapman and Hall.

Davis, J. A. 1967. "Clustering and Structural Balance in Graphs." *Human Relations* 20:181-87.

DiMaggio, P. J., and W. W. Powell. 1991. "The Iron Cage Revisited: Institutional Isomorphism and Collective Rationality in Organizational Fields." Pp. 63-107 in *The New Institutionalism in Organizational Analysis*, edited by W. W. Powell and P. J. DiMaggio. Chicago: University of Chicago Press.

Doreian, P. 1979. "Structural Control Processes for Group Processes." Pp. 201-21 in *Perspectives on Social Network Research*, edited by P. W. Holland and S. Leinhardt. San Diego, CA: Academic Press.

Doreian, P. 1980. "Linear Models with Spatially Distributed Data: Spatial Disturbances or Spatial Effects?" *Sociological Methods & Research* 9:29-60.

Doreian, P. 1989. "Network Autocorrelation Models: Problems and Prospects." Pp. 369-89 in *Spatial Statistics: Past, Present and Future*, edited by D. A. Griffith. Institute of Mathematical Geography.

Doreian, P. 1995. "Social Network Analysis as a Scientific Revolution: Thinking in Circles or Genuine Progress?" Pp. 1-21 in *International Conference on Social Networks*, edited by M. G. Everett and K. Ronnolls. London: University of Greenwich.

Doreian, P., and N. P. Hummon. 1976. *Modeling Social Processes*. New York: Elsevier.

Doreian, P., and A. Mrvar. 1996. "A Partitioning Approach to Structural Balance." *Social Networks* 18:149-68.

Doreian, P., and F. Stokman, eds. 1997. *Evolution of Social Networks*. New York: Gordon and Breach.

Elster, J. 1983. *Explaining Technical Change*. Cambridge, UK: Cambridge University Press.

Elster, J. 1998. "A Plea for Mechanisms." Pp. 45-73 in *Social Mechanisms: An Analytic Approach to Social Theory*, edited by P. Hedström and R. Swedberg. Cambridge, UK: Cambridge University Press.

Fararo, T. J. 1978. "An Introduction to Catastrophies." *Behavioral Science* 23:291-317.

Fararo, T. J. 1989. *The Meaning of General Theoretical Sociology: Tradition and Formalization*. New York: Cambridge University Press.

Fararo, T. J., and P. Doreian. 1984. "Tripartite Structural Analysis: Generalizing the Breiger-Wilson Formalism." *Social Networks* 6:141-76.

Fararo, T. J., and N. P. Hummon. 1994. "Discrete Event Simulation and the Theoretical Models in Sociology." *Advances in Group Processes* 11:25-66.

Fararo, T. J., and J. Skvoretz. 1986. "Unification Research Programs: Integrating Two Structural Theories." *American Journal of Sociology* 92:1183-1209.

Fararo, T. J., J. Skvoretz, and K. Kosaka. 1994. "Advances in E-State Structuralism: Further Studies in Dominance Structure Formation." *Social Networks* 16:233-65.

Feinberg, S. E., and S. S. Wasserman. 1981. "Categorical Data Analysis of Single Sociometric Relations." Pp. 156-92 in *Sociological Methodology, 1981*, edited by S. Leinhardt. San Francisco: Jossey-Bass.

Flache, A., and M. W. Macy. 1997. "The Weakness of Strong Ties: Collective Action Failure in a Highly Cohesive Group. Pp. 19-44 in *Evolution of Social Networks*, P. Doreian and F. Stokman. New York: Gordon and Breach.

Freedman, D. A. 1997. "From Association to Causation From Regression." Pp. 113-61 in *Causality in Crisis: Statistical Methods and the Search for Causal Knowledge in the Social Sciences*, edited by V. R. McKim and S. Turner. Notre Dame, IN: University of Notre Dame Press.

Hedström, P., and R. Swedberg. 1998. "Social Mechanisms: An Introductory Essay." Pp. 1-31 in *Social Mechanisms: An Analytic Approach to Social Theory*, edited by P. Hedström and R. Swedberg. Cambridge, UK: Cambridge University Press.

Heider, F. 1946. "Attitudes and Cognitive Organization." *Journal of Psychology* 21:107-12.

Heise, D. R. 1995. *Journal of Mathematical Sociology: Sociological Algorithms*. New York: Gordon and Breach.

Holland, P. W., and S. Leinhardt. 1981. "An Exponential Family of Probability Distributions for Directed Graphs." *Journal of the American Statistical Association* 76:33-65.

Hummon, N. P., and P. Doreian. 2001. *Dynamics of Social Balance Processes*. Pittsburgh, PA: University of Pittsburgh.

Hummon, N. P., and T. J. Fararo. 1995. "The Emergence of Computational Sociology." *Journal of Mathematical Sociology* 20:79-87.

Leavitt, H. J. 1951. "Some Effects of Certain Communication Patterns on Group Performance." *Journal of Abnormal Psychology* 46:38-50.

Leenders, R.T.A.J. 1997. "Longitudinal Behavior of Network Structure and Actor Attributes: Modeling Interdependence of Contagion and Selection." Pp. 149-84 in *Evolution of Social Networks*, edited by P. Doreian and F. Stokman. New York: Gordon and Breach.

Morgan, M. S. 1997. "Searching for Causal Relations in Economic Statistics: Reflections From History." Pp. 47-80 in *Causality in Crisis: Statistical Methods and the Search for Causal Knowledge in the Social Sciences*, edited by V. R. McKim and S. Turner. Notre Dame, IN: University of Notre Dame Press.

Padulo, L., and M. Arbib. 1974. *System Theory: A Unified State-Space Approach to Continuous and Discrete Systems*. Philadelphia: W. B. Saunders.

Pearl, J. 1995. "Causal Diagrams for Empirical Research." *Biometrika* 82:669-710.

Pearl, J., D. Geiger, and T. Verma. 1990. "The Logic of Influence Diagrams." Pp. 67-87 in *Influence Diagrams, Belief Nets and Decision Sciences*, edited by R. M. Oliver and J. Q. Smith. New York: John Wiley.

Rogers, E. M. 1987. "Progress, Problems and Prospects for Network Research: Investigating Relationships in the Age of Electronic Communication Technologies." *Social Networks* 9:285-310.

Schelling, T. C. 1998. "Social Mechanisms and Social Dynamics." Pp. 32-44 in *Social Mechanisms: An Analytic Approach to Social Theory,* edited by P. Hedström and R. Swedberg. Cambridge, UK: Cambridge University Press.

Snijders, T. A. 1997. "Stochastic Actor-Oriented Models for Network Change." Pp. 185-208 in *Evolution of Social Networks*, edited by P. Doreian and F. Stokman. New York: Gordon and Breach.

SPrensen, A. B. 1998. "Theoretical Mechanisms and the Empirical Study of Social Processes." Pp. 238-66 in *Social Mechanisms: An Analytic Approach to Social Theory*, edited by P. Hedström and R. Swedberg. Cambridge, UK: Cambridge University Press.

Spirtes, P., C. Glymour, and R. Scheines. 1993. *Causation, Prediction, and Search. Lecture Notes in Statistics 81*. New York: Springer-Verlag.

Spirtes, P., and R. Scheines. 1997. "Reply to Freedman." Pp. 163-76 in *Causality in Crisis: Statistical Methods and the Search for Causal Knowledge in the Social Sciences*, edited by V. R. McKim and S. Turner. Notre Dame, IN: University of Notre Dame Press.

Stinchcombe, A. L. 1968. *Constructing Social Theories*. New York: Harcourt, Brace and World.

Stinchcombe, A. L. 1998. "Monopolistic Competition as a Mechanism: Corporations, Universities, and Nation-States in Competitive Fields." Pp. 267-305 in *Social Mechanisms: An Analytic Approach to Social Theory*, edited by P. Hedström and R. Swedberg. Cambridge, UK: Cambridge University Press.

Tuma, N., and M. T. Hannan. 1984. *Social Dynamics: Models and Methods*. Orlando, FL: Academic Press.

Wasserman, S., and K. Faust. 1994. *Social Network Analysis: Methods and Applications*. Cambridge: Cambridge University Press.

Wasserman, S., and P. Pattison. 1996. "Logit Models and Logistic Regressions for Social Networks: An Introduction to Markov Graphs and p*." *Psychometrika* 61:401-25.

Wellman, B. 1988. "Structural Analysis: From Method and Metaphor to Theory and Substance." Pp. 19-61 in *Social Structures: A Network Approach*, edited by B. Wellman and S. D. Berkowitz. University of Cambridge, Cambridge, England.

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