

## ARTICLE

# Predictive models are indeed useful for causal inference

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**Funding information**

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**Handling Editor:** Chloé R. Nater

**Abstract**

The subject of investigating causation in ecology has been widely discussed in recent years, especially by advocates of a structural causal model (SCM) approach. Some of these advocates have criticized the use of predictive models and model selection for drawing inferences about causation. We argue that the comparison of model-based predictions with observations is a key step in hypothetico-deductive (H-D) science and remains a valid approach for assessing causation. We draw a distinction between two approaches to inference based on predictive modeling. The first approach is not guided by causal hypotheses and focuses on the relationship between a (typically) single response variable and a potentially large number of covariates. We agree that this approach does not yield useful inferences about causation and is primarily useful for hypothesis generation. The second approach follows a H-D framework and is guided by specific hypotheses about causal relationships. We believe that this has been, and continues to be, a useful approach to causal inference. Here, we first define different kinds of causation, arguing that a “probability-raisers-of-processes” definition is especially appropriate for many ecological systems. We outline different scientific “designs” for generating the observations used to investigate causation. We briefly outline some relevant components of the SCM and H-D approaches to investigating causation, emphasizing a H-D approach that focuses on modeling causal effects on vital rate (e.g., rates of survival, recruitment, local extinction, colonization) parameters underlying system dynamics. We consider criticisms of predictive modeling leveled by some SCM proponents and provide two example analyses of ecological systems that use predictive modeling and avoid these criticisms. We conclude that predictive models have been, and can continue to be, useful for providing inferences about causation.

**KEYWORDS**

causality, directed acyclic graphs, predictive models, strength of inference, study design

“Hume saw clearly that certain concepts, for example that of causality, cannot be deduced from our perceptions of experience by logical methods...”—A. Einstein

(Isaacson, 2007)

**INTRODUCTION**

Throughout history, science has proven to be the most successful process by which humans have learned how the biological/physical world works, with much of that

learning focused on understanding cause–effect relationships. A key step in the scientific process entails the comparison of model-based predictions against observations as a primary means of testing single hypotheses and discriminating among competing hypotheses. Recent literature includes several criticisms that ecologists have not used appropriate methods for studying causation, coupled with recommendations to follow a structural causal model (SCM) framework (Pearl, 2009), especially when dealing with observational data. SCM advocates frequently contrast their recommended approach with predictive modeling, claiming that the task of prediction is distinct from, rather than a component of, the process of investigating causation (e.g., Arif & MacNeil, 2022b; Laubach et al., 2021) and that “Predictive Models Aren’t for Causal Inference” (Arif & MacNeil, 2022b). Other SCM proponents have emphasized difficulties in meeting assumptions underlying the use of predictive models for assessing causation (Ferraro et al., 2019). Our purpose here is not to disparage the SCM framework, which has made especially important contributions in emphasizing specification of a priori hypotheses and in identifying and removing biasing effects of potential confounding variables. Instead, we respond to criticisms of predictive modeling and argue that it can be effectively used for causal analysis in ecology and elsewhere.

We conclude that the criticisms leveled at predictive modeling and model selection by some proponents of SCM are sometimes appropriate for exploratory uses of these former approaches. However, we argue that these criticisms do not apply generally to thoughtful uses of predictive modeling and model selection. In fact, ecologists have been conducting effective causal analysis based on these approaches for decades. Predictive modeling has been a key component of the scientific methodology responsible for much of what we currently know of ecology and remains an effective approach to inference about cause–effect relationships.

We consider definitions of predictive modeling and causation and then highlight difficulties in investigating causal relationships. We consider approaches to study design that vary in the strength of inference that they permit but are consistent in their use of predictive models. These sections of the paper present our perspectives on these topics as points of reference for the subsequent discussion. We do not contrast SCM versus other approaches, as we believe that these topics provide common ground for advocates of different approaches to inferring causation.

We briefly introduce the SCM framework and consider motivations for its development. We also introduce the general hypothetico-deductive (H-D) framework, which relies on model-based predictions. We outline a

specific H-D approach that we believe to be especially useful for investigating causation. We then consider criticisms of predictive modeling by some SCM proponents, arguing that they do not apply to thoughtful and informed uses of predictive models. We present two examples of studies that use predictive modeling and model selection to investigate causal relationships. Both studies deal effectively with potentially confounding variables that were identified, a topic of emphasis in SCM. We conclude that there are multiple viable approaches for investigating causal relationships in ecology and elsewhere and that predictive models play an essential role in at least some of these approaches.

## DEFINITIONS

We define a *predictive model* as a simplified and abstract representation of a system that can be used to quantify the probabilities associated with future system dynamics or responses. Some predictive models are based on correlations and associations that have no identified or even hypothesized causal basis. However, other predictive models are developed to represent hypothesized causal relationships, often based on mechanisms, and we argue that these can be very useful in investigating causation.

Formal definitions of *causation* specify antecedent conditions, consequent effects, and a rule of correspondence for their conjoint occurrence (Williams, 1997). *Necessary causation* specifies that a particular condition (putative cause) must be present for a specific effect to occur. The absence of the cause implies the absence of the effect, but the presence of the cause does not guarantee the presence of the effect (e.g., other conditions may be required as well). *Sufficient causation* specifies that a particular condition (cause) guarantees the presence of the effect. However, the presence of the effect does not ensure the presence of the cause. These definitions of both necessary and sufficient causation can be viewed as models that make clear predictions about the presence of putative causes and effects.

The above definitions of causation admit no exceptions; for example, sufficiency states that a condition guarantees the effect. When dealing with ecological systems, we often deal with putative causes that do not guarantee an effect, but instead increase the *probability* that the effect will occur (see Reichenbach, 2008, translation of 1915 thesis). Ecological effects are frequently defined as changes in the value of a state variable (a variable selected to characterize system condition such as population size, species presence, species richness), causing us to focus on the process parameters governing such change (e.g., rates of survival and reproduction; local

extinction; and colonization). For purposes of this paper, we define a *probabilistic causal factor* as one that increases the probability for a process governing an effect.

This definition is consistent with the “causes as probability-raisers-of-processes” perspective, in which “causation is rooted in the comparative probability of the connecting line to the effect with the cause versus without” (Schaffer, 2001). Under this view, necessary and sufficient causation are based not on the presence of the effect but instead on raising the probability that the effect will occur. This perspective is consistent with the usual situation in ecological systems of multiple complementary causes (e.g., Williams, 1997) and multiple hypotheses (Chamberlin, 1897). Inferences about multifactor causation focus on comparisons of model-based predictions about the existence of the causes with estimates of their effects on dynamic rate parameters. We will refer to the “causes as probability-raisers-of-processes” perspective as simply *probabilistic causation*.

Although our focus will be on investigating probabilistic causation using H-D science, we also note that a different definition of causation, *Granger causation*, is based on prediction (Granger, 1969). Specifically, a time series of variable  $x_t$  is concluded not to be “causal” of the time series of another variable  $y_t$ , if the history of  $x_t$  does not reduce the variance of prediction of the  $y_t$  series (Shojaie & Fox, 2022). Ecologists have borrowed methods from nonlinear dynamics to investigate Granger causation using time series data (e.g., Moniz et al., 2007; Sugihara et al., 2012). However, some of the assumptions that underlie this type of causation are relatively restrictive, and we currently believe that the “causes as probability-raisers-of-processes” perspective is likely to be most useful to ecologists.

## INVESTIGATING CAUSATION

Investigations of causation are plagued by several difficulties. An initial difficulty is best described via the framework of Rubin’s causal model, which is based on the idea of potential outcomes (Rubin, 1974; also see related counterfactual arguments of Lewis, 1973). For a single individual at any point in space and time, we would like to observe the outcomes of both applying, and not applying, a proposed causal factor. The “causal effect” would then be measured as the difference between the two outcomes. But we can either apply the factor or not, so one of the potential outcomes is always unobserved. This dilemma is relevant to probabilistic causation as well (Schaffer, 2001).

The natural response to our inability to observe both outcomes on any one individual is to rely on samples of

individuals, applying the causal factor to all individuals from one sample and not to those of another. The difference between average outcomes for these two groups would then provide an estimate of effect. However, effects can have multiple causes, and samples of individuals may differ with respect to one or more of them. Thus, differences in average outcomes can reflect the effects of both focal and non-focal causal factors. Non-focal causal factors pose a problem, because we can seldom identify all possible causes of an effect, or much less control, detect, or measure them during investigation. True randomized experiments (see below) provide a means of dealing with this issue, but virtually all other kinds of investigation are vulnerable.

Another difficulty is the asymmetric nature of evidence required for falsification versus confirmation (e.g., Popper, 1962). A single observation of an effect in the absence of a hypothesized necessary cause is adequate to falsify the causal hypothesis and eliminate it from further consideration. However, repeated observations of the effect always accompanied by the presence of the hypothesized cause cannot prove necessary causation, as a future observation can always be inconsistent with this hypothesis. Similarly for sufficient causation, repeated observations of the effect following the purported cause do not rule out the possibility that this sequence may not always occur.

This asymmetry of evidence does not occur with probabilistic causation, as a study either does or does not corroborate a hypothesis about a causal effect. If model selection, likelihood ratio tests, or tests of an effect coefficient do not support the effect, this does not necessarily remove the effect from further consideration. Inferences about probabilistic causation can be based on the accumulation of evidence from multiple studies (e.g., Hilborn & Mangel, 1997; Nelder, 1986; Nichols et al., 2019, 2021; Tredennick et al., 2021).

An additional difficulty associated with probabilistic causation entails observations. Many inferences about necessary and sufficient causation (and many SCM examples) are based on direct observation of the outcome when a causal factor is, or is not, applied. However, probabilities cannot be directly observed, but must instead be estimated, usually from multiple trials in which the presumed causal variable is applied to the focal variable or system. Such estimation produces a variance component that should be accounted for when investigating causation.

Collection of observations for investigations of causal relationships can occur in several different ways, dictated by study design. For all the above definitions of causation, different study designs provide evidence that supports different degrees of confidence, regardless of

whether analysis follows the SCM approach or a H-D approach using predictive models. The phrase *strength of inference* has been used in multiple ways, but here, we use it to refer to the general level of confidence that a study's results support the corroboration or falsification of a hypothesis. For this paper, the hypotheses focus on the existence of cause–effect relationships.

## Experiments

Randomized, controlled experiments are designed to deal with the difficulty of not being able to identify and thus control for all potential causes of a focal effect. They are sometimes termed manipulative experiments, because treatments are under the control of the investigator and can be assigned to experimental units. Such experiments permit some of the strongest possible inferences about causation and are distinguished from other study designs by their use of controls, replication, and randomization (e.g., Fisher, 1947; Hariton & Locascio, 2018; Williams et al., 2002). Controls refer to experimental units to which no purported causal “treatment” is applied, providing a baseline against which to quantify treatment effects.

Replication refers to the assignment of each treatment (and control) to multiple experimental units, providing information about variation expected within a treatment group. This information is then used to distinguish treatment effects from natural variation not associated with differing treatments.

Randomization refers to the random assignment of treatments and control to experimental units. Randomization offers protection against the first difficulty listed above: our inability to identify (or detect, control, or measure) all possible causes of any effect. Nonrandom assignment of treatments to experimental units admits the possibility of bias caused by experimental units assigned to different treatment groups also experiencing differences in unidentified causal factors (e.g., Fisher, 1947; Hariton & Locascio, 2018; Kimmel et al., 2021; Larsen et al., 2019; Williams et al., 2002).

## Constrained designs

Constrained designs represent efforts to draw inferences about causation from studies that focus on treatment effects but that lack at least one of the three design features that define an experiment. Various names have been attributed to constrained designs based on which features of an experiment are missing (e.g., Green, 1979; Skalski & Robson, 1992). Strength of inference is reduced

for constrained designs, when compared with experiments. But constrained design studies are widely used in ecology and conservation because experiments are extremely difficult, if not impossible, to conduct for many kinds of questions.

One example of a constrained design uses “treatments” that are applied by someone other than the investigator. For example, consider the hypothesis that high-voltage transmission lines cause various problems in humans, other animals, and plants (e.g., Balaji, 2015). A true experiment entailing the random selection of locations for installation of such lines, as well as for controls with no lines, would simply not be feasible or recommended in most instances. Thus, a constrained approach to studying this problem would be to select multiple sites with existing transmission lines (treatments), and multiple sites with similar habitats but without high-voltage lines (controls), and to measure hypothesized effects (e.g., plant growth) on these two sets of sites. Some SCM proponents refer to such constrained designs as “matching studies” or “propensity analyses” (e.g., Arif & MacNeil, 2022a). Such a study includes controls and replication, but the assignment of treatment (and control in some cases) to experimental units (sites within the landscape) was not under the control of the investigator and hence not carried out randomly. If such a study showed reduced plant growth in the sites with transmission lines, we would take this as evidence supporting the possibility of transmission line effects. However, we would recognize that some other factor(s) might have been responsible for the plant growth differences between the two groups of sites, despite our attempts to select similar control sample units.

## Observational studies

Many studies in ecology and conservation lack all three design features of an experiment (controls, randomization, replication) and are often referred to as observational (or “mensurative”) studies. For purposes of this paper, we specify that such studies are guided by a priori hypotheses in the sense that predictions are made and compared against observations. This specification distinguishes such studies from exploratory data analyses focused on pattern recognition and hypothesis generation. Observational studies can provide inferences about causation, although strength of inference is reduced relative to experimentation and frequently to constrained designs as well.

As an example of an observational study, we consider a 7-year capture–recapture study of European Dippers (*Cinclus cinclus*; Lebreton et al., 1992; Marzolin, 1988).



A major flood occurred during the study and was hypothesized to have negatively affected two years of dipper survival rates (possibly because of reduced access to the aquatic invertebrates on which dippers feed), leading to a model with two survival rate parameters, one for the flood years and another for the non-flood years. Model selection statistics and likelihood ratio tests led to the conclusion that the flood model was the most appropriate relative to the other three models under consideration (Lebreton et al., 1992). Estimated annual survival for the flood years (0.47) was smaller than that of the non-flood years (0.61), as predicted. These results thus supported the hypothesis that severe floods were a cause of the reduced survival. The absence of geographic replication (multiple study sites), a control site (a site with no flooding during the flood years), and random assignment of “treatments” (flood) all weakened the inference of causation, and factors other than the flood could have operated to reduce survival in the flood years.

We emphasize that the hypothesis of reduced survival during the two flood years guided the modeling and testing in the analyses of this study. This kind of effort differs from another common approach of gathering a list of available covariate data (e.g., multiple weather variables) and asking whether one or more is related to annual survival, without any specific predictions. We view this latter exploratory approach as perhaps useful for hypothesis generation, but not very useful for the scientific step of testing predictions against observations (see Rexstad et al., 1988; Romesburg, 1981; Williams et al., 2002). Many of the papers advocating causal analysis methods for ecologists compare the SCM approach to such exploratory analyses.

## SCM FRAMEWORK

Our focus in this paper is to suggest that use of predictive modeling (e.g., generalized linear models, GLMs) and related tools such as model selection can provide an effective means of investigating causation. Hence, we make no attempt to describe the SCM framework in any sort of detail (see Laubach et al., 2021; Pearl, 2009, 2010). Instead, we briefly discuss (1) the directed acyclic graphical (DAG) approach used by many for SCM and (2) the motivation for development of SCM, arguing that more traditional approaches can be used to achieve similar results.

### Directed acyclic graphs

Proponents of the SCM framework frequently recommend use of DAGs to specify and visualize the causal

structure of a modeled system (Arif & MacNeil, 2022a, 2022b; Cronin & Schoolmaster, 2018; Elwert, 2013; Ferraro et al., 2019; Grace & Irvine, 2020; Greenland et al., 1999; Laubach et al., 2021; Pearl, 2009, 2010). DAGs are qualitative graphical models consisting of nodes that represent variables and directed arrows between nodes that represent potential causal effects. Algorithms developed by proponents of SCM can be used in conjunction with DAGs to identify potential confounding variables that could produce misleading results in causal analyses and represent an important contribution of the SCM framework (e.g., Grace & Irvine, 2020; Greenland et al., 1999).

The “acyclic” term in DAG specifies the absence of (1) reciprocal causation in which variable A can produce an effect on variable B, and variable B can affect variable A as well, and (2) feedback loops in which a variable can affect itself (Elwert, 2013). However, Greenland et al. (1999) note that cyclic graphs (e.g., causal arrows going in both directions between two variables) can be transformed into acyclic graphs by incorporating time-specificity into the diagram. So,  $X_t$  can affect both  $Y_{t+1}$  and  $X_{t+1}$ , and  $Y_t$  can affect both  $X_{t+1}$  and  $Y_{t+1}$ , for example.

Importantly, DAGs are qualitative models used to illustrate investigator hypotheses about causation within a focal system. We view this emphasis of SCM on a priori hypotheses as very important, especially when they are based on mechanistic knowledge and hard thinking about how a system works (e.g., Grace, 2024; Grace & Irvine, 2020). Such thinking is not restricted to DAG construction. Instead, it characterizes the development of hypotheses in most of the good ecological research conducted over the last century (Nicholson, 1954) and more recently has been incorporated formally via the use of informed priors in a Bayesian inferential framework (e.g., Link & Barker, 2009).

### Motivation

A stated motivation for the development of SCM is a focus on system dynamics. Pearl (2010) describes standard statistical analyses as having a focus on association among variables. He contrasts standard approaches with SCM: “Causal analysis goes one step further; its aim is to infer not only the likelihood of events under static conditions, but also the dynamics of events under changing conditions, for example, changes induced by treatments or external interventions.” (Pearl, 2010). We strongly agree with this emphasis on dynamics. Indeed, the temporal sequencing of cause and effect forces a focus on dynamics.

## THE H-D SCIENTIFIC METHOD AND PREDICTIVE MODELS

### General description

The H-D approach to science has a relatively long history in ecology (e.g., Chitty, 1960; Fretwell, 1972; Romesburg, 1981) and other disciplines (Platt, 1964; Popper, 1962) and has led to many important advances, even if it is not used as frequently as we would like. The approach can be briefly summarized in the following steps: (1) develop one or more hypotheses about how a system works, (2) deduce predictions from those hypotheses, (3) collect relevant observations, (4) compare observations with predictions, and (5) use these comparisons to either discriminate among competing hypotheses or support, or fail to support, a single hypothesis. Support for one or more of the tested hypotheses often leads to additional H-D testing, whereas lack of support may motivate the investigator to modify existing hypotheses or develop new ones. Although our focus is on the utility of predictive modeling, we note that SCM approaches can be used in Steps (1) and (4) of a general H-D framework (Grace, 2024; Grace & Irvine, 2020).

Step (4) of H-D science, the comparison of observations with predictions, is used to discriminate among competing hypotheses, to assess appropriateness (e.g., goodness of fit) of specific hypotheses, to estimate parameters of key model relationships (a focus of SCM), and to compute predictions (e.g., for subsequent H-D iterations and for conservation decisions). The importance of comparing observations with predictions as a path to understanding has been emphasized many times over the years to ecologists, most recently in contributions dealing with forecasting (Dietze et al., 2018; Houlahan et al., 2016). For this essay, the key point is that hypothesis-based predictions (typically produced by models corresponding to the hypotheses) are an essential ingredient for H-D science.

### Focus on causal relationships

H-D science provides a general approach to learning, with few restrictions on the kinds of data and observations that can be used with it. Thus, H-D approaches to assessing causation can be based on the analysis of static patterns of state variables (e.g., with some sort of spatial replication), specifically, by interpreting these patterns in terms of causal relationships about the processes that generated them. However, any static pattern can usually be generated by many different processes, so conclusions

from static analyses are frequently ambiguous and have produced controversy (e.g., Connor & Simberloff, 1984; Gilpin & Diamond, 1984). Plausible hypotheses about pattern generation often include both causal and noncausal (neutral) models (e.g., Caswell, 1976; Connor & Simberloff, 1979), with accompanying discussion (and disagreement) about what constitutes an appropriate noncausal model (e.g., Connor & Simberloff, 1984; Diamond & Gilpin, 1982; Gilpin & Diamond, 1984).

These difficulties associated with interpreting pattern-based analyses have led many ecologists to address causal hypotheses by focusing directly on the process parameters that underlie system dynamics (e.g., rates of extinction/colonization, birth/death, and habitat transition). Causation is addressed in such modeling by including parameters for the effect of putative causal factors on one or more process parameters. This approach is consistent with the view of causation as a “probability-raiser-of-processes” and usually entails observations from temporal sequences of state and other variables within the same system. Explicit incorporation of time in system models also provides an avenue to directly deal with reciprocal causation (expected from many important ecological relationships, e.g., competition, predation, parasitism, disease) and feedback loops (e.g., density dependence). Because the effects of causal variables are modeled directly, confounding is not a major problem. We can use the entire system of equations, modify one variable, and then estimate the effect of that variable as the difference between the response variable value before and after the modification. If the estimated effect approaches 0, or model selection does not favor this model, then this indicates the absence of probabilistic causation. Van Horne (1983), Tyre et al. (2001), Yackulic et al. (2015), and MacKenzie et al. (2018) present more detailed discussions contrasting pattern- versus process-based approaches for investigating ecological dynamics, arguing for direct inference on parameters that govern processes (referred to as dynamic rate parameters and vital rates).

With respect to the utility of resulting causal inferences, consequences of management and conservation actions are frequently modeled via their effects on dynamic rate parameters (Nichols, 2021). In addition, management applications often require time-specific models for making state-dependent decisions (Eaton et al., 2021; Johnson et al., 1997; McGowan et al., 2015), and average causal effects based on static variables at multiple locations are less likely to be useful for this purpose. These observations argue for drawing inferences about causation using the process-based approach described above, as such inferences will be useful for management and conservation.

## CRITICISMS OF PREDICTIVE MODELING AND MODEL SELECTION BY SOME SCM PROPONENTS

### Predictive modeling is not useful for inferring causation

Arif and MacNeil (2022b) discuss the analysis of observational data and state that “predictive approaches are not appropriate for drawing causal conclusions,” distinguishing between prediction and causal inference. They summarize their view of the distinction between prediction and causation by stating that “predictive inference (i.e., which model best predicts Y?) ... is fundamentally distinct from causal inference (i.e., what is the effect of X on Y?).” Some SCM proponents who discuss causal inference even define “prediction” as having “no focus on the causal or temporal structure among the explanatory variables” (Laubach et al., 2021). Although the tasks of prediction and inference differ, we emphasize that one important use of prediction is as a component of inference.

Predictive modeling is sometimes carried out in the absence of a causal model. For example, attempting to predict one variable with many covariates in the absence of a priori causal hypotheses may be useful for causal hypothesis generation, but does not lead directly to inferences about causation. Such uses differ substantially from most descriptions of the scientific method, in which the key step is the comparison of observations against model-based predictions (e.g., Chamberlin, 1897; Platt, 1964; Popper, 1962; Williams et al., 2002). In such comparisons, the models are frequently based on causal hypotheses, leading to direct inferences about causation.

We thus disagree with efforts to divorce the concepts of prediction and causation, instead viewing predictions as essential for most inferences about causation. Definitions of causation make explicit predictions about the relationship between the occurrences of cause and effect. Indeed, the potential to use causal models to make predictions about effects is one of the primary reasons for wanting to understand causation.

### GLMs and model selection can yield biased results

Focusing on the relationship between a response variable and potential causal variables, proponents of SCM describe situations in which GLM analyses can produce biased estimates of causal effects (e.g., Arif & MacNeil, 2022a, 2022b; Elwert, 2013; Ferraro et al., 2019; Greenland et al., 1999; Laubach et al., 2021; Pearl, 2009).

For example, these situations include the use of a predictor (hypothesized causal factor) that is an intermediate variable along a causal pathway and the use of covariates that affect both predictor and response variables. Analytic approaches based on DAGs (most prominently, the “backdoor criterion” procedure; Pearl, 2009) were developed to produce unbiased inferences about causation in the face of these potential sources of bias and are an important contribution of SCM. We believe that influences of non-focal variables can also be dealt with in a traditional analytic framework by hard thought devoted to model construction and by the direct modeling of vital rates.

Criticisms of predictive modeling include a simulated example (Arif & MacNeil, 2022b) in which simulations were based on a DAG that represented an exact depiction of the system’s causal structure. Estimation naturally reproduced a focal causal effect parameter accurately. Multiple GLMs were fit to the simulated data as well, and the selected model did not estimate the magnitude of the effect accurately (Arif & MacNeil, 2022b). This would be expected, as the simulated DAG model was not a member of the GLM model set. Rather than providing evidence against predictive GLM modeling, this example illustrated that reasonable inferences can be obtained when estimation is based on perfect knowledge of a system’s causal structure and that such inferences can be superior to multimodel inference when a good approximating model is not included in the model set.

In contrasting SCM and predictive modeling, many of the proponents of SCM target the use of GLMs and related analytic methods with multiple covariates and an “all possible models” approach. Similar criticism has been leveled by many other ecologists over the years (e.g., Burnham & Anderson, 2002; Nicholson, 1954; Romesburg, 1981; Rexstad et al., 1988; Williams et al., 2002). Such criticisms are especially relevant now, with the current emphasis by some on “big data” analyses using machine learning (ML) and artificial intelligence (AI) methods (e.g., Nichols et al., 2012). Such approaches can be defended as potentially useful in exploratory analyses, but the outcome of such analyses is hypothesis generation rather than reliable inference about causation. In summary, predictive models and model selection have often been misused, but when thoughtfully used, they continue to be very useful to hypothesis-driven science.

The use of a DAG, the identification of potential sources of bias, and the development of methods that properly deal with such bias are important and useful contributions of SCM. However, the accompanying criticism, by some, of GLMs, predictive models, and model selection is misleading. These criticisms should target

only the subset of such analyses not guided by a priori hypotheses.

## INVESTIGATING CAUSATION USING GLMs AND MODEL SELECTION

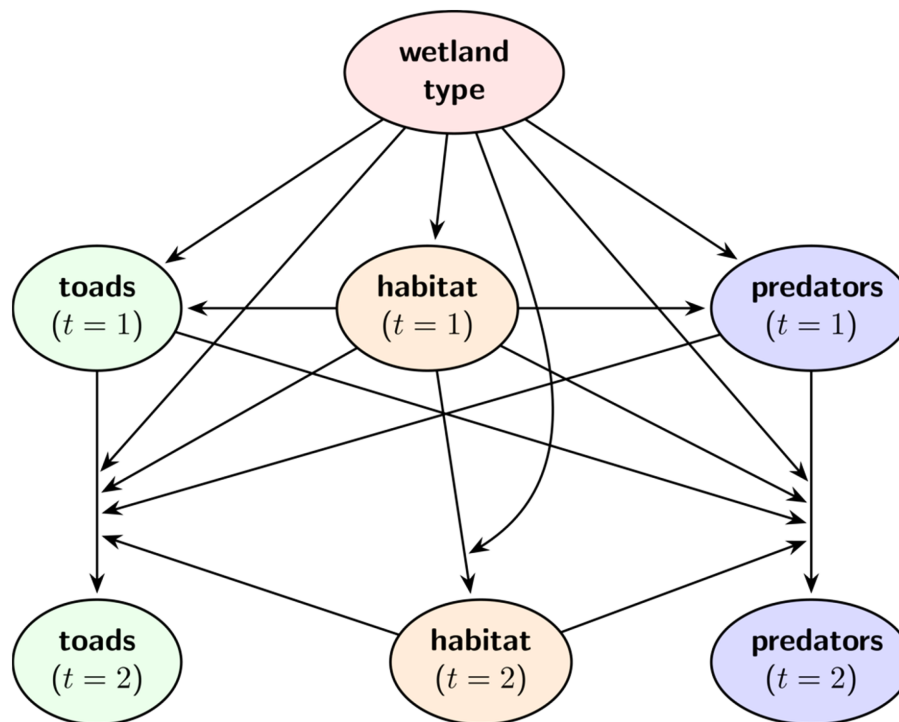
### Example 1

Here, we present an example analysis of an ecological system based on GLMs and model selection. We use an example that we know to avoid potentially misinterpreting what others have done. Miller et al. (2012) studied an endangered species of toad (Arroyo toad, *Anaxyrus californicus*) and two variables thought to have important causal influences on toad presence at a site: (1) presence/absence of a predator community composed of bullfrogs, crayfish, and predatory fish; and (2) habitat suitability—whether a site contained water or not at the beginning of the breeding season. Toad presence, predator presence, and habitat suitability were all dynamic state variables. An additional static variable was wetland type, indicating whether the site was populated with ephemeral or perennial streams. The study was conducted within three watersheds at

Camp Pendleton in southwestern California. Details of sampling were very important to model development in this study and were appropriately modeled (for details, see Miller et al., 2012), but here, we focus only on causal structure of the model.

A diagram specifying hypothesized causal relationships for this system is presented in Figure 1. This is not a DAG, but rather our attempt to diagram causal structure in a dynamic model. It includes two different time steps, year  $t = 1$  and year  $t = 2$ , corresponding to the first two annual sampling occasions of the Miller et al. (2012) study. Subsequent time steps would be diagrammed in a similar manner to  $t = 2$ . The inclusion of time permitted a focus on the vital rates governing state variable transitions, dealt with reciprocal causation between toads and predators (e.g., see Greenland et al., 1999), and incorporated Markovian dependence; for example, toad state for a site at time  $t + 1$  depends in part on toad state for that site at time  $t$ .

The causal structure of Figure 1 shows variables affecting vital rates, rather than only system state variables. The arrows in Figure 1 are based on ideas of probabilistic causation (Schaffer, 2001). For  $t = 2$  (and subsequent times), potential causal factors directly affect the state transition parameters (i.e., probabilities of site



**FIGURE 1** Causal influence diagram for the first two sampling periods in the general model of Miller et al. (2012). Time  $t = 1$  corresponds to the initial sampling period of the study and  $t = 2$  to the second sampling period. At  $t = 1$ , there is no prior information on state variables (toads, predators, and habitat), so potential causal factors are modeled as affecting the state variables directly. For  $t = 2$  (and subsequent times not represented in this figure), potential causal factors directly affect the transition parameters (i.e., probabilities of site extinction and colonization) that govern the arrows connecting state variables at time  $t = 1$  and time  $t = 2$ .



extinction and colonization). At  $t = 1$ , there is no prior ( $t = 0$ ) information on state variables (toads, predators, habitat), so potential causal factors are modeled as affecting the state variables directly.

As an example of our diagramming of state transition parameters following time  $t = 1$ , note that four arrows point to the arrow specifying the transition between toad state at  $t = 1$  and at  $t = 2$ . Two of these arrows originate at habitat suitability (presence/absence of water at times  $t = 1$  and  $t = 2$ ). If a site is unsuitable at time  $t = 2$ , then neither toads nor predators can be present. If the habitat is suitable at  $t = 2$ , the probability of toad presence then is partly dependent on habitat state at time  $t = 1$ . If toads are absent from the site at time  $t = 1$ , then the probability that toads colonize between  $t = 1$  and  $t = 2$  is hypothesized to be greater if habitat is suitable at  $t = 1$  than if habitat is unsuitable. If toads are present at a site at time  $t = 1$ , the presence of predators at the site at time  $t = 1$  is hypothesized to increase the probability of local extinction. If toads are absent at time  $t = 1$ , then the presence of predators at that time is hypothesized to decrease the probability of local colonization of toads between  $t = 1$  and  $t = 2$ . Wetland type (ephemeral or perennial) is not time-specific and may influence toad dynamics beyond simply its effect on habitat suitability. The inclusion of wetland type represents an effort to deal with unidentified causal variables that may be associated with these sites (e.g., akin to the inclusion of site in the modeling of Dee et al., 2023). Finally, the toad state at time  $t = 1$  clearly influences the Markovian transition to toad state at time  $t = 2$  by specifying the appropriate process transition parameter, extinction, or colonization probability.

Rather than creating a diagram such as Figure 1, Miller et al. (2012) specified this structure in a transition matrix, elements of which were functions of vital rates (extinction, colonization) for the three dynamic variables. Our central point with this description is that the analysis of Miller et al. (2012) included multiple hypotheses about causal relationships among the variables of this system. This detailed specification provided a means of dealing with the various sources of bias identified by proponents of SCM. For example, “confounding bias” is stated to occur when a variable that affects both a predictor and the response variable of interest is not “controlled for” (e.g., Arif & MacNeil, 2022b; Greenland et al., 1999; Laubach et al., 2021). Habitat suitability at time  $t = 1$  would be such a variable in Figure 1, affecting the probability of both toad and predator presence at time  $t = 2$ . However, this causes no problems for models based on Figure 1, because the multiple relevant relationships that were identified are all included. The effect of predators at  $t = 1$  on toad presence at  $t = 2$  can be directly estimated

with no confounding habitat effects (habitat effects are part of the model).

A dynamic occupancy modeling (MacKenzie et al., 2003, 2018) framework was used to deal with nondetection of toads and predators during sampling. Transition and detection parameters were modeled as functions of system variables using a GLM approach with logit links in a likelihood framework (Miller et al., 2012). The diagram of Figure 1 represents the most general (most relationships and parameters) model investigated, but simpler models were considered as well. Effect parameters associated with the different variables were estimated directly, conditional on model structure.

In distinguishing between standard statistical analysis and causal analysis, Pearl (2010) stated “Causal analysis goes one step further; its aim is to infer not only the likelihood of events under static conditions, but also the dynamics of events under changing conditions, for example, changes induced by treatments or external interventions.” Based on the analysis of Miller et al. (2012), predicted effects of a change in any system variable on a response variable can be computed directly based on the modeled transition parameter estimates. Such predicted effects can be computed conditional on specified values for each nonintervention variable or else computed as averages over sets of such values.

As with most observational approaches to investigating causation, the results of Miller et al. (2012) did not prove any of the hypothesized causal relationships. Instead, some of the hypothesized relationships of Figure 1 were supported, and estimates of effect parameters were obtained. Subsequent studies of this system would be required to produce additional support and increase strength of inference.

## Example 2

Our second example represents one step in a sequence of studies focused on a specific causal question of conservation concern. Various lines of evidence based on more mechanistic studies of behavioral interactions, food habits, habitat preferences, etc., led to the hypothesis that the barred owl (*Strix varia*) invasion of the Pacific Northwest may have been partially responsible for declines in northern spotted owl (*Strix occidentalis caurina*) populations (see reviews in Dugger et al., 2011; Forsman et al., 2011; Yackulic et al., 2014). Replicate detection data were collected during spring and early summer over a 22-year period for spotted owls and barred owls on a study area in western Oregon. A total of 158 contiguous polygons (i.e., potential spotted owl territories, “patches”) were surveyed, covering an area of

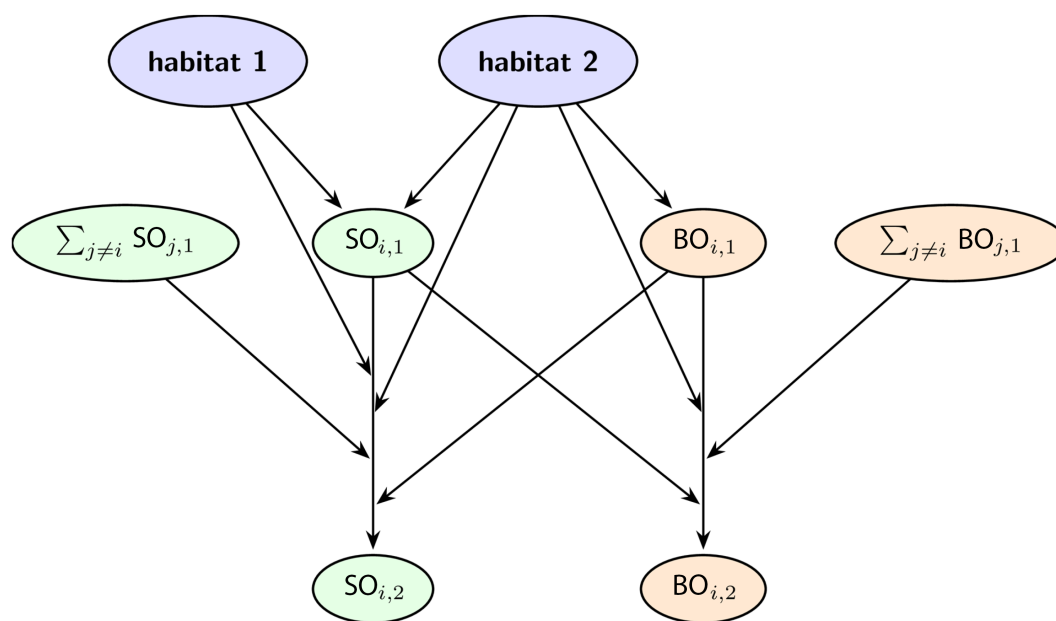
approximately 1000 km<sup>2</sup> (see Yackulic et al., 2014 for details). In 1990, at the beginning of the study, barred owls occupied virtually no patches, but had increased to about 90% occupancy in 2011.

Yackulic et al. (2014) focused on the primary hypothesis that barred owl presence in a patch increases the probability of patch-level spotted owl extinction and decreases the probability of patch-level spotted owl colonization. Reciprocal effects of spotted owl presence on barred owls were hypothesized but were expected to be much smaller in magnitude. A previous study (Yackulic et al., 2012) supported an effect of a habitat variable (denoted in Figure 2 as habitat 2) on barred owl occupancy, extinction, and colonization. For spotted owls, habitat 2 and an additional habitat variable (habitat 1) were hypothesized to be positively related to initial occupancy (sampling period 1) and colonization, and negatively related to extinction. Total study area patches occupied by conspecifics (obtained by summing occupancies of all non-focal patches) was hypothesized to positively affect colonization (mechanistically, more sources of colonists) and negatively affect extinction (rescue effect, Brown & Kodric-Brown, 1977). A causal diagram for this system (Figure 2) shows both the first and second years of sampling, with all subsequent years modeled as for year 2.

Figure 2 does not show the modeling of the sampling process, which was important, as nondetection of one species at a site does not mean its absence. Even more important, detection probability for each species was modeled as a function of presence of the other species, and results supported previous work (Bailey et al., 2009) that barred owl presence results in decreased detection probabilities for spotted owls. The modeling of components of the sampling process (e.g., detection probability, misclassification) as functions of covariates has been yet another effort by ecologists over the years to deal with potential confounding variables, where the confounding in this case affects the process of data generation.

The effect parameters for all Figure 2 arrows were estimated by Yackulic et al. (2014). A key result was that patch-level extinction probability for spotted owls increased from 0.09 per year when barred owls were absent to 0.29 when they were present in the patch, at 2011 occupancy levels, supporting a primary causal hypothesis of substantial conservation relevance.

A single analysis of observational data, whether using DAGs, GLMs, and model selection, or any other approach is very unlikely to provide definitive evidence of a causal effect. Multiple studies in which predictions of a causal hypothesis are consistently supported naturally increase our belief in the relationship. Analyses very



**FIGURE 2** Causal influence diagram for the first two sampling periods in the general model of Yackulic et al. (2014).  $SO_{i,t}$  = spotted owl occupancy for site  $i$  at sampling period  $t$ , and  $BO_{i,t}$  = barred owl occupancy for site  $i$  at sampling period  $t$ .  $\sum_{j \neq i} BO_{j,1}$  and  $\sum_{j \neq i} SO_{j,1}$  represent the number of non-focal sites occupied in the entire study area by barred owls and spotted owls, respectively, at the first sampling period. Time  $t = 1$  corresponds to the initial sampling period of the study, and  $t = 2$  to the second sampling period. At  $t = 1$ , there is no prior information on state variables ( $SO_{i,1}$ ,  $BO_{i,1}$ ), so habitat variables are modeled as affecting the state variables directly. For  $t = 2$  (and subsequent times not represented in this figure), potential causal factors directly affect the transition parameters (i.e., probabilities of site extinction and colonization) that govern the arrows connecting state variables at time  $t = 1$  and time  $t = 2$ .

similar to those of Yackulic et al. (2014) were repeated for multiple study areas in the Pacific Northwest by Dugger et al. (2016) and Yackulic et al. (2019), and results supported the initial findings. Experimental-barred owl removals, and subsequent spotted owl response, at a single study area provided additional support (Diller et al., 2016). Finally, a larger BACI-design investigation across multiple study areas provided strong support for negative barred owl effects on spotted owls (Wiens et al., 2021). This cumulative work provides a nice example of a sequence of studies, all based on predictive modeling, yielding stronger and stronger inferences about a causal relationship. Although formal “proof” of causation is elusive for complex ecological systems, accumulated evidence of the barred owl-spotted owl relationships is very strong and has appropriately led the US Fish and Wildlife Service to consider a large-scale barred owl removal program as a measure to conserve spotted owls. Accumulation of evidence about causation through multiple studies provides one approach to combatting the crisis of reproducibility and replicability in observational science (Nichols et al., 2019, 2021).

## Summary comments on examples

The work of Miller et al. (2012) and Yackulic et al. (2014) represents careful analyses of simple ecological systems and provides examples of many such analyses carried out using predictive models embedded within capture–recapture and occupancy modeling frameworks. Within these frameworks, the importance of carefully developing causal hypotheses is reflected in software development, with the most widely used data-analytic programs (e.g., Cooch & White, 2024; Hines, 2006; Kéry & Royle, 2021; Lebreton et al., 1992) requiring users to carefully specify their models of both ecological and sampling processes. As advocated by the proponents of SCM, the analyses of Miller et al. (2012) and Yackulic et al. (2014) were hypothesis-driven.

A central point of these examples is that ecologists have conducted careful analyses permitting inferences about causation using predictive modeling and model selection within an H-D process and in the absence of SCM guidance. Data in our two examples were fit to multiple plausible models developed for a small set of a priori causal hypotheses, an approach found in some SCM efforts (e.g., Grace, 2024; Grace & Irvine, 2020). Model selection was used to help assess support for the different models, and model-averaging was used, when appropriate, to express model uncertainty for estimation of some focal parameters (see Buckland et al., 1997; Burnham & Anderson, 2002).

Both examples based causal modeling on the vital rates governing state variable dynamics. Predictive modeling for investigating causation can be used with static state variables as well, but we believe that process-based modeling is one step more “mechanistic” and provides some advantages. For example, assume a causal factor that influences the proportion of patches in a metapopulation that is occupied. The causal factor must act upon patch-level probabilities of colonization and extinction. Even when these effects of a causal factor on vital rates do not vary, effects on changes in the proportion of patches occupied will depend strongly on the current state of the system (Yackulic et al., 2015). If few patches are occupied at time  $t$ , then the proportion occupied at  $t + 1$  will be dominated by the effect of the causal factor on colonization probability. However, if a large proportion of patches is occupied at  $t$ , then occupancy at  $t + 1$  will be largely determined by the effect of the causal factor on local extinction probability. The imposition of the causal factor will typically produce different changes in the proportion of patches occupied, depending on the initial system state. Initial system state is likely to differ for systems characterized by different vital rates, by whether the system is experiencing transient dynamics versus near equilibrium, etc.

Both of our examples included potentially confounding variables, as a means of clarifying the specific influences of these variables and obtaining unbiased estimates of effect parameters. In the first example, unidentified variables associated with wetland type were dealt with via the inclusion of this variable (see Dee et al., 2023 for similar approach). Both examples also included models for both ecological and sampling processes, recognizing that confounding in either process can become a source of bias. Estimated variances of effect parameters included variance components associated with detection probability, the stochastic variation associated with the modeled dynamic processes, and, in some cases, model uncertainty. Considerations of the various issues illustrated in these two examples have a long history in careful ecological investigations and methodological texts (e.g., Kéry & Royle, 2015, 2021; MacKenzie et al., 2018; Seber, 1982; Seber & Schofield, 2019; Williams et al., 2002).

Finally, we note that both of our examples are based on dynamic occupancy modeling (MacKenzie et al., 2018), in which the state variable is species presence and vital rates are probabilities of local extinction and colonization. However, the same approach can be used within a capture–recapture framework (e.g., Cooch & White, 2001; Lebreton et al., 1992; Seber, 1982; Seber & Schofield, 2019; Williams et al., 2002), where species abundances are the state variables and rates of survival and recruitment are the vital rates (e.g., see Yackulic et al., 2018).

## DISCUSSION AND CONCLUSIONS

Proponents of SCM generally argue for an approach to causal inference that is heavily dependent on a priori hypotheses, and we believe that this argument is important and deserving of ecologists' attention. Indeed, structural uncertainty about causal hypotheses can yield DAGS that cannot be distinguished by observational data alone but require interventions and observations of resulting dynamics to resolve (Castellitti & Consonni, 2020). SCM approaches also devote special attention to identifying potential confounding variables and eliminating their biasing effects on casual parameter estimation, an important focus that is also deserving of greater attention in ecological analyses.

Some proponents of SCM criticize the use of predictive modeling and model selection for drawing causal inferences. We agree that the use of GLMs and model selection with many available covariates, absent strong a priori hypotheses about causal relationships with the response variable and interrelationships among covariates, can often be affected by different forms of bias and are frequently not useful for causal inference. However, we also believe that the thoughtful use of predictive modeling and model selection within an H-D framework is an important means of learning, both in general terms and with respect to causation. Within this framework, predictive models are developed for hypotheses that are themselves frequently based on knowledge of relevant mechanisms. Well-developed methods exist to discriminate among competing predictive models, assess model fit, provide estimates of key parameters and associated variances–covariances, and make predictions (with associated measures of uncertainty) of system response to changes in causal factors. Formal approaches have also been developed for accumulating knowledge across multiple studies using comparisons of observations with model-based predictions (e.g., Hilborn & Mangel, 1997; Nichols et al., 2019, 2021).

The temporal component of causation forces an interest in dynamics, leading us to outline a specific H-D approach focused explicitly on system dynamics. Under this approach, causal relationships are modeled as effects on system vital rates, consistent with a probability-raiser-of-processes perspective (Schaffer, 2001). Effects of confounding causal variables that can be identified, potentially confounding sampling processes, feedback loops, and reciprocal causation can be modeled directly. Well-developed approaches to model selection are available, as are methods for incorporating model uncertainty into estimated variances. Of course, causal modeling within a H-D approach is not restricted

to this focus on vital rates and can proceed using static state variables as well.

Although identified confounding variables can be dealt with using SCM and predictive modeling approaches, the issue of potential unidentified causal factors casts uncertainty over most causal inferences, whether they are based on predictive modeling or SCM. SCM and predictive modeling proponents have developed useful approaches for addressing this issue to a degree (e.g., see Dee et al., 2023 and above), but none of these guarantees that results are not affected by unidentified confounders. This issue argues strongly for (1) a combination of mechanistic knowledge and hard thinking in the development of hypotheses (e.g., Grace, 2024; Grace & Irvine, 2020; Nicholson, 1954) and (2) repeated testing of hypotheses to accumulate evidence (e.g., Grace, 2024; Hilborn & Mangel, 1997; Nelder, 1986; Nichols et al., 2019, 2021; Tredennick et al., 2021). This recommendation for repeated testing should not be interpreted as a recommendation to delay decisions. As noted in the discussion of spotted owls, conservation decisions need not await “proof,” but should be based on the best available information, that is, that which has accumulated up until the time of the decision.

Our perspective that predictive modeling in conjunction with H-D science can be useful for investigating causation is consistent with the concept of evidential pluralism, which argues that causal investigations require both mechanistic hypotheses and tests of association (Grace, 2024; Shan & Williamson, 2023). The hypothesis-generation step of H-D science is generally informed by mechanistic knowledge of underlying causal relationships, and there are multiple kinds of mechanisms and multiple ways to model associations as well. Further extending the general idea of pluralism, our own view is consistent with that of Fretwell's (1972) comments on H-D science: “A discussion of philosophy is, or should be, a plea for tolerance, not gratuitous advice. Whatever works should be acceptable...”

In conclusion, SCM provides one approach to the study of causation using observational data, and the SCM emphasizes on specifying causal hypotheses and identifying causal effects in the presence of potential confounders deserve more attention. Counter to claims by some, predictive modeling can also be used to investigate causation. The comparison of model-based predictions against empirical observations provides the evidence that we use to either corroborate focal hypotheses or fail to support them. These comparisons also provide the basis for updating model “weights” (reflecting the relative confidence in the different models of the model set) in efforts to accumulate knowledge across studies (e.g., Hilborn & Mangel, 1997; Johnson et al., 1997; Nichols et al., 2019,



2021). In addition, model-based predictions provide the basis for management and conservation decisions, a fact responsible for much of our interest in causation. It would be unfortunate if readers of some SCM advocates concluded that model-based predictions, and their degree of correspondence with observations, were somehow divorced from drawing inferences about causation. This comparison between predictions and observations forms the basis for the H-D scientific method.

## ACKNOWLEDGMENTS

We wish to thank Jim Grace, Aaron MacNeil, Andy Royle, and Charles Yackulic for insightful discussions about this topic and suggestions for improving this manuscript. We thank reviewer Kaitlin Kimmel-Hass for useful and constructive comments on an earlier draft of this manuscript. Any use of trade, firm, or product names is for descriptive purposes only and does not imply endorsement by the US government.

## CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

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**How to cite this article:** Nichols, James D., and Evan G. Cooch. 2025. "Predictive Models are Indeed Useful for Causal Inference." *Ecology* 106(1): e4517. <https://doi.org/10.1002/ecy.4517>