CONCEPTS & SYNTHESIS
EMPHASIZING NEW IDEAS TO STIMULATE RESEARCH IN ECOLOGY

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Scientist's guide to developing explanatory statistical models using causal analysis principles

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Abstract. Recent discussions of model selection and multimodel inference highlight a general challenge for researchers: how to convey the explanatory content of a hypothesized model or set of competing models clearly. The advice from statisticians for scientists employing multimodel inference is to develop a well-thought-out set of candidate models for comparison, though precise instructions for how to do that are typically not given. A coherent body of knowledge, which falls under the general term *causal analysis*, now exists for examining the explanatory scientific content of candidate models. Much of the literature on causal analysis has been recently developed, and we suspect may not be familiar to many ecologists. This body of knowledge comprises a set of graphical tools and axiomatic principles to support scientists in their endeavors to create "well-formed hypotheses," as statisticians are asking them to do. Causal analysis is complementary to methods such as structural equation modeling, which provides the means for evaluation of proposed hypotheses against data. In this paper, we summarize and illustrate a set of principles that can guide scientists in their quest to develop explanatory hypotheses for evaluation. The principles presented in this paper have the capacity to close the communication gap between statisticians, who urge scientists to develop well-thought-out coherent models, and scientists, who would like some practical advice for exactly how to do that.

Key words: causal analysis; causal diagrams; explanatory models; multimodel averaging; multimodel comparison; path analysis; regression; science methodology; structural equation modeling.

Introduction

Building regression models using a set of candidate explanatory variables is a common practice in ecology. In recent years, Burnham and Anderson (2002) have championed an approach for choosing amongst alternative models using Akaike information (AIC) and multimodel inference. As a result, the use of information criteria and multimodel comparisons has become common practice in many disciplines, particularly in the environmental sciences (Symonds and Moussalli 2011). A careful reading of the literature on model comparison and selection reveals persistent mention by statisticians of fundamental difficulties with ascribing explanatory interpretations to the coefficient estimates obtained using regression models. Burnham and Anderson (2002) highlighted this pervasive issue in their classic text on multimodel inference (pp. 440–441), where they criticize ecologists for not doing enough "critical thinking" when crafting candidate models for evaluation. In the absence of a carefully crafted set of competing models, model selection cannot lead to scientifically interpretable findings. The proposed remedy, given repeatedly in the Burnham and Anderson writings, is simply for the scientist to "think hard" about the candidate models. Scientists might wonder, however, "*How* am I supposed to 'think hard' about my candidate model set? Where is the chapter in Burnham and Anderson or other statistics books that present the fundamental rules for thinking hard about candidate models in a set so that scientific interpretability is supported?" The unambiguous message being conveyed by statisticians is that it is the responsibility of the scientist to bring to the analysis a set of candidate models with clearly distinguishable scientific content. The reason for this is that the necessary expert

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knowledge to permit interpretation lies with the scientist, not the statistician. This raises the question of where scientists can find a set of general rules for developing well-reasoned explanatory hypotheses.

We propose that the answer lies in the logical system referred to as *causal analysis* (Pearl 2000, 2009, Hernán and Robins 2020). Recent advances in the field of causal analysis provide the core principles for how to evaluate the causal content of competing hypotheses. Some aspects of modern causal analysis will be familiar to ecologists through the related literature on structural equation modeling (SEM; Shipley 2000*a*, 2016, Grace 2006), whereas other elements are relatively new and ecological examples are scarce. In this paper, we first illustrate the challenges that accompany attempts at drawing scientific inferences from conventional regression models. We then describe and illustrate a set of principles to guide scientists when developing explanatory hypotheses for evaluation.

The Problem with Drawing Scientific Interpretations from Multiple Regression Models

The challenge to drawing causal conclusions by employing conventional regression methods is tied, in part, to the limited capacity of such models to represent complete hypotheses. This problem can be made more tangible through graphical representation (Fig. 1). As revealed in Fig. 1A, the predictors in a multiple regression model can be (and typically are) intercorrelated. However, no causal explanations for the correlations among predictors are specified as part of the regression model. Why are X_1 and X_2 correlated? Is it because of some unmeasured common cause? Perhaps X_1 has a causal influence on X_2? Perhaps the other way around? As shown in Fig. 1B, there are many possible reasons for three predictors to be correlated, each implying a different scientific explanation for the nature of the relationships between predictors and response. Because scientific knowledge about possible explanations for the intercorrelations cannot be encoded in the multiple regression equation because of its simplicity, the hypothesis represented by a multiple linear regression model (Fig. 1A) is incomplete regarding potential mechanisms underlying observed correlations, and thereby, interpretationally ambiguous.

Compounding the problem of interpreting regression coefficients is the fact that the correlations among predictors have profound effects on the estimated regression coefficients. To show how this is manifested in regression studies, let us turn to a specific example. In the fall of 2003, Keeley et al. (2005) established 1,000-m^2 plots in each of 90 sites to investigate the ecological effects of a series of wildfires that burned through southern California. The investigators measured a set of variables that might explain variations in postfire vegetation recovery throughout the region. Measurements included: landscape position variables (distance from the coast and elevation), prefire stand age (estimated from ring counts of stems), fire severity (based on skeletal remains of shrubs), and vegetation cover the spring following the fires. The intercorrelations among variables in the wildfire study (Table 1) were only moderately strong; yet, intercorrelation still presents a substantial problem for interpreting the contributions of individual factors, as we will show.

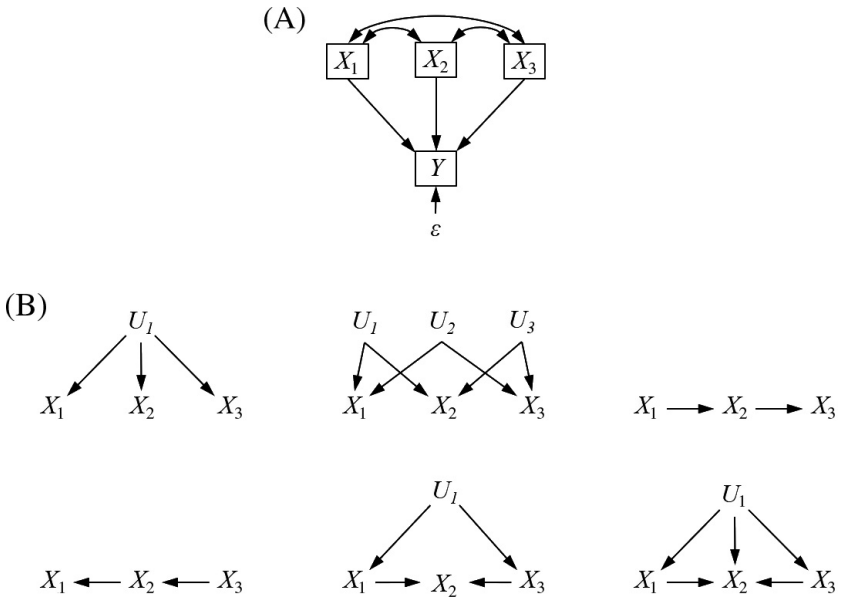

Fig. 1. (A) Graphical depiction of a regression model. Observed variables are in boxes and ε represents errors of prediction. Double-headed arrows represent intercorrelations and regression relationships by unidirectional arrows. (B) Six potential causal hypotheses for the three correlated predictors (X_1–X_3). *U* variables represent unmeasured causes.

Table 1. Bivariate correlations among variables used in the wildfire example.

	vegcover	*firesev*	*age*	*elev*	*coastdist*
vegcover	1				
firesev	−0.437	1			
age	−0.350	0.454	1		
elev	0.218	−0.117	0.093	1	
coastdist	0.243	−0.278	−0.278	0.606	1

Using conventional regression, all-subsets model comparison might be employed (Fisher et al. 2018). In the case of four explanatory variables, there are 16 possible regression models that could be specified (Table 2). Candidate models were estimated and ranked (Table 3; Appendix S1, Data S1), and ultimately a single model selected for scientific interpretation (model 12 in this case). The reader can observe in Table 2 that the unstandardized partial regression coefficients obtained from different models exhibit substantial variations in magnitude depending on the other predictors included. In the case of distance from the coast, for example, values actually shift from positive (0.875 in model 5) to negative (−0.201 in model 16). The mathematical reasons for these shifts are well understood (Cohen et al. 2003). Each regression coefficient is adjusted (also known as partialed) based on all of the other predictors in the model. The fundamental mathematical rule is that the bivariate associations between *X*s and *Y* are taken to be fixed values regardless of the other predictors in a model. When predictors are strongly correlated, adding or subtracting a predictor from a regression model can lead to large changes in the regression coefficients for the included predictors, including reversals of sign. In regression models, such as Fig. 1A, understanding the scientific implications of such shifts in coefficients is extremely difficult and there is a long history of failed efforts (reviewed by Elwert and Winship 2014, Pearl and MacKenzie 2018). For the interested reader, we provide an illustration of exactly why coefficients change when variables are added to a model in Appendix S2, Data S2.

Although the problem with interpreting coefficients from regression models has long been recognized, it was recently brought to the fore by a series of papers discussing problems with the practice of model averaging (Cade 2015, Fieberg and Johnson 2015, Banner and Higgs 2017). Among those papers, Cade (2015) provided the bluntest assessment when he said that averaging coefficients over models with different sets of predictors produces "muddled" inference because the scientific meaning of coefficients shifts whenever a predictor is added or removed. In this paper, we seek to clarify, from the perspective of the scientist, why regression models are difficult to use for explanatory modeling and what can be done to remedy that situation. To accomplish this, we provide investigators with a work flow for explanatory modeling, a set of principles for developing causal hypotheses, and references to statistical modeling methods for evaluating those hypotheses against data. Before diving into those key parts of our presentation, we must first provide some essential background for understanding causal effects.

Table 2. All-subsets regression models for vegetation recovery following wildfire.

	Explanatory variables included in the model			
Model no.	Fire severity	Preburn age of stand	Elevation	Distance from coast
1				
2	−0.084			
3		−0.088		
4			0.027	
5				0.875
6	−0.067	−0.048		
7	−0.080		0.021	
8	−0.077			0.475
9		−0.094	0.031	
10		−0.077		0.568
11			0.014	0.634
12	−0.059	−0.058	0.025	
13	−0.063	−0.043		0.373
14	−0.078		0.018	0.155
15		−0.097	0.033	−0.093
16	−0.060	−0.062	0.029	−0.201

Notes: Numbers in cells are raw coefficients relating predictors to vegetation cover in the various models. Coefficients for a predictor can vary among models, depending on the other variables included (e.g., distance from coast).

Operational Definition of a Cause–Effect Relationship—Counterfactuals and Potential Outcomes

One approach to thinking about cause–effect rests on the concept of *counterfactuals*. The counterfactual question is, "What would have happened if…?" Behind this question is an implied manipulation of the sort conceived of in an idealized experiment, that is, "What would have been the fate of an individual or sample if they had been in the control group instead of the treatment group?" This conceptualization of the problem allows causal effects to be defined using either experimental or observational data, which is a major part of its appeal.

Consider an ecological experiment investigating the effects of pesticides on frogs. Our concept of a causal effect assumes each individual frog has *two potential outcomes*, the individual's potential response after exposure to pesticide and their potential response to nonexposure (Neyman 1923, Rubin 1974). Only one of these will be observed. Let us denote the treatment variable as *Tmt* (e.g., exposure to pesticide or not). The question the experiment is attempting to answer is "What would have happened to individual *i* (or individual sample unit *i*), which did not receive the treatment (i.e., was assigned to the control group, *Tmt* = 0), if that individual had been assigned to the pesticide exposure group (*Tmt* = 1)." The causal effect of the treatment is estimated by the

difference between the two outcomes (treated or not treated). In this two-treatment case, each individual subject has two potential outcomes:

- Y_{i1}: potential outcome if individual i receives Treatment = 1 (exposed to pesticide).
- Y_{i0}: potential outcome if same individual receives Treatment = 0 (not exposed).

In the case of explanatory models based on observational data, we can exploit Pearl's (2000) introduction of a new mathematical operator, the *do*-operator, to express the idea of a hypothetical manipulation. With this operator, we can unambiguously represent the probability for an individual's hypothetical response when a treatment variable *Tmt* is set to the specific value t (which is 0 or 1 in this case) as $P[Y|\text{do}(Tmt = t)]$. Notice the ability to manipulate the variable *Tmt* physically is not required (Bollen and Pearl 2013), though the ability to imagine the value of *Tmt* being set to some value by humans or nature is implied. Note that the common statistical notation "$P(Y|X = x)$" means the "probability of observing Y, given that X is observed to equal x," whereas Pearl's notation "$P(Y|\text{do}(X = x))$" means "the probability of observing Y, given that X is forced to equal x." In general $P(Y|X = x)$ is not equal to $P(Y|\text{do}(X = x))$. Pearl's do-calculus tells us when the two are equal and when they are not equal (Pearl et al. 2016: Chapter 3).

This conceptualization of causal relations reveals a problem, but also a possible solution to that problem. The problem is that we can only observe one of the potential outcomes for any given individual frog during an experiment. Therefore, individual-level causal effects are counterfactual and not directly observable, even in randomized experiments. For this reason, the focus is typically on the average causal effect, *ACE*, where

$$ACE = E[Y_1] - E[Y_0] \quad (1)$$

averaged across all i. When the consistency assumption (Pearl 2009:99) holds,

$$E[Y_1] = E[Y|T = 1] \quad \text{and} \quad E[Y_0] = E[Y|T = 0] \quad (2)$$

In words, Eq. 2 says that the potential outcomes for the total population, $E[Y_1]$ or $E[Y_0]$ are being inferred from the subpopulations assigned to the treatments, $E[Y|T = 1]$ and $E[Y|T = 0]$. For observational studies, this assumption permits causal effects to be estimated from data as long as confounding effects are controlled for (i.e., the treatment subpopulations are comparable). This is a critical and also powerful assumption that determines our ability to infer causal information from observational (but also experimental) data. Note that this conceptualization can be extended to the situation where X is continuous, in which case the causal effect is captured in a parameter or set of parameters (for complex functions).

Explanatory Modeling in Action

In this paper, we make a fundamental distinction between the hypothesis and the inferential model. The structure of the hypothesis and its logical requirements are independent of any particular set of data. Inferential models, however, are inevitably dependent on the available data and the statistical machinery used for estimation and evaluation. Based on this distinction, we place causal analysis, a set of procedures for examining hypotheses independent of data, into the broader context of explanatory modeling, which encompasses all the information that influences the interpretation of the results (Fig. 2). Causal analysis describes the scientific assumptions upon which explanatory interpretations depend, whereas the latter must consider the imperfections of the data, the limits of our knowledge, and the need to accommodate computational relationships in models. Because it depends so critically on knowledge outside of an individual study, explanatory modeling is best thought of as a process that builds confidence in our understanding through a series of investigations. In this paper, our presentation focuses on causal analysis to serve our objective of showing how to think hard about the scientific content of candidate models. More about the details associated with explanatory modeling, moving from theory to models to results to interpretations, is outlined in Grace et al. (2012) and Shipley (2016: Chapter 8). Many of the general points we present are widely applicable. However, the specific techniques herein will most commonly be useful in observational studies and field experiments with important covariate effects.

An overview of explanatory modeling is presented in Fig. 2. The first step, assembling background information about the system under study, is critical to success and represents the mechanistic foundation required for scientific interpretations. Step 2 is the process of causal analysis, which describes the requirements for drawing scientific (cause–effect) interpretations, our primary focus in this paper. Step 3 refers to the subsequent step where models are confronted with data and their testable implications are evaluated. This step is described by the literature on structural equation modeling (Appendix S3, Part S1). Step 4 relates to drawing interpretations with proper respect for assumptions. Finally, confident interpretations ultimately depend on the consistency of findings and our knowledge of mechanisms, which we refer to as *sequential learning* (Step 5). Although this step is widely recognized as important, it is often omitted from formal discussions of the requirements for drawing causal inferences.

Four Key Principles of Causal Analysis—the Practice of "Thinking Hard"

Principle 1: Causal networks provide a powerful and convenient interpretive structure for causal analysis

The first of the four principles for causal analysis we present in this paper (Table 4) deals with the merits of

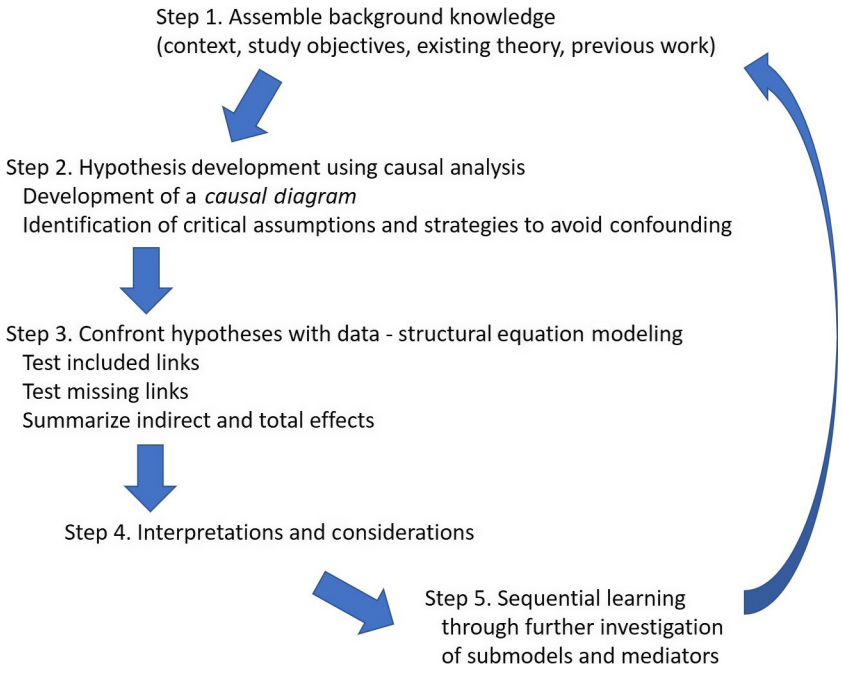

FIG. 2. Flow chart showing major steps in the process of explanatory modeling.

TABLE 3. Ranking of candidate models based on AIC_c.

Model	K	AIC_c	ΔAIC_c	AIC_cWt	Cum.Wt	LL	Predictors
m12	5	31.41	0.00	0.39	0.39	−10.35	F + A + E
m16	6	33.51	2.10	0.14	0.52	−10.25	F + A + E + C
m6	4	33.83	2.42	0.12	0.64	−12.68	F + A
m7	4	33.96	2.54	0.11	0.75	−12.74	F + E
m2	3	34.94	3.52	0.07	0.81	−14.33	F
m13	5	34.95	3.54	0.07	0.88	−12.12	F + A + C
m8	4	35.32	3.90	0.06	0.94	−13.42	F + C
m14	5	36.07	4.66	0.04	0.97	−12.68	F + E + C
m9	4	37.70	6.28	0.02	0.99	−14.61	A + E
m15	5	39.90	8.49	0.01	1.00	−14.59	A + E + C
m10	4	42.02	10.61	0.00	1.00	−16.78	A + C
m3	2	42.23	10.82	0.00	1.00	−17.98	A
m5	3	48.52	17.11	0.00	1.00	−21.12	C
m4	3	49.66	18.25	0.00	1.00	−21.69	E
m11	4	49.97	18.56	0.00	1.00	−20.75	E + C
m1	2	51.88	20.47	0.00	1.00	−23.87	Intercept only

Notes: Also presented are K = number of parameters in the model, including intercept and error variance (thus, K = number of predictors + 2); AIC_c = sample-size adjusted Akaike information criterion; ΔAIC_c = numeric difference between the AIC_c for a model and the minimum value for the model set; AIC_cWt = probability that an individual model is the best in the set of candidates; Cum.Wt = cumulative value of AIC_cWt values; LL = log likelihood for a model. Predictor abbreviations: F, fire severity; A, age of stand; E, elevation, C, distance from coast.

representing hypothesized relationships among a set of variables using probabilistic causal networks. There are two essential components of causal networks. One is the *causal diagram*, which allows us to convey the logic of our hypotheses, and the second is an appropriate equational framework for estimating network relationships.

Understanding the structure of causal diagrams.—A causal diagram is a pictorial graph of cause–effect relationships that is used to represent scientists' ideas about how a system works (Robins 1986, Pearl and Verma 1991, Greenland et al. 1999). The diagram is meant to represent a translation of the scientist's ideas and hypotheses

Table 4. Four key principles for causal analysis.

Principle	Description
1	Causal networks provide a powerful and convenient interpretive structure for causal analysis.
2	Many elements of network structure can be tested with appropriate data, such as both omitted and included links.
3	Confounding due to model misspecifications can bias parameter estimates. Analysis of causal diagrams can provide strategies for addressing this issue.
4	The inclusion of mediators can encode testable hypotheses about mechanisms and strengthen inferences.

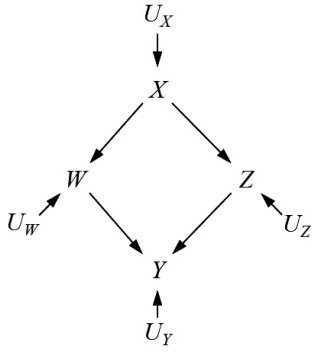

Fig. 3. Causal diagram consisting of four observable variables, X, Y, Z, and W connected by four directed arrows representing cause–effect relationships. U variables represent unspecified, unobserved causes of variation. By convention, models and causal diagrams are distinguishable by the fact that in causal diagrams, variables are not outlined as they are in models (Fig. 1).

about the data-generating mechanisms into a set of observational expectations.

Consider a simple example involving four random variables, X, Y, Z, and W (Fig. 3). Also shown in Fig. 3 are four unspecified causes of variation (U variables), which create the random component of the variables. Causal diagrams are based in a particular graph form, referred to as a *directed acyclic graph—directed* because all arrows are unidirectional, and *acyclic* because it contains no causal loops. Variables directly connected by a link are *adjacent* and those not directly connected are *nonadjacent*. Pathways can be traced through a series of links either following the flow of causation (*directed pathways*) or ignoring directionality (*undirected pathways*). Directed pathways passing through two or more links represent *indirect effects* in the model, whereas those conveyed by a single link constitute *direct effects* in the model. To say that W is a direct cause of Y in causal model M means "a change in W will induce a change in Y even if we hold constant all other variables in M except for W and Y." To say that X is an indirect cause of Y in causal model M through W means a change in X will induce a change in Y if we hold constant all other variables in M except for X and Y and those variables along the directed path (causal chain) between X and Y being discussed (W in this case). Importantly, variables and links omitted from a causal diagram also represent a set of explicit assumptions with empirical implications.

There are several definitional features of causal diagrams. First, directed arrows imply cause–effect relationships. This means that if we physically change the values of the variable at the base of an arrow (e.g., W in Fig. 3), while holding constant all other predictors, it can alter the values of the variable at the tip of the arrow (e.g., Y in Fig. 3). There are two corollaries that go with this definition of arrows. One is that there is understood to be a finite passage of time between the cause and the response. Thus, the arrows represent movement of information from some time in the past to present. Another corollary is that we can force changes to the quantity at the tip of the arrow (e.g., Y) without influencing the quantity at the base of the arrow (W) (also referred to as the assumption of *asymmetry*). This means that changing present conditions should not change past conditions. Thus, arrows represent implied experimental manipulations and routinely we use thought experiments to analyze the logical relations in causal diagrams. When we can be more rigorous, we use physical experiments to test our assumptions (Gough and Grace 1999). Note that in regression models (Fig. 1A), the arrows simply represent computations—if we see a particular value of X_1, we expect to see a particular value of Y. This conceptual distinction between the predictive interpretation of regression and causal relationships is sometimes described as the difference between "seeing" (an association) and "doing" (hypothesizing responses to manipulations); Lindley (2002).

A second attribute of directed relations in causal diagrams is that they are *transitive*, meaning that if X causes W and W causes Y, then X is a cause of Y. There is sufficient confusion about this point (particularly when people start thinking in terms of "true" causes) that we offer a memory aid for the transitive property, "relative to some response, a cause of a cause is a cause." This phrase reminds us that distal causes are just as axiomatic as proximal ones. This idea will help us clarify causal logic when we return to the wildfire example later in the paper.

A third attribute of causal diagrams is known as the *Markov condition* (Hitchcock 2019). This means that we only need to know proximate causes to compute the expected values for a response variable. Relative to Fig. 3, if the diagram is a true reflection of the data-generating mechanism, we can compute the expected values for Y from the values of W and Z. Any influences from X on Y can be seen to be conveyed through indirect effects; therefore, we can say that X is conditionally ignorable given W and Z.

Finally, causal diagrams are nonparametric in the sense that no specific statistical assumptions about the type of responses, linkage functions, or error

distributions are implied. As a result, the interpretation of a causal diagram is independent of statistical details.

Understanding how associations among variables are generated.—To understand the logic of causal diagrams, we need to understand the various ways that associations between variables can be created. If the causal diagram in Fig. 3 is true, a fundamental expectation is that all adjacent variables will show conditional associations. Considering the nonadjacent pairs of variables, there are two primary ways associations can be created indirectly: (1) through causal chains and (2) through common causes. (There are actually other ways that associations among variables can accidentally be created, which we will describe later in the paper.) Causal chains (e.g., $X \rightarrow W \rightarrow Y$) produce correlations between nonadjacent variables (X and Y in this case) through indirect, compound pathways. The expected value for a bivariate association between X and Y simply as a result of the three-variable chain is the product of the strengths of effects along the chain. For more complex relations, as in Fig. 3, where there are two causal chains connecting X to Y in the diagram ($X \rightarrow W \rightarrow Y$ and $X \rightarrow Z \rightarrow Y$), we expect the total bivariate association between X and Y to be the sum of the indirect effects. Nonadjacent variables connected by a common cause, for example, $W \leftarrow X \rightarrow Z$, will also be expected to exhibit a bivariate association. Note that for this type of pathway there is an implied historical effect of X on W and Z. The expected value for the bivariate association between W and Z is also the product of the strengths of effects along the connecting pathway.

It is worth pointing out that there are special situations where variables are inherently nonindependent quantities. For example, if W is the proportion of a plant community biomass made up of legumes and Z is the proportion made up of grasses, then both variables are derived quantities with a common denominator, total community biomass. The consequence is that W and Z will be inherently nonindependent and be associated even in a null model (where all links are nonsignificant). This is a fundamental problem that can contribute computational association to any pair of variables, including adjacent ones. Adjustments for this fundamental issue in data analysis are possible (Pawlowsky-Glahn and Buccianti 2011), but beyond the scope of the present discussion. For another example, it is possible for two sets of variables to be correlated because they follow a common developmental program. Again, this special case is beyond the scope of our presentation.

Network equations: mathematical representation of causal networks.—Generally, scientists represent information about systems using networks (ecosystem models, food webs, metabolic models) and those who apply science for engineering and decision making rely on information networks. Statistical models, in contrast, have traditionally been reductionist, adopting the simplest representation possible in order to isolate a single effect of interest. One might argue that the most notable separation between statistical modeling and scientific modeling is reflected in the difference between the equational form for regression vs. causal networks.

This dichotomy can be traced back to competing world views established by Ronald Fisher and Sewall Wright in the 1920s. Fisher, whose dominant influence over the development of statistics remains to this day, was the father of the regression equation (Aldrich 2005). There are many types of regression models, but they all have the same general weaknesses with regard to explanatory interpretation. Regression models are based on the fundamental equational form

$$\mathbf{Y} = f(\mathbf{X}) \tag{3}$$

where $\mathbf{Y}$ is a vector of response variables (or single response variable), $\mathbf{X}$ is a vector of predictors, and f is one of many functional forms, ranging from General Linear Models to Generalized Linear Mixed Models. The limiting features of this equation were discussed above in relation to Figure 1.

Causal networks are based on a fundamental equational form developed by Wright (1921) that can be seen as an extension of the regression equation

$$\mathbf{Y} = f(\mathbf{X}, \mathbf{Y}). \tag{4}$$

here, $\mathbf{Y}$ is a vector of the response variables embedded within a network of relationships, $\mathbf{X}$ is a vector of exogenous variables that define the boundaries for our model, and f is a set of functional forms, again with no inherent limit as to type. Importantly, it is the network of equations that permits and requires a priori scientific knowledge.

Principle 2: Many elements of network structure can be tested with appropriate data, including both omitted and assumed links

Although adopting a network structure for our hypotheses is an essential step, it is important to point out that many aspects of hypothesized networks can be tested, given appropriate data. This testing can lead to the discovery of previously unsuspected mechanisms and the specification of new models that require additional testing and verification. Although we will illustrate this process below, it is important to make the point that the enterprise described in this paper is not part of so-called assumption-based modeling. Many network-style models that ecologists might encounter are deductive expressions of accumulated assumptions (see, for example, the journal *Ecological Modelling*). The same description can be applied to most theoretical models. Explanatory statistical models, however, provide methods for ruling out a large number of possibilities using the principles described next. Omitted links from hypothesized models represent strong assumptions that create testable

implications for data. Included links, which only claim nonignorability, represent less strong, but nonetheless testable, premises. The directionality of arrows (i.e., of causal relationships), however, is one type of assumption that requires theoretical justification and is not testable from observational data alone (though can be tested through manipulative experiments).

Principle 3: Confounding because of model misspecifications can bias parameter estimates. Analysis of causal diagrams can provide strategies for addressing this issue

Much of the methodological work on causal inference has dealt with ways of addressing potential impacts from model misspecification (models that do not match the true data-generating process). Until fairly recently, very little work focused on a formal analysis of the problem. Rules for recognizing potential sources of bias and devising remedies using causal diagrams now exist. These rules have been summarized in axiomatic form using a principle called *d-separation* (Pearl 1988).

The formal definition of d-separation gives the requirements for two nonadjacent variables embedded in a graph (e.g., W and Z in Fig. 3) to be conditionally independent. Conditional independence is manifested through the absence of residual association, which means that if we were to include a directed link or error correlation between W and Z, analyses with an appropriate data set would find the link to be unsupported. To break d-separation down, the first two (of the three) rules of d-separation tell us what we need to do to identify (permit estimation of) causal effects properly. The third d-separation rule relates to things to *not* do to avoid inadvertently creating false signals.

The elements of the d-separation criterion include the two diagram structures that generate associations between nonadjacent variables mentioned earlier: (1) causal chains and (2) common causes. It also addresses a third diagram structure, referred to as collider relationships. A collider variable is one receiving two or more incoming arrows. In Fig. 3, $W \rightarrow Y \leftarrow Z$ is our only collider pathway, with Y being the collider variable. It is characteristic that tracing pathways through colliders involves a reversal in causal direction. The implications of collider relationships can be understood by recognizing that the collider Y is a descendent of its parent variables W and Z.

Conditioning is a key concept for understanding causal analysis generally and d-separation specifically. This word describes a number of statistical operations, so its use can be a source of confusion. The variety of ways to "condition on a variable" involve (1) including the variable in a model, (2) preconditioning (residualize) the variables of prime interest in order to remove the influences of variables that are to be omitted from the model, (3) stratifying the data by levels of the variable, (4) sampling from a population using the variable in the sampling scheme, and (5) through various roles the variable might play in censoring or truncating the data or the sample. The rules that follow are meant to apply to all these different situations.

d-separation rule 1.—Given a causal chain, $X \rightarrow W \rightarrow Y$, *complete or full mediation* means once we regress Y on W, X provides no capacity to explain additional variation in Y (i.e., the effect of X on Y is explained through W). Therefore, Y is independent ($\perp$) of X, given that we have conditioned Y on W ($|W$).

$$Y \perp X | W \tag{5}$$

In d-separation parlance, X and Y are said to be d-separated when Y is conditioned on W.

d-separation rule 2.—Given a common-cause pathway, such as $W \leftarrow X \rightarrow Z$, conditioning W and Z, the descendent variables, on the common cause variable X renders W and Z d-separated (conditionally independent):

$$W \perp Z | X \tag{6}$$

d-separation rule 3.—Considering an undirected path including a collider, such as $W \rightarrow Y \leftarrow$ Z, conditioning the ancestral variables (W and Z) on the collider (Y) can create an association between otherwise independent causes of the collider. Thus,

$$WZ | \{X, Y\} \tag{7}$$

where the symbol ♯ means nonindependent. To make this somewhat nonintuitive principle clear, a computational demonstration is given at the end of Appendix S4 for the interested reader.

Note that we would not normally condition on a collider if we were using a causal diagram to build our model because the direction of causality should be clear. However, there are multiple ways one might accidentally condition antecedent variables based on a collider in other contexts. One situation is in regression studies where analysts use preconditioning to remove confounding effects from some relationship of interest (for example, a treatment-response study where there is adjustment for unequal treatment effectiveness). Another situation is where data collection is restricted by an outcome, such as looking for associations among causes of death using a sample of dead animals (in this case, being dead, and therefore part of the sample, is a descendent of the causes of death). There are legions of examples where inadvertently conditioning on a collider has introduced bias into analyses (Elwert and Winship 2014, Pearl and Mackenzie 2018). Awareness of this issue is one reason the use of causal diagrams is spreading into fields that have historically relied on regression to address treatment-response questions, such as epidemiology and econometrics.

For more complicated diagrams than examined here, the d-separation criterion goes a step further and describes how to find the minimum sets of conditioning variables to achieve d-separation. The implementation of d-separation was first described in Shipley (2000*b*). Several software packages are now available for working through all the possibilities prior to fitting a model with data (Textor et al. 2011, Marchetti et al. 2015). Routines also exist in some SEM software (Lefcheck et al. 2018). Both Shipley (2016) and Kline (2016) provide thorough discussions of conditioning sets based on d-separation.

Principle 4: The inclusion of mediation relationships, in the form of causal chains, encodes testable mechanistic explanations

One of the most common scientific applications of a network structure is to permit formal evaluation of mediation hypotheses. By permitting the specification of a mediation relationship in a causal chain, it is possible to represent mechanistic explanations within models. For example, the diagram in Fig. 3 encodes the hypothesis that X affects Y through two mediators, W and Z. As we shall see, once we transition to a fully specified statistical model and data are used for estimation (Step 3 in Fig. 2), tests of conditional independence allow us to discover that the specified causal chain is either clearly supported (all links are deemed biologically meaningful), unsupported (no evidence for some links), or incomplete (there is evidence that additional links are needed). When unsupported, we may conclude that there is no empirical evidence for the proposed mediation mechanism. When incomplete, we conclude that there is some additional mechanism whereby X influences Y that is *not* through W or Z. This latter finding represents the discovery of a new mechanism that was unsuspected and only revealed via tests of mediation hypotheses. Many other surprises can also be revealed through the use of causal networks, a few of which we illustrate below in the second example presented.

Confronting Hypotheses with Data—Structural Equation Modeling

Our primary objective in this paper is to show scientists how to "think hard" about their hypotheses to promote explanatory analyses (Fig. 2, Steps 1 and 2). Our four principles of causal analysis should assist in representing causal hypotheses and establishing their empirical claims. Now, we need to say something about methods for confronting those hypotheses with data so we can complete the illustration through the use of examples (Fig. 2, Steps 3 and 4). No longer are we dealing with diagrams, but instead, fully specified models. We use the general term *explanatory modeling* in this paper to describe the intention. For the context in which we are working, the statistical methodology of structural equation modeling (SEM) provides a well-established framework for estimation, evaluation, and summarizing findings.

There are many modern treatments of SEM (Kline 2016), some directed at natural scientists (Grace et al. 2015, Shipley 2016). It is beyond our purpose in this paper to provide an in-depth introduction. Rather, we offer a high-level view for scientists not already familiar with the methodology, as well as executable code for the examples in this paper in the Appendices. A survey of what ecologists find helpful about SEM can be found in Laughlin and Grace (2019).

Perhaps the most important point to make about SEM is that it is a scientific framework for explanatory modeling rather than a specific statistical technique. There are no statistical assumptions that are inherent to SEM; those depend on the implementations supported by particular software programs. Thus, SEM is perfectly compatible with the nonparametric description of Pearl's (2009) *structural causal model*, the primary source for modern causal analysis. Related to that, the methodology continuously evolves to incorporate procedures supportive of explanatory modeling. This paper is an illustration of that point through our demonstration of how modern causal analysis integrates with SEM (Fig. 2). Specialized regression procedures that are sometimes presented as alternatives to SEM, such as instrumental variables, simultaneous equations, two-stage least squares, and hierarchical Bayesian models are all implementable within the SEM framework. This potential can be understood by reference back to the form of the equations (Eq. 4), which allow the full suite of regression-type statistical models as elements in an SEM model. Another distinct attribute of SEM is that it permits the evaluation of integrative system-level hypotheses, as opposed to reductionist ones. It is certainly true that more complex models pose more risks for violations of assumptions. Nonetheless, the evaluation of causal-network-style hypotheses can yield more scientific insights and more complete explanations when applied successfully.

Example 1: A return to the wildfire recovery example

Our explanatory modeling work-flow process requires explicit consideration of expert knowledge in order to construct plausible hypotheses to convey in a causal diagram. Following the data collection and initial examinations of relationships conducted by Keeley et al. (2005), Grace and Keeley (2006) compiled a carefully considered list of biological assumptions relevant to the measured variables (Table 5). This table is an example of Step 1 in Fig. 2 and we feel a useful companion to the causal diagram. In this demonstration, we first considered a naïve causal diagram (Fig. 4A) that considered all possible links (denoted by the corresponding numbers in Table 5). We used our naïve causal diagram (Fig. 4A) and assembled expert knowledge (Table 5) to arrive at an informed diagram (Fig. 4B). In this example, we start

Table 5. Expert opinion relevant to an initial hypothesis of how vegetation cover following wildfire could be explained by the measured predictors.

Number	Interpretation
1	In southern California, where the wildfires took place, elevation generally increases as one moves eastward from the coastal lowlands to the interior highlands (see Keeley et al. 2005, Fig. 1). Thus, increasing the distance from the coast tends to cause an increase in elevation (expected effect: positive).
2	Increasing elevation should produce more mesic conditions, which in turn, would reduce the frequency of wildfires and thereby increase the average ages of forest stands (because age is determined by time since last stand-replacing fire). The net relationship expected is thus increased age with increasing elevation (expected effect: positive).
3	As stands age, they accumulate more biomass (alive and dead) that can serve as fuel during a wildfire. Therefore, increasing stand age should lead to more severe fires when burning finally takes place (expected effect: positive).
4	Independent from other effects, moving away from the coast should result in less wildfire suppression and larger fires because of reduced urbanization and population densities (expected effect: negative).
5	Fire severity depends not only on fuel, but also humidity. Thus, increasing elevation should contribute to less severe fires, all other things equal (expected effect: negative**).**
6	Once the effects of stand age and elevation are taken into account, it is not obvious, based on a priori information, that additional processes related to distance from the coast should have a major influence on wildfire severity (expected effect: undetectable).
7	Once effects of elevation, stand age, and fire severity are controlled, it is not obvious, based on a priori information, that vegetation recovery will be influenced by distance from the coast (expected effect: undetectable)
8	In the study region, a marked, orographic gradient leads to a strong positive effect of elevation on precipitation, which would lead to faster plant regrowth and a more rapid recovery of the vegetation (expected effect: positive).
9	A negative effect of stand age on vegetation recovery might be observed because of the fact that increasing overstory dominance in older stands will lead to a reduced understory. If the understory (including the seed bank) is a strong contributor to vegetation recovery, an independent effect of stand age on vegetation recovery might be expected (expected effect: negative).
10	There is a very strong a priori expectation that increased fire severity will result in reduced vegetation because of the damage to the regenerating tissues of plants and possibly effects on soil that reduce water penetration (expected effect: negative).

with a single hypothesis for consideration, which is derived from our summary of expert knowledge. We do this knowing that we will have the opportunity to compare the support for this hypothesis against all the alternative hypotheses based on the same causal ordering of predictors.

The backbone of the causal diagram is a directed chain relating vegetation recovery (V) to fire severity (F) to stand age (A) to elevation (E) to distance from the coast (C). This sequence of variables represents an ordering from proximal to distal potential drivers of vegetation response following fire. In an earlier review of this paper, we were asked to explain how it can be argued that the various predictors in this example actually constitute causes. The rule related to this point is very basic and can be called the *intervention-response rule*: if we can manipulate something and induce a response in another system property, then the thing manipulated qualifies as a cause of the thing that responds. Confusion comes about when someone starts wanting to distinguish what they think of as "true" causes. Let us consider the most distal cause in the chain, distance from the coast. Intuitively, it might seem that this is a placeholder for some "actual" cause. A simple statement shows that location qualifies as a causal variable. That statement is, "If you think location in space is not an actual cause, then stand closer to the fire and see if your opinion changes." In reality, there are many potential mediators that make up a causal chain. We usually work with mediators that are meaningful for our explanations and thereby avoid the problem of infinite regress in explanation.

The causal diagram in Fig. 4B was translated into a structural equation model using the software package piecewiseSEM (Lefcheck et al. 2018). As shown in Appendix S3, Data S3, estimation produces a set of d-separation tests, one for each omitted link in the model. These tests evaluate whether there is evidence to include a missing link, and thus, whether it should be added to the model. Global fit of the initial model suggested it was a plausible explanation for the data. Further, tests suggested equivocal support for some of the links, however, so simpler models were also considered. During the evaluation process, individual d-separation tests suggested the possibility of local violations of conditional independence. We ultimately considered four alternative models, omitting and adding various links. The model selected from that process is shown in Fig. 4C. The scientific conclusion we draw from the results is that vegetation recovery varies widely in the landscape, primarily as a function of fire severity, which in turn is influenced by greater fuel accumulation in older stands of woody plants. Distance from the coast (dismissed as important by all-subsets regression) is shown to influence recovery, but only indirectly through effects on both stand age and fire severity, ultimately being quite important in the system. The explanation achieved is based on careful and explicit considerations, something we could not easily achieve using an all-subsets regression approach.

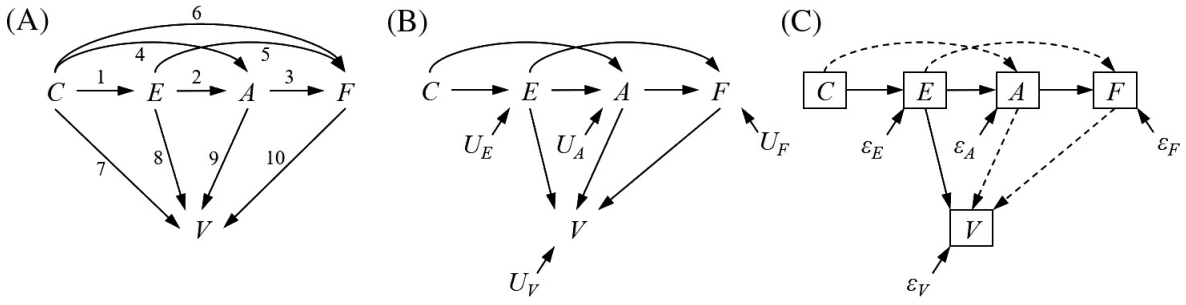

FIG. 4. (A) Naive causal diagram with all possible links. (B) Hypothesis based on available expert opinion (Table 5). (C) Model informed by data and d-separation tests (Appendix S3). Dashed lines represent negative effects, solid lines positive effects.

Example 2: In situ experimental study of a marine food web

Models developed using experimental data are also often evaluated using model comparisons, and such data allow us to illustrate the utility of the principles in this paper further. For this example, we draw upon a study of food webs in marine seagrass beds (Whalen et al. 2013). In this system, the dominant herbivores are small invertebrates (specifically, microcrustaceans), which graze on the epiphytic algae that grow on seagrasses while also being important prey for fish. A global decline in seagrass beds has raised concern that reduced populations of larger fish (due to commercial harvest) is resulting in an increase in small fish and subsequent reduction in microcrustaceans (a trophic cascade). The microcrustaceans have been hypothesized to serve in a protective role for seagrasses by grazing on the epiphytic algae that grow on seagrass leaves and that impair plant photosynthesis and survival. Convincing field experimental demonstration of this top-down process has been lacking. In this study, the authors examined the responses of microcrustaceans and their predominant food supply, epiphytic algae, to treatment with slow-release insecticide in seagrass beds. Experimental plots were established by sinking plastic poles into the sediment and suspending insecticide-impregnated blocks, or insecticide-free blocks, within the seagrass canopy 20 cm above the sediment surface. Forty experimental plots were located randomly within the study area and were randomly assigned to receive insecticide or not. Sampling of microcrustaceans and epiphytes were conducted at the end of the experiment. Microcrustaceans were divided by taxa and abundance was estimated. Epiphyte abundance was also measured.

Given the three response variables (epiphyte biomass, Gammarid crustaceans, and Caprellid crustaceans) and two covariates (macroalgae and seagrass density) measured, there are a variety of different analyses that an investigator might perform. First to come to mind is the suite of options that include ANOVA, MANOVA, ANCOVA, and MANCOVA. We present causal diagrams for these classic models in Fig. 5. The primary differences from our earlier example to keep in mind are (1) treatment was randomly assigned and (2) the treatment "Trt" is a binary (0/1) variable. As with regression, we are generally limited in ANOVA, ANCOVA, and MANOVA by the equational form of the model, $Y = f(X)$, where the X vector includes the treatment variable and any variables included as covariates. In this case, Y can be a single response variable or any subset of the three responses.

Let us consider some possible rationale for the options shown in Fig. 5. Diagram A is the classic ANOVA. Because of randomization, we might reason that covariates like macroalgae (Mac) and seagrass density (Gra), should be independent from the treatment assignment; thus there is no reason one must include the covariates in the diagram, as the effect of treatment should be the same regardless. Comparison to results from the ANCOVA (Diagram E) is expected to confirm this assumption, though one should always check, as random assignment does not guarantee comparable control and test groups. Diagram B could be justified because microcrustaceans (Gammarids and Caprellids) are a distinctly different type of response from epiphytes. We might, for example, see one or both microcrustacean groups respond, but no response from epiphytes. For this reason, we might want to perform separate analyses. Diagram C represents a classic MANOVA with all three responses treated as a single response. For this particular study, it might seem more logical to interpret results from Diagrams A and B instead of C because the response types are clearly different.

Diagram D represents an interesting possibility. Because epiphytes constitute the food supply for microcrustaceans, then an investigator might use epiphytes as a covariate to adjust for differential food supplies among plots. They could either perform an ANCOVA individually for the Gammarids and Caprellids, or look at their joint response as depicted in Diagram D. There are merits and demerits to either approach. Diagrams E, F, and G represent different versions of ANCOVA with epiphytes as the focal response. Diagram E is a standard ANCOVA, including the two covariates abundance of macroalgae (Mac) and density of the seagrass in a plot (Gra). In Diagram F, the logic shifts from that used in Diagram E. Because the treatment method (slow-release insecticide) cannot kill 100% of the microcrustaceans in the open plots, experimental control is incomplete in this

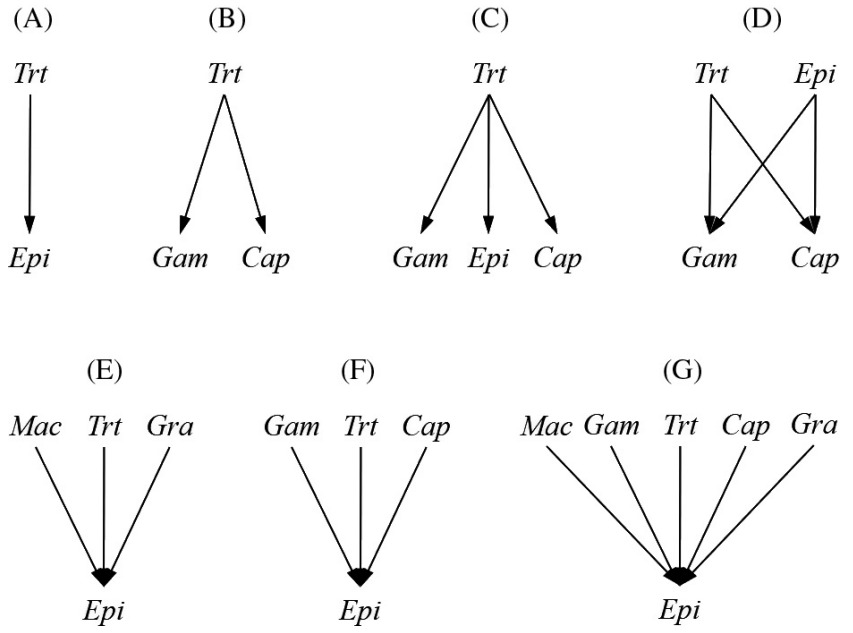

FIG. 5. Causal diagrams representing (A) ANOVA 1, (B) ANOVA 2, (C) MANOVA 1, (D) MANCOVA 1, (E) ANCOVA 1, (F) ANCOVA 2, (G) ANCOVA 3. *Trt* = treatment, *Epi* = epiphyte abundance, *Gam* = Gammarid abundance, *Cap* = Caprellid abundance, *Mac* = macroalgae abundance, and *Gra* = seagrass density. Results for these models can be found in Appendix S4.

study. Controlling the variable responses of microcrustacean by including these variables as covariates might seem to be a useful way to remove their variations from the assessment of treatment effects on epiphytes. Diagram G represents a commonly employed approach, which is to control for as many possible influences as possible. This cursory discussion of possibilities is meant to point out that causal hypotheses will have to be brought to bear in order to decide which of these models will produce defensible results.

It is instructive to compare the hypotheses that can be examined using classical statistical models (Fig. 5) to the possibilities that emerge from adopting a causal network perspective. For this comparison, we refer the reader to a causal diagram representing a dual mediation hypothesis in Fig. 6A. Here, impacts of treatment on Gammarids and Caprellids, the two dominant groups of microcrustaceans, are hypothesized to explain the effect of treatment on epiphytes. This model also includes macroalgae and seagrass density as covariates potentially influencing epiphytes. This diagram was treated as the initial hypothesis, which was subsequently evaluated using the available data. The structural equation model presented in Fig. 6B summarizes results for the final model once d-separation tests were performed and links were added to resolve d-separation violations (Appendix S4, Data S4).

There are numerous, interesting scientific findings revealed by the SEM (Fig. 6B; see also Whalen et al. 2013). First, we are able to evaluate the full-mediation hypothesis formally. The two mediation pathways (those combining links 1 → 3 and 2 → 4) were found to explain the response of epiphytes to treatment completely. As a result, there is no link directly from treatment to epiphytes in the final model. Finding support for full mediation is a highly desirable outcome in an experiment, because we are not scientifically interested in the effects of insecticide and prefer a model where artificial treatments are conditionally ignorable. Second, empirical evaluation of the implied conditional independences from the initial hypothesis (Fig. 6A) revealed three nonindependences that could be resolved by adding links (Fig. 6B, added links shown with asterisks). This produced evidence to suggest previously unanticipated biological discoveries related to the system under study.

The positive effects discovered by the analysis (links 7, 8, and 9) can be interpreted as indications that macroalgae (to a large degree) and eelgrass density (to a lesser degree) provide protective refuges for microcrustaceans. By promoting microcrustaceans, macroalgae have indirect negative effects on epiphytes (through the pathways 7 → 3 and 8 → 4). Within the total food web, this could translate into macroalgae facilitating seagrasses by harboring microcrustaceans, the grazers of epiphytes (which are the enemies of seagrasses). We believe scientists will find this system-level set of results substantially more informative than results from traditional ANCOVA.

Interpretations and Considerations

Explanatory modeling requires adequate expert knowledge to defend scientific interpretations. Appropriate data are also required and frequently limit the

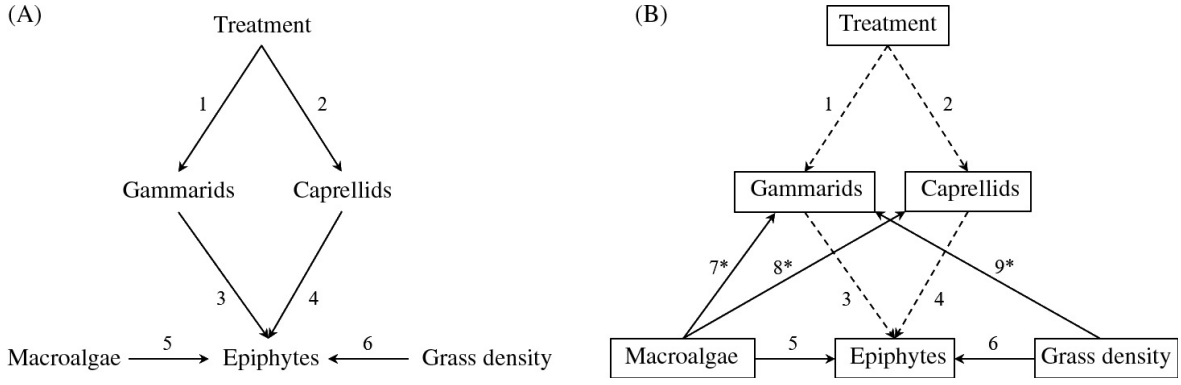

FIG. 6. (A) Causal diagram representing initial hypothesis and (B) final structural model supported by the data (see Appendix S4). Solid lines in (B) represent effects of positive sign and dashed lines represent effects of negative sign. Linkages in B not seen in A (7, 8, and 9; indicated with asterisks) represent important effects uncovered because of failed d-separation (conditional independence) tests.

conclusions that can be drawn. There are many ways our models and interpretations can deviate from the truth, to either major or minor degrees. Here we list three increasingly challenging types of assumptions upon which interpretations depend. (1) Foundational assumptions—arrows in models indeed represent directional cause–effect relationships. Generally, this must be supported by a priori expert knowledge of mechanisms. (2) Parameter estimates approximate true values to the degree that overall conclusions are not confounded. Again, expert knowledge of the system under investigation needs to be sufficient to defend general conclusions. Further, it is important that investigators be aware of the ways that confounding can occur and the need to guard against the omission of critically important common causes. (3) Inferring that one or more parameter estimates are unbiased (true) estimates of causal effects constitutes a stricter assumption. Both omitted confounders and measurement error, along with a host of more technical issues, can bias estimates to various degrees (though remedies to even these challenges exist; Bollen 2019). For these reasons, appropriate caution is required when drawing conclusions.

Suitably cautious language for expressing findings will vary depending on the above-mentioned factors. As one illustration, we present the language Grace and Keeley (2006) used when reporting their findings from the wildfire recovery study.

> We infer from the SEM results that postfire richness in this system is strongly influenced by local conditions and that these conditions are, in turn, predictably related to landscape-level conditions. For example, we observed that older stands of shrubs were characterized by more severe fires, which were associated with a low recovery of plant cover and low richness. These results may have implications for the use of prescribed fire in this system if these findings extrapolate to prescribed burns as we would expect.

Subsequent SEM studies (Keeley et al. 2008) have enhanced our confidence in the general inferences drawn from the original study. That said, we would not claim that all our parameter values are unbiased causal estimates without further evidence to support such inferences.

Conclusions and Future Directions

Causal understanding is ultimately about understanding mechanisms. The majority of studies that scientists conduct are directed to that end. Classical statistical models do not easily accommodate the explicit incorporation of mechanisms into hypotheses, limiting their capacity for explanatory application. Adopting a causal network framework and the principles of causal analysis for hypothesis development and evaluation greatly increases possibilities for the development of explanatory models and clear expression of their logic. We hope this presentation provides a guide toward that future by helping scientists see their own responsibilities and opportunities in quantitative analysis.

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Supporting Information

Additional supporting information may be found in the online version of this article at http://onlinelibrary.wiley.com/doi/10.1002/ecy.2962/suppinfo