

An integrative paradigm for building causal knowledge

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Abstract

A core aspiration of the ecological sciences is to determine how systems work, which implies the challenge of developing a causal understanding. Causal inference has long been approached from a statistical perspective, which can be limited and restrictive for a variety of reasons. Ecologists and other natural scientists have historically pursued mechanistic knowledge as an alternative approach to causal understanding, though without explicit reference to the requirements of causal statistics. In this paper, I describe the premises of an expanded paradigm for causal studies, the Integrative Causal Investigation Paradigm, that subsumes causal statistics and mechanistic investigation into a multi-evidence approach. This paradigm is distinct from the one articulated by causal statistics in that it (1) focuses its attention on the long-term goal of building causal knowledge across multiple studies and (2) recognizes the essential role of mechanistic investigations in establishing a causal understanding. The Integrative Paradigm, consequentially, proposes that there are multiple methodological routes to building causal knowledge and thus represents a pluralistic perspective. This paper begins by describing the crux of the problem faced by causal statistics. To understand this problem, it should be recognized that the word *causal* has multiple meanings and a variety of evidential standards. An expanded vocabulary is developed so as to reduce ambiguities and clarify critical issues. I further show by example that there is an important ingredient typically omitted from consideration in causal statistics, which is the known information related to the mechanisms underlying relationships being evaluated. To address this issue, I describe a procedure, Causal Knowledge Analysis, that involves an evaluation and compilation of existing evidence indicative of causal content and the features of mechanisms. Causal Knowledge Analysis is applied to three example situations to illustrate the process and its potential for contributing to the development of causal knowledge. The implications of adopting the proposed paradigm and associated procedures are discussed and include the potential for advancing ecology, the potential for clarifying causal methodology, and the potential for contributing to predictive forecasting.

An author biosketch is provided in Appendix S1.

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INTRODUCTION

Ecologists frequently draw causal interpretations based on their investigations though it is widely known that there are cautions against this practice. Most of the time, the word “causal” is not used explicitly by scientific investigators while terms such as “effects,” “responses,” “drivers,” “influences,” and numerous others are used to convey a similar intent in a more context-dependent fashion. When the word causal is used, there is commonly some elevated degree of cautiousness applied, such as reliance on randomized experiments or use of special methods of design and analysis that others have described as causal methods. One could say that there is considerable mystery surrounding the precise requirements for drawing explicitly causal interpretations while the use of alternative terms seems to depend on some different set of evidential standards that fall within the scientist’s domain of expertise.

There exists a vast literature on the topic of causality that has been generated by discussions in the fields of statistics, the social sciences, and by philosophers over the past century. For the sake of this presentation, I use the phrase “causal statistics” to refer to the techniques and evidential requirements that have originated from statisticians and adherents to their empirical viewpoint (e.g., Angrist & Pischke, 2010; Hill et al., 2018; Imbens & Rubin, 2015; Rubin, 1974; VanderWeele, 2015). Modern derivations of causal statistics are now being introduced into ecology (e.g., Ferraro et al., 2019), potentially creating confusion that may be addressed with a broadening of perspective. Despite superficial overlap due to modern fusions of notation and terminology, it is best to distinguish causal statistics from the enterprise of structural modeling which originated from scientists (Pearl, 2009; Wright, 1921) and whose application depends strongly on mechanistic knowledge, a central topic in this paper.

In this paper, I describe an integrative approach to causal investigation that differs from traditional causal statistical analyses in two ways. First, it shifts the focus away from the restrictive requirements for isolating bias-free causal effect estimates to the long-term/across-study scientific ambition of building causal knowledge. By itself, this has a fundamental influence on the types of evidence to be considered in causal investigations. Second, I propose that there is a key ingredient

missing from standard causal statistical analysis, specifically a consideration of preexisting mechanistic knowledge. Here a *mechanism* refers to, *a collection of spatiotemporally contiguous structures and processes along which a signal can be propagated from one entity to another with the potential of producing a response*. A mechanism is often made up of a set of *mechanistic elements* that work together. As an example of a mechanism, if two trees establish near each other, as they grow their canopies and root zones begin to use the same space. Those proximities generate changes in one another’s resource supplies and environments. Many consequences are possible, but the shared pursuit of common resources establishes a physical, chemical, and biological basis for mechanistic interactions. Those interactions are appropriately describable as causal regardless of whether we know all the details or not (regardless of the *depth of our mechanistic understanding*). An analogous set of opportunities for mechanistic interactions occur when organisms of any type share space or a common resource pool. Mechanisms in some case may involve behavioral responses. What is important for the subject of this paper is that such underlying mechanistic information is not typically considered in causal statistical analysis. Causal statistics focuses on the challenge of inferring causal relationships from data and from information related to how it was obtained.

In the current paper, I approach these issues by first describing the crux of the problem of drawing causal interpretations from data analyses. I then present brief examples of causal statistical analysis and causal mechanistic investigation and discuss differences in how they are approached and what they achieve. I go on from there to define new terminological distinctions that can reduce ambiguities and thereby facilitate a broader discussion of methods for causal investigations. I follow this material by describing an integrative approach to causal investigation more compatible with the process of scientific inquiry as practiced in the natural sciences, which I refer to as the Integrative Causal Investigation Paradigm (Integrative Paradigm hereafter). I then describe a new procedure, Causal Knowledge Analysis, that involves the evaluation of existing knowledge documenting the evidence for causal content and the nature of the mechanistic elements. Three examples are presented to illustrate Causal Knowledge Analysis and to further make the case

for the role of mechanistic investigation in building causal knowledge. I end by considering the implications of adopting the described paradigm, which can reconcile competing viewpoints and promote greater progress toward the scientific goal of increased causal understanding.

CAUSAL STATISTICS AND THE CRUX OF THE PROBLEM

The causal statistics literature dates back to Karl Pearson, who is credited with creating the first journal specifically dedicated to the subject of statistics, *Biometrika*, in 1901. It was his view that drawing causal inferences from data was not possible because of the challenge posed in Figure 1 (discussed in Pearl & Mackenzie, 2018, chapter 2). This opinion held among statisticians until Ronald Fisher developed the method of randomized experiments in 1925 (Hall, 2007), which provided a narrowly defined exception to that generalization. Beyond that, instrumental variable methods, a specialized technique capable of yielding bias-free causal estimates from observational data under special circumstances (Grace, 2021), was developed by Joseph and Sewall Wright in the 1920s, (Angrist & Pischke, 2015) though it only became popular many years later. In general, however, the field of statistics has held a highly skeptical view of the possibility of extracting causal inferences from data under most circumstances through much of its history.

Despite the advice of statisticians, scientists commonly draw causal inferences from the results of nonexperimental investigations while avoiding the word

causal. Common examples in ecology include statements such as, “Refuge quality *impacts* the strength of nonconsumptive *effects* on prey” and “Resource pulses from periodical cicadas *propagate* to belowground food webs” (italics added for emphasis). These examples point to a disconnect between statistical advice and scientific practice in ecology that is discussed in this paper.

There are major differences among the sciences regarding the degree to which nonexperimental causal methods have been discussed. It is notable that the natural sciences, including ecology, have largely avoided the topic while the social sciences and certain fields like economics and epidemiology have engaged in continuous discussions of the subject for many decades (e.g., Heckman, 2022; Morgan & Winship, 2015, sect. 1.2; Pearl, 2009, Epilog). Due to the lack of transfer of methodological information across disciplines, ecologists are likely to be unfamiliar with the causal statistics literature. However, some of this literature is now being presented to ecologists primarily by economists (e.g., Ferraro et al., 2019), precipitating a need for clarifying several issues.

Figure 1 attempts to summarize the underlying basis for historical debate and ongoing confusion related to causal methodology. In this illustration, the interest is in estimating the causal effect of X on Y . While there is a large collection of literature dealing with the meaning of the word “causal” (see for example Beebe et al., 2009; Cartwright, 2004), at this juncture I will simply describe $X \rightarrow Y$ as a causal relationship if there is reason to think that variations induced in X can propagate to subsequent variations in Y . This modest explanation is intended to be minimally sufficient to allow me to explain why there

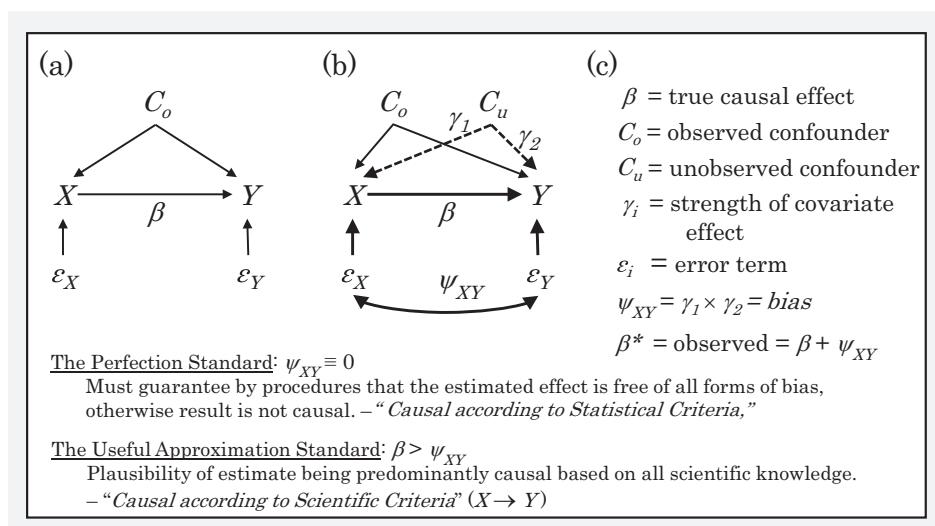


FIGURE 1 Distilled summary of the basis for historical debate and confusion over causal methodology. (a) Causal diagram for the case where observed confounders C_o are included in the model and thereby controlled for. (b) Causal diagram illustrating the case where there are unobserved confounders C_u omitted from the model. (c) Definitions for parameters. See text for further description.

has been so much disagreement over what constitutes causal methods, causal effects, causal inference, and causal knowledge.

The drawings in Figure 1a,b represent *causal diagrams* (Grace & Irvine, 2020; Pearl, 2009). As such, they represent hypothesized cause–effect relationships among random variables. Such diagrams are considered to be non-parametric in the sense that any variable type and functional form (nonlinear, nonadditive) is permitted, which allows for general rules. In the calculation of bias presented in the figure, I adopt a linear model specification for convenience.

Let us first consider Figure 1a. Imagine X has a causal effect on Y such that variations induced in X may lead to changes in Y . Here, we further imagine that there exists some set of observed exogenous covariates C_o at least some of which have influences on both X and Y (and thus represent common-cause confounders). By controlling for the observed covariates (in this case by including the covariates in the model), we avoid having *endogeneity*, defined as a covariance between a cause and the error term of its accompanying response variable (Grace, 2021). In such a situation, we can estimate the true causal effect β without bias using standard statistical estimation methods.

Now, what if there are important unobserved covariates C_u having effects on both X and Y , as in Figure 1b? Such common causes omitted from our model create a so-called *backdoor pathway* that can lead to endogeneity and thus add some amount of bias to the estimate of the effect of X on Y that is observed, β^* . Assuming the error terms ε_X and ε_Y are on the same scale as X and Y , a linear estimate of the strength of the unmeasured backdoor pathway will be the product of its component segments $\gamma_1 \times \gamma_2$. This backdoor association induces an error correlation between X and Y , Ψ_{XY} . The consequence of this unmeasured confounding is that the observed association between X and Y is equal to the sum of the direct effect plus the backdoor association, so our observed estimated effect $\beta^* = \beta + \psi_{XY}$. Formally then, when $\psi_{XY} > 0$, we will have some degree of endogeneity and bias, which can be also created by measurement error in X and from feedback from Y to X (see Grace, 2021, supporting information).

The tradition within causal statistics has been to define a *causal method* as one where a perfectly unbiased effect estimate can be assured from the study design and/or method of analysis. I describe this requirement as a *perfection standard*. The axiomatic method that qualifies when executed successfully is the ideal randomized experiment, which assumes many things, including that randomization removes common cause confounding (C_u variables), and that we are only interested in the net effect of X on Y . Possession of an exogenous

(aka *instrumental*) variable that predicts a large fraction of X provides the ability to isolate a randomized portion of X and thereby qualifies as a causal method when used in combination with certain analytical techniques (Grace, 2021).

Now, a host of techniques have been developed to deal with confounding using statistical control. One approach attempts to approximate randomized experiments using matching methods such as propensity scores (e.g., Andam et al., 2008). Such an approach is often referred to as quasi-experimental. Strictly speaking, these are not causal statistical methods because they cannot guarantee pure estimates due to the untestable assumption that there is no unmeasured confounding. However, we can refer to the use of such methods as *perfection-seeking* as they attempt to confront and eliminate possible sources of bias, often by restricting the data that can be used and the analyses that can be made.

Scientists have long drawn causal interpretations without providing assurances of bias-free perfection or some approximation to that standard. Instead, a relaxed standard has often been used, which I will refer to as a *useful approximation*. The most common approximation that has been suggested is to argue on behalf of a qualitative declaration that X has a causal effect on Y , without knowing the exact amount of bias and effect magnitude. The counterargument that has been made is that only a pure estimate can ensure a proper qualitative conclusion (Morgan & Winship, 2015).

For the sake of the argument being made here, I will suggest that a causal effect estimate that is a useful approximation should be predominantly causal. Referring to Figure 1, this means the minimum standard would be, in absolute terms, $\beta > \psi_{XY}$ (the causal component is greater than the bias component). We can perhaps be somewhat encouraged by recognizing that for a case where γ_1 , γ_2 , and β are all = 0.5, ψ_{XY} will be $0.5^2 = 0.25$, and the observed estimated effect of $0.5 + 0.25 = 0.75$ will be 2/3 causal and 1/3 bias. This logic may be sufficient to allow us to expect that confounding will not always be fatal to the causal interpretation of data analysis. Nonetheless, not knowing exactly the degree of bias is problematic if we do not have a way of involving additional information.

It can be argued that one of the most pervasive impediments to involving additional information so as to address confounding is the standard multiple regression model. In contrast to the causal diagrams shown in Figure 1, which represent networks of relationships using a series of equations, multiple regression is described by a single equation, $y = \mathbf{BX} + \varepsilon$, where \mathbf{X} is a vector of potentially correlated predictors, \mathbf{B} is a vector of coefficients, and ε is a set of independent errors. Missing from

the regression equation is any overt means of representing hypotheses that explain the correlations among predictors (Grace & Irvine, 2020). This problem can be solved by recasting hypotheses in the form of causal networks.

A second impediment to involving additional information is that statistical models relying on random independent variables and random errors do not reflect the true contents of underlying mechanisms. This proposition will be explained further through three examples presented in the paper, one dealing with the causal relationship between the annual number of aviation accidents and the annual number of fatalities, another involving the physiology of African savanna grazing animals, and a third describing some of the ways interactions among grassland plants propagate competitive effects.

INDICATIONS OF A MISSING INGREDIENT—MECHANISTIC KNOWLEDGE

Let us now consider examples that indicate that there is an important ingredient frequently omitted from causal statistics analyses. Looking back at Figure 1, we are reminded that a primary objective of causal statistical analysis is to find ways to eliminate the threat of uncontrolled confounding so that we are assured perfect or near-perfect estimates. Commonly, the phenomena of interest or importance to ecologists cannot practically be examined through the exclusive use of randomized experiments. Requiring control of all imaginable unmeasured confounders (Figure 1a) puts severe restrictions on the use of nonexperimental data, though frequently such data are the only available form of evidence. Are we indeed stuck with a need for perfectly unbiased estimates in order to make confident causal claims or are there situations where reasonable certainty can be achieved without adherence to that requirement (Figure 1b)? Stated differently, are causal statistics the only path to causal knowledge?

A recent study (Martinez et al., 2023) examined the effects of an extreme freeze event in the southcentral United States in 2021 on a subtropical tree that has been expanding northward in recent years, the Black Mangrove. In this study, the authors conducted both causal statistical analyses and mechanistic characterizations. This one study thus allows me to briefly characterize and contrast the two approaches to building causal knowledge.

The foundational description of causal statistics involves *counterfactual* contrasts (Morgan & Winship, 2015). The goal is to solve the dilemma posed in Figure 1 by comparing

how individuals change from before to after some exposure compared with how those individuals would have changed if, counter to fact, they had not experienced the exposure. This can also be viewed as an “all else equal” comparison, which is a logic that can be applied to both dynamic and static observations (though the latter case involves additional assumptions). The idealized randomized manipulative experiment serves as a benchmark for how one obtains the data suitable for counterfactual contrasts by randomizing individuals into separate groups for each treatment and measuring responses before and after treatment applications. A related aspiration for causal statistical studies is to process data into different groups “as if randomized,” which describes the *quasi-experimental study design*. An *average causal effect* estimated according to the counterfactual principle is thus the quantitative difference between groups that are assumed to be, on average, equivalent in their potential responses to some exposure. For nonexperimental studies it can be challenging to meet this requirement, especially if one is adhering to the perfection standard (Figure 1a). A popular approach to this challenge is to rely on “event study methods” (Huntington-Klein, 2021), which require temporal data with measurements before and after some known event. Various procedures can be applied so as to estimate for the study units exposed what their later values would have been if not exposed. Typically, some version of fixed-effects statistical estimation procedure (Huntington-Klein, 2021, chap. 16) is employed so as to avoid confounding from differences among study units.

For the mangrove study, average causal effect estimates were made using the temporal dynamics of the aerial cover of trees quantified using Normalized Difference Vegetation Index (NDVI) values calculated from satellite data; Figure 2, upper panel. Data were obtained for several regions within the southern United States that experienced differing intensities of freezing. Examinations of the temporal trends in pre-event data and of samples that avoided exposure to freezing temperatures found no overall upward or downward trend in NDVI, but instead, seasonal cycles in NDVI. This information was used to derive counterfactual expectations for causal effect magnitude estimates for the effects of freezing on mangroves using appropriate time comparisons (see vertical gray zones in the upper panel of Figure 2 defining comparable times before and after the freeze). More details can be found in Martinez et al. (2023).

In the study by Martinez et al. (2023), a second set of analyses focused on characterizing underlying mechanisms determining the responses of mangroves to freezing. The lower panel in Figure 2 shows results from estimating the freeze tolerance of black mangrove (a key mechanistic element determining their responses to

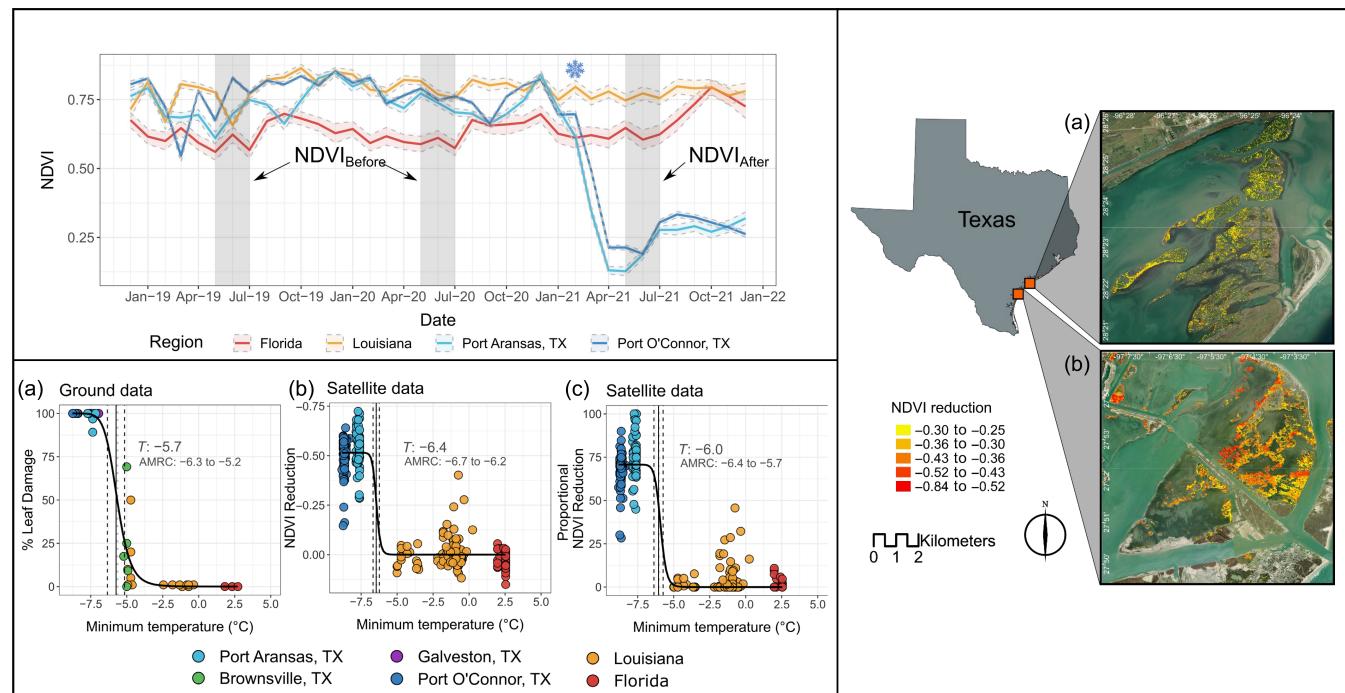


FIGURE 2 Illustration of the application of causal statistical methods (upper left panel) and mechanistic characterization using traditional statistics (lower left panel). From Martinez et al. (2023) with permission. Both sets of results are from an investigation of the effects of an extreme freeze event on black mangrove trees near a northern range limit. Counterfactual comparison methods were applied to the data represented in the upper panel as described in the text. The vertical gray zones in the upper panel represent comparable before-after time periods in the seasonal oscillations of the normalized difference vegetation index (NDVI). The bottom panel illustrates the estimation of the freeze tolerance of mangrove trees using standard logistic regression (note that solid vertical lines within-panel indicate discrete minimum temperature thresholds [T], while vertical dashed lines indicate threshold ranges [that is, areas of maximum rate of change, AMRC]). The right panel shows a map of landscape-scale spatial variation in causal effect estimates. Figure provided with permission from Melinda Martinez and Springer Nature.

freezing temperatures) using differing sources of data and expressions of response. Key to these analyses was to relate the heterogeneity in responses in space to corresponding temperature records. The analyses characterizing mangrove freeze tolerance limits utilized standard logistic regression and did not rely on causal statistical principles (counterfactual contrasts). Rather, the approach to causal inference used relied on external consistency (stability of threshold estimates across different data sources) and mechanistic knowledge (knowledge of the biology determining freeze tolerance thresholds, Osland et al., 2020).

It is useful to point out some distinctions between the information content of the causal effect estimates (shown in the right panel of Figure 2) and causal mechanism characterizations (bottom panel) for the mangrove study. Causal effect estimates, as they are commonly described, will often yield summaries that are specific to the sample studied. Perhaps the only transportable constraint on values we might find in future studies that estimate the causal effects of freezing on mangrove cover is that values will be expected to range between 0% and 100%. We can

imagine that there will be a great deal of effect size heterogeneity among individual plots because effect sizes reflect the convergence between the spatial distribution of the plants and the distribution of freezing temperatures, along with the distribution of conditional factors (such as duration of exposure of meristematic tissues, pre-event plant resources for recovery, etc.). The result is that causal effect estimates describe “what happened” and within the Integrative Paradigm qualify as causal observations but tell us little about the underlying machinery. A demonstration of the summary nature of causal effect estimates can be seen in the right frame of Figure 2, where effect sizes within a relatively small area are mapped and shown to vary from 84% to 25%. In cases such as this one, estimates of net effect will contribute only modestly to an accumulation of transportable causal knowledge since what is measured is very much influenced by the particular sample over which the effects are averaged. Characterizations of causal mechanisms, however, represent important contributions to transportable knowledge and understanding.

DISTINGUISHING THE MULTIPLE MEANINGS OF THE WORD “CAUSAL”

Before proceeding, we should further address the potential for confusion surrounding the use of the word “causal.” A key requirement for progress on any topic is to have sufficient linguistic distinctions so as to avoid semantic confusion, particularly when there are multiple meanings associated with a single term. In Figure 1, I propose that “causal according to scientific criteria” conforms to the typical use of the term causal by scientists—which is a statement that some entity can have a causal effect on another. To support such a claim, there is no mention of a conceptualization of causation in the Oxford Handbook of Causation (Beebee et al., 2009) that requires pure estimates or perfect methods. In contrast, causal statistics presentations use the word causal to imply a pure unbiased estimate. I propose this could be described as, “causal according to statistical criteria.” If we adopt a pluralistic perspective, it follows that we recognize a wide variety of situations and circumstances where pure bias-free estimates are either highly desirable or necessary, but also many situations where that is not a reasonable or necessary requirement. One goal of this paper is to demonstrate that a perfection standard cannot represent a universal requirement. Linguistic qualifiers can help us more easily consider the full variety of situations so as to avoid confusion and I suggest *causal according to statistical criteria* and *causal according to scientific criteria* or some other equivalent phrases, such as *causal in the statistical sense* versus *causal in the scientific sense*. Only by having sufficient terminological distinctions can we understand statements such as, “The relationship may be causal, but its estimate is not causal” (VanderWeele, 2016). This could more clearly be restated as, “The relationship may be causal according to scientific criteria, but the effect estimate is not causal according to purely statistical criteria.”

Even with distinctions in hand that clarify the multiple senses of the word causal, what remains is a very limited set of techniques that can guarantee pure unbiased estimates. Oversimplifying the situation enormously, we might view the 120-year debate over causal methods and drawing causal inferences as revolving around a divergence of opinions about how to deal with the nearly ubiquitous situation depicted in Figure 1b. Generally speaking, the field of causal statistics represents a paradigm that is focused on meeting the standard represented in Figure 1a. In this paper, I propose that knowledge of the mechanisms connecting two variables can allow us to proceed towards externally validated and even transportable causal knowledge based on useful approximations. This constitutes the basis for an alternative, pluralistic paradigm that

recognizes multiple approaches and permits a wide variety of types of evidence in the pursuit of causal knowledge and understanding.

In the discussion that follows, numerous terminological distinctions are presented to provide sufficient language for the task at hand. Box 1 defines terms and provides certain explanations so as to describe the pluralistic viewpoint presented in this paper. The list is not strictly alphabetical as some concepts depend or elaborate on the ones that come before.

EVIDENTIAL PLURALISM AND THE PHILOSOPHICAL ARGUMENT FOR INCLUDING EVIDENCE OF MECHANISM IN CAUSAL ANALYSIS

Causal philosophers have recently offered a conceptually broad alternative to the narrow strictures of causal statistics. This alternative is referred to as *Evidential Pluralism* (Williamson, 2021). The main idea behind this epistemic system is the recognition that in many scientific fields, causal knowledge is established through mechanistic studies. As this system is grounded in the tenets of science philosophy, it deserves formal treatment here in order to try to avoid, as much as possible, distortion through translation. An up-to-date treatment of this system is articulated in the first three parts of Shan and Williamson (2023).

Evidential Pluralism is described by its authors as “an account of causal inquiry” (Shan & Williamson, 2023). Its defined domain is the process of establishing and assessing causal claims. In the next section of the paper, I will present an alternative that extends the domain beyond these limits. Before that, it is important to first properly represent this source material and its described intent.

The nature of causality can be discussed from a number of different philosophical perspectives (Oxford Handbook of Causation—Beebee et al., 2009). The most commonly described high-level viewpoints in philosophy include metaphysical and epistemic. Evidential Pluralism represents an epistemic approach, which reflects an interest in the practical matter of how causal knowledge and understanding is built, rather than in the more abstract metaphysical issue of “the nature of causality.” Evidential Pluralism interprets causal claims (such as “A has a causal influence on B”) as beliefs that allow us to make certain practical inferences, not necessarily as claims about some absolute truth that exists independent of an observer. Causal claims described in this fashion serve to enable us to construct explanations, to make certain predictions about the new observations we might

BOX 1 Glossary

A1: Causal Relationship—Situation where two variables are connected through some mechanism or means such that variations in one can propagate to subsequent variations in the other.

A2: Causal Based on Statistical Criteria—Presumption that procedures used assure that the estimated effect is free of all forms of bias (aka *the Perfection Standard*), otherwise result is not causal.

A3: Causal Based on Scientific Criteria—Presumption that estimate is predominantly causal based on all scientific knowledge (aka *the Useful Approximation Standard*).

A2: Causal Mechanism—Typically some collection of spatiotemporally contiguous structures and processes along which a signal can be propagated from one entity to another resulting in a response.

A2.A: Mechanistic Element—Some functional structure, component, or property of a mechanism.

A2.B: Mechanistic Parameter—Value associated with some property of a transfer function where the value arises from underlying physical, chemical, biological, or behavioral features. For example, the maximum photosynthetic rate for a plant, by which I mean the inherent trait resulting from its biochemical machinery. These are to be conceptually distinguished from parameter estimates from statistical models, which typically rely on simplified representations such as independent random variables with random errors.

A3.C: Depth of Mechanistic Understanding—It should be recognized that characterizations of mechanisms range from superficial, as in statistical mediation relationships, to deep (an understanding of the machinery and processes whereby effects are propagated).

B1: Causal Statistical Methods—Methods of data pre-processing and analysis that attempt to isolate pure causal effect estimates (see Figure 1). Often statistical methods are called causal if they are capable of guaranteeing perfectly unbiased estimates as sample size goes to infinity.

B1.A: Causal Effect Estimate—Estimate of some observed manifestation of a causal relationship. We should be careful to not equate effect estimates with mechanistic parameters,

though there can be cases where those are equivalent.

B1.B: Causal in the General Scientific Sense—

A causal effect estimate between two entities (say X and Y) might be considered to be causal in the general scientific sense if there is reason to think that the estimated value is at least a useful approximation of the unbiased effect estimate. What constitutes a useful approximation will depend on the evidentiary standards being applied for the situation.

B1.C: Causal in the Statistical Sense—Causal statistical effect estimates are ones presumed to be pure and isolated counterfactual differences (usually between treated and untreated individuals, groups, or numeric categories). The purpose of such a strict requirement is to enable causal inferences to be drawn in the absence of adequate supporting knowledge.

C1: Causal Scientific Methods—Any scientific method that seeks to reveal the operation or characteristics of a causal mechanism.

C1.A: Association Studies—Analyses of data that seek to quantify conditional associations so as to isolate unbiased effect estimates.

C1.B: Perturbation Studies—Manipulative experiments and event studies where some abrupt change in driving conditions has taken place. Interpretations are clearest when the causes of the perturbation are exogenous to the study system (e.g., by random assignment) and measurements are made before and after the perturbations, though generally perturbations play a critical role as causal evidence because they can demonstrate responsiveness to change.

C1.C: Structure Investigation—Examination of the structures that serve as mechanistic elements and that contribute to the operation of a mechanism. Tree survival following cyclones can be causally influenced by the wood xylem density, for example.

C1.D: Process Investigation—Study of rate functions so as to determine potential responses to change.

C1.E: Theoretical Investigation—Hypothetical explorations of relationships among variables under different sets of assumptions. For ecological systems assumptions are often extrapolated from fundamental principles of mathematics, physics, chemistry, and biology.

D1: External Consistency—The general property of a causal claim being consistent with multiple

sources of evidence. Types of external consistency may include, (1) repeatability, (2) congruity (observations consistent with proposed mechanisms), (3) transportability.

D2: *Transportability*—The ability to extrapolate to a related but different situation. Transportability is a hallmark of causal mechanistic understanding.

E1: *Causal Proposition*—The suggestion that two variables or some system of variables may exhibit the manifestations expected of a causal process. This is often a beginning step in scientific inquiry or hypothesis testing.

E2: *Causal Claim*—The claim that a relationship exhibits the minimum qualifications for a causal relationship (e.g., conditional association, responsiveness to exogenous variations).

E3: *Causal Observation*—Causal claim with at least minimal support.

E4: *Causal Knowledge*—Accumulated evidence for some set of variables that indicates responsiveness to variations in presumed drivers, evidence related to a plausible mechanism for conveyance of effects and demonstrated external consistency.

E4.A: *Empirical Causal Knowledge*—The accumulation of replicated and thereby substantiated causal claims based on statistical studies without reliance on a plausible mechanistic hypothesis.

E4.B: *Mechanistic Causal Knowledge*—Characterization of a set of key components (e.g., physical entities) that interact in systematically predictable ways such that their collective behavior can be generalized to new situations. Extrapolability to new situations is a hallmark characteristic of mechanistic causal knowledge.

E4.C: *Causal Knowledge Diagram* A type of causal diagram for which there is a mechanistic basis for the links instead of a statistical/probabilistic basis. Note in Figure 4a that double-line arrows are used instead of standard single-line arrows to signify this feature.

E4.D: *Complex Cause*—Collection of causes whose individual contributions have not or cannot be disentangled or isolated. The collection will be generally expected to exhibit manifestations of an underlying mechanism at a minimum requirement.

E5: *Causal Understanding* The result of a sufficient accumulation of causal knowledge to explain the conditional behavior of a system.

F1: *Predictive Knowledge*—General term that simply means the ability to successfully predict

values in some sample other than the one used for estimating parameters. Predictive knowledge is not necessarily non-causal but may simply be a case whether the precise causal content has not yet been isolated.

F1.A: *Extrapolative Predictive Knowledge*—The ability to successfully predict values in some situation that differs in important ways from previous situations and samples of data. It is expected that often if not usually *extrapolative predictive knowledge* contains causal content.

G1: *Evidential Pluralism*—An epistemic approach to causal inquiry focused on the processes of establishing and assessing causal claims. Evidential Pluralism is distinctive in the foundational way it dictates a role for mechanistic studies separate from association studies, in establishing causal claims.

H1: *Causal Knowledge Analysis*—A new procedure proposed in this paper that involves an evaluation of existing knowledge related to some relationship of focal interest. Causal Knowledge Analysis involves the documentation of pre-existing evidence related to observed patterns of behavior, documented structures and processes comprising an underlying mechanism, and evaluation of the nature of the mechanistic elements (their reliability, the precision of their actions, and their potential transportability to other situations).

I1: *Integrative Causal Investigation Paradigm*—A representation of the Integrative Paradigm is depicted in Figure 4. It is distinct from other current formal approaches to causal investigation in (at least) three ways. First, it focuses on the fact that scientific investigations generally aspire to build causal knowledge and ultimately add enough mechanistic depth to achieve causal understanding. Second, it highlights a unique role for evidence from system perturbations separate from association studies in supporting causal interpretations. Third, applications of the Integrative Paradigm will typically require an additional enterprise, Causal Knowledge Analysis (as defined above), which is the characterization of preexisting causal knowledge about a relationship prior to conducting statistical analyses.

J1: *Integrated Causal Analysis*—When Causal Knowledge Analysis is combined with the analysis of new data and the preexisting knowledge informs the analyses of new data, it can be referred to as Integrated Causal Analysis.

Additional descriptors

Conditional—Situation where manifestations vary depending on factors that alter underlying mechanisms, either by altering parameter values or mechanistic structures and/or processes.

Common-Cause Explanation—Situation where some observed relationship can be largely or entirely explained by common causes. Within the Integrative Paradigm, deferring to common-cause explanations due to omitted variables requires explicit evidence providing a more convincing explanation for the observations.

Invariant/Stable—Situation where mechanistic elements are relatively fixed, such as freeze tolerance for a species. There can be variation in parameters caused by evolutionary history (genetic variation) or preconditioning (developmental history). However, relative to other mechanistic elements or chains, some properties are considered to be invariant. For example, the mechanistic performance of chronographs is quite invariant while the mechanistic performance of racehorses is much less so (see *Heterogeneity* below).

Heterogeneity—Common situation for biological elements where there are variations among individuals or other study units (contrast with *invariant* processes, above).

Strong Mechanisms—Ones that produce very reliable manifestations.

Weak Mechanisms—One that produce less consistent manifestations.

observe, and to anticipate how we might intervene to achieve desired outcomes. This avoidance of commitment to a single philosophical account of causation seeks to sidestep the problem of counter-examples, which commonly arises when adopting a metaphysical approach (Beebee et al., 2009, part II, chap. 7–11). A more detailed discussion of this subject can be found in Shan and Williamson (2023), sect. 5.

A causal epistemology such as Evidential Pluralism can be thought of as a set of principles that builds our confidence in causal beliefs as a function of the available supporting evidence. A distinctive feature of Evidential Pluralism is that it is not anticipated that there will be a single set of attending principles, guidelines, or rules that are universally ideal for all scientific circumstances. Rather, an optimal causal epistemology is one that best balances a set of desiderata (desired properties). These include being reliable, strong, stable, complete, and

feasible (Shan & Williamson, 2023, p. 59). Unlike most other descriptions of causal methodology, Evidential Pluralism attempts to avoid implying a monistic system that claims there is a single universal set of criteria (e.g., implying that only certain statistical techniques can lead to causal knowledge). The fallibility of monistic causal perspectives is well documented (Beebee et al., 2009).

In general terms, Evidential Pluralism can be described as a pluralistic account of causal methodology that places evidence of mechanisms on par with evidence of association. Evidential Pluralism therefore subsumes causal statistics as one form of causal methodology but not as a complete approach by itself. Further, Evidential Pluralism states that it is a desideratum of scientific inquiry that a causal relationship between two variables is not considered to be fully established without evidence supporting a plausible mechanism that can explain why influences are manifested under certain circumstances but not others. The Evidential Pluralism perspective can be interpreted as suggesting the need for an alternative paradigm to that described by the causal statistics literature (though Evidential Pluralism does not claim to represent a paradigm by itself). A pluralistic paradigm can be appropriate for ecology and other sciences that are built upon a substantial foundation of mechanistic knowledge, something that is not adequately addressed in causal statistics.

Evidential Pluralism's description of evidence of isolated associations

Evidential Pluralism recognizes the principles of modern causal statistics and the consequences of endogeneity arising from omitted common causes. Because it contends that evidence of mechanism is at least as important as evidence of isolated association, its position on the evidential standards for the associational approach is more liberal than the perfection standard depicted in Figure 1a. The specific advice Evidential Pluralism gives is to attempt to establish probabilistic dependence conditional on known confounders rather than all possible confounders, as many of the latter will be unknown and will go unmeasured. It further contends that exclusive dependence on associational evidence conditioned on all possible confounders leads to unrealistic skepticism about the prospects for establishing causal knowledge.

While not an element of Evidential Pluralism as described, it may be useful to elaborate on the subject of evidential standards here, since this is one of the undisclosed components of disagreement between statistical and scientific perspectives. In human discourse

and decision-making, it is axiomatic that there are different standards of evidence that apply to different situations, as dictated by circumstance, perceived risk of error, and consequences of approximation. Figure 3 attempts to represent gradations in two types of evidential requirements, the desired degree of certainty about concluding that a relationship is causal and the needed level of perfection of effect estimates.

The stated position of those who adhere closely to a causal statistics paradigm is that one needs to be assured of obtaining a pure causal estimate to have sufficient certainty about whether the relationship is causal according to scientific criteria (Figure 1a). A first point is that certainty does not depend on estimate purity when there is sufficient mechanistic knowledge. This will be explicitly demonstrated in the examples presented below. Shan and Williamson (2023) support such an idea by pointing out that confidence in the effectiveness of parachutes does not require randomized trials. Even if we recognize that parachutes can fail to function properly and there may be other causes of harm to skydivers (Barthel et al., 2023), the application of causal statistical methods is not needed to support a claim of causal effect. This simple example illustrates (1) the need for a pluralistic view of causal inquiry, (2) the value of mechanistic knowledge, and (3) the disconnect between estimation method and confidence in causal claims.

Part of the intent in presenting Figure 3 is to promote disclosure of the evidential standards that are used by methodologists and investigators. At present, this is not common practice and the standards behind methodological recommendations are not defended against counter-arguments. While causal statisticians may argue for

assured perfection of estimates and certainty of conclusions, scientists engaged in scientific investigations will often argue for the acceptance of useful approximations and a standard of preponderance of evidence. The formalities of decision science not only tell us that standards will vary from study to study but also that standards should be disclosed (Runge et al., 2020). Further, as the body of knowledge regarding a relationship accumulates, the community is climbing the pyramids of evidence as the research standards become more rigorous and the research community may become more strict as to when to accept new evidence. Allowing mechanistic knowledge to play a role in establishing causal claims engages scientists in the process of establishing evidential standards and dethrones causal statistics as the ultimate arbitrator of causal interpretations.

Evidential Pluralism's description of evidence of mechanisms

A very great deal has been written about basing causal inferences on statistical evidence and I will not attempt to characterize that vast body of literature here. In striking contrast, there has been much less explicit discussion of how one arrives at causal conclusions based on mechanistic evidence. Fortunately, several of the contributors to Evidential Pluralism have made a start at enumerating forms of mechanistic investigation and evidence through collaborations with the medical community (Clarke et al., 2014; Parkkinen et al., 2018). These efforts have yielded some general definitions that are broadly applicable.

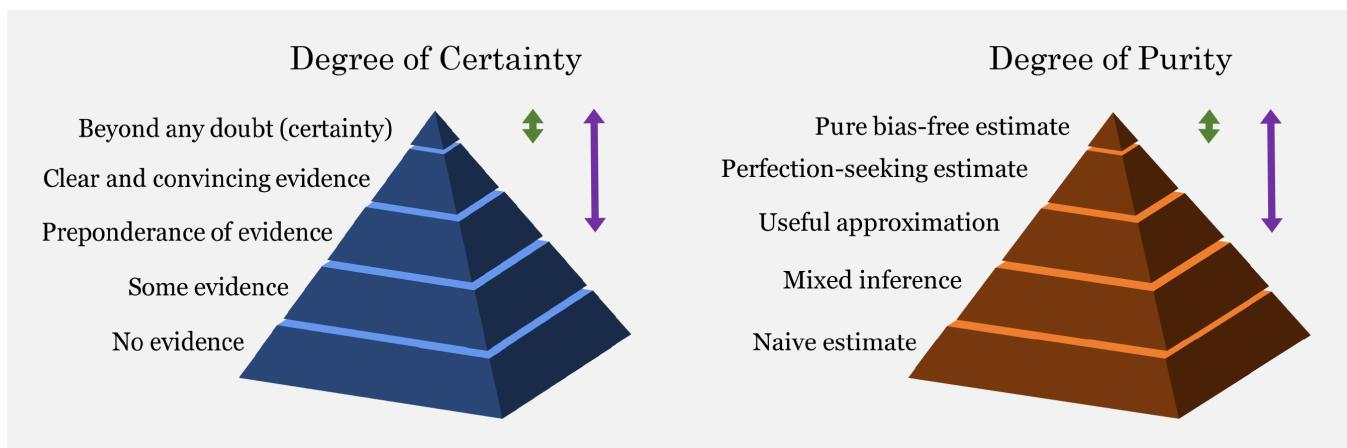


FIGURE 3 Representation of two types of evidential standards that influence discussions of causal evidence and knowledge. The small green arrows correspond to the standards often proposed for causal statistical methods while the larger purple arrows represent the standards associated with causal scientific investigations.

Parkkinen et al. (2018) offer the following definitions and explanations:

Mechanism – “... entities and activities organized in such a way that they are responsible for some phenomenon...”

Mechanistic Process – “... a spatiotemporally contiguous process along which a signal can be propagated.”

Mechanistic Study – “... one that seeks to shed light on features of mechanisms, including intermediary variables, entities, activities and the spatiotemporal organization of these entities and activities, as well as processes and their interactions.” They add, importantly, that, “Theoretical reasoning and reasoning by analogy can contribute here.”

To these I add an additional term,

Mechanistic Element – “A subcomponent of a compound mechanism, which can be seen as a link in a chain or network connecting causes of interest to responses of interest.”

The need to move beyond Evidential Pluralism

The domain of Evidential Pluralism is clearly defined in the following statement (from Shan & Williamson, 2023, p. 27). “Evidential Pluralism holds that establishing causation requires establishing the existence of a correlation and the existence of a mechanism, not necessarily the extent of the correlation, nor the details of the mechanism.” Scientists will need to go beyond Evidential Pluralism’s specified domain if we are to develop a methodological worldview that matches our ambitions, which is to build, across a series of studies, sufficient causal knowledge so as to develop a causal understanding. In the next major section, I will expand the objective to move past the determination as to whether a relationship is causal or not to the broader goal of building on our depth of causal knowledge and understanding, which is often an open-ended enterprise. Considering the characteristics of causal knowledge can help achieve that purpose.

THE INTEGRATIVE CAUSAL INVESTIGATION PARADIGM

A fundamental limitation to the ability to build causal knowledge using causal statistics is the exclusive focus it

places on critiquing individual studies and its reliance on individual datasets. This is, of course, reflective of the domain of statistics, which often focuses on the analysis of datasets and on critique of the inferences drawn from that limited body of evidence.

One of the most limiting aspects of causal methodology as it is described today is the implication that scientists are simply interested in establishing whether a relationship between two variables is causal or not and in estimating a net effect size. While the new data analysis techniques being developed for reducing bias in estimates are useful where they can be applied, the greater task of building causal knowledge remains.

Figure 4 presents a depiction of the Integrative Causal Investigation Paradigm. The Integrative Paradigm goes beyond Evidential Pluralism in three aspects. First, the Integrative Paradigm places focus on the fact that scientific investigations generally aspire to build causal knowledge and ultimately add enough mechanistic depth to achieve causal understanding. It also highlights a unique role for system perturbations as a means of obtaining evidence for an underlying machinery or mechanism. Beyond those differences, applications of the principles of the Integrative Paradigm will require an additional enterprise, which is the characterization of pre-existing causal knowledge about a relationship, prior to conducting statistical analyses, as described in the following two sections.

WHAT IS CAUSAL KNOWLEDGE?

Because the focus in this paper is on the enterprise of building causal knowledge, it is important that we consider the characteristic features of such knowledge. Since evidence of mechanism is one of the key characteristics, it is also important we consider how evidence of mechanisms can be recognized and its sufficiency evaluated. A practical approach to the assessment of preexisting causal knowledge is described in the next major section.

According to Johnson and Ahn (2017), *causal knowledge* refers to the accumulated evidence that supports our (1) explanations of phenomena and (2) expectations for how some phenomenon/system properties will behave in future situations. In this presentation, I emphasize knowledge related to mechanisms as the basis for building and characterizing causal knowledge. There are problems in ecology that may lend themselves to alternative approaches due to their complexity, such as the study of earth systems using convergent cross mapping (Gao et al., 2023). I will not however describe those approaches here, but simply recognize that those sources of evidence can be interpreted as fitting within the Integrative Paradigm.

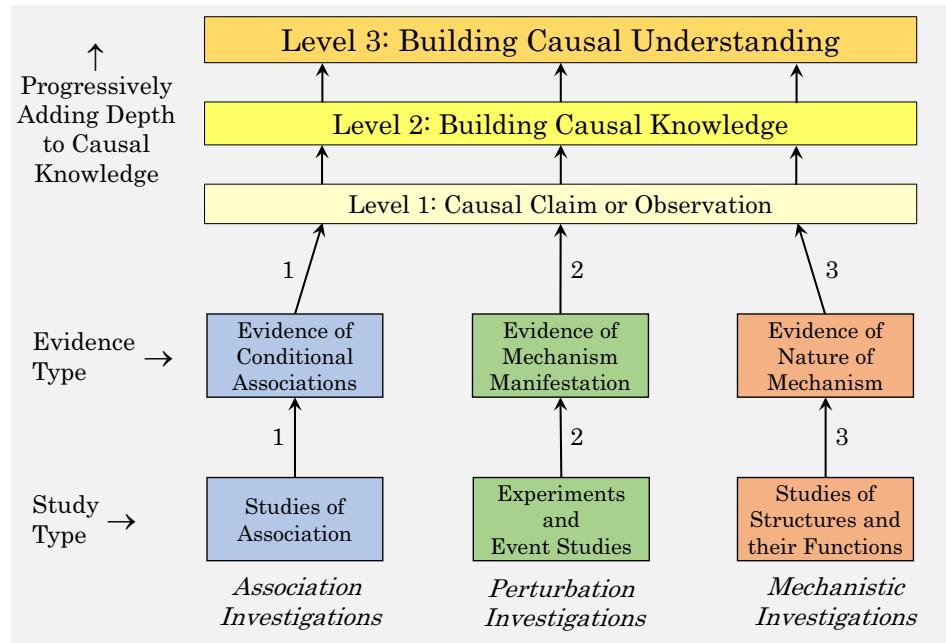


FIGURE 4 Representation of the Integrative Causal Investigation Paradigm. Two of the most important features of this paradigm meant to be conveyed here are (1) directing attention to the long-term goal of building causal knowledge and understanding across multiple studies and (2) recognizing the essential role of mechanistic investigations in establishing causal knowledge.

Elaborating on various definitions given earlier, a *causal mechanism* can be thought of as a set of components (e.g., physical structures as well as biological, chemical, and physical processes) that interact in systematically predictable ways such that their collective behavior can be generalized to new situations. Causal knowledge ranges from superficial to deep, where deep refers to detailed and reasonably complete knowledge of the essential component parts of a mechanism as well as the moderating forces controlling expression under differing circumstances.

While mechanistic knowledge forms the basis for causal knowledge, there has yet to be a systematic description of the nature of mechanistic causal knowledge incorporated into the causal methodology literature. Remedying that situation involves considering two topics, (1) the manifestations of causal mechanisms (how do we recognize that a mechanism is at work), and (2) the methodological steps for characterizing mechanisms. On this second topic, it should be recognized that methods for revealing and characterizing mechanisms vary greatly across science, and only select examples are feasible to present here.

Manifestations of causal mechanisms

Characteristics typically associated with causal knowledge (Box 2) can be seen as manifestations or expressions of underlying causal mechanisms. These characteristics

BOX 2 Some characteristics associated with causal knowledge

Basic characteristics

1. Indications of the propagation of variations from one system property to another
2. Plausible mechanism for the conveyance of effects

Advanced characteristics

1. External consistency with similar cases
2. Successful prediction for new and novel situations
3. Transportability
4. Diagnoses leading to successful remedies
5. Utility for achieving desired future outcomes

can be classified as basic ones typically expected in order to validate that a relationship qualifies as causal as well as more advanced characteristics reflective of deeper knowledge. Once again, this listing is neither complete nor prescriptive given that we can be confronted with a very wide array of situations in science. More tangible illustrations of such evidence will be provided when examples are presented below.

Methods for obtaining evidence of specific causal mechanisms

There exists an extraordinary array of mechanisms that operate in ecological systems, including those common to the parent fields of biology, chemistry, and physics. I provide a list of some general types of evidence in Table 1. I go on to discuss the topic further in conjunction with the examples presented below and encourage the reader to think of evidence types relevant for the systems they study.

Complex causal relationships and evidence limitation

Many of the patterns we observe in the world we presume to be the consequence of historical causal events and processes. However, there frequently exists a gap between causal hypotheses and the data available to assess them. The ideal situation is where precise measurements are made prior to, during, and consequent to some well-defined event. Causal statistics relies heavily on these requirements and the calculation of temporal differences to estimate causal effects. However, these requirements are overly restrictive for the enterprise of building causal knowledge, which relies on a multitude of sources of evidence pertaining to structures and

TABLE 1 Common sources of evidence of mechanisms (modified from Clarke et al., 2014).

Sources of evidence	Examples
Direct observation	Evidence of pathogen damage, plant remains in paleo soils, anatomical and physiological characterizations
Perturbations	Randomized manipulative experiments, before-after-control experiments, removal studies, natural perturbations, management actions
Established knowledge from physics, mechanics, chemistry, and biology	Biophysical relationships between body size and metabolism, biochemical requirements and tolerances, organismal traits, population dynamics
Confirmed theory	Resource-based competition models, logistic growth models, predator-prey models
Simulation results	Mechanistic and agent-based models

functions, observed effects, and the mechanistic processes producing observed temporal and spatial relationships.

A special case of data limitation is where all variables are observed simultaneously. Success in using such static data to learn about temporal processes depends on temporal consistency. If we use A_{T+1} to stand in for A_T , then we are assuming a high degree of correlation between variable values at the two time periods. Within the Integrative Paradigm, with our focus on building causal knowledge across studies through the investigation of mechanisms, the use of static data is not forbidden. However, the associated challenges can contribute serious bias to estimated effects in practice, which is not always considered. As with all causal investigations where ideal evidence is not available, a strong reliance on mechanistic knowledge is needed.

Beyond issues of limited measurement, an under-discussed challenge is where causes are complex or are emergent properties. There may be a collection of causal drivers and moderators whose influences may have occurred over a short or long span of time, and further, those influences may be intertwined. This is a common problem for studies that rely on spatially separated measurements. Environmental gradients, specifically, and spatial variations in general, presumably represent the historical results of causal processes. However, our ability to treat such information as if it is causal depends heavily on strong mechanistic knowledge. There are cases where adequate knowledge exists, but also many cases where it is insufficient. Complexity places limitations on the depth of our understanding, which can motivate further scientific investigation. Beyond that, explicit discussion of complex causes is needed, with the option of treating those as predictive relationships usable for statistical control.

What I refer to here as *complex causation* does not imply non-causal relationships. Rather, taking a long view suggests we may wish to interpret complex causal relationships as situations where scientists have not yet developed sufficient data and/or knowledge to provide a convincing explanation for how systems work. Methodological advances such as the analysis of environmental DNA, to give just one example out of thousands, allow us to obtain evidence not previously imagined, helping us to reduce unknowns and reduce the number of alternative explanations.

The hypothetical manipulation as a conceptual device

As described earlier, the Integrative Paradigm interprets causal claims as beliefs that allow us to make certain practical inferences, such as constructing explanations

for how observed patterns arise, predicting future responses to changed conditions, and anticipating how we might intervene to achieve specific outcomes. A practical approach to thinking about causal knowledge in this context involves the use of hypothetical manipulations. For example, if we claim *A* has a causal relationship with *B*, we suppose that variations induced in entity *A* might lead to changes in the values for entity *B*. The most frequently discussed cases of *non-causal relationships* involve associations resulting from common causes. If *A* has a causal effect on *B* and also on *C* but the latter two do not have causal effects on one another, then *B* and *C* may show association, but manipulations of *B* will not produce changes in *C* and changes in *C* will not produce changes in *A*. This is referred to as *common-cause association* (aka, a *spurious relationship*). Of course, there can be situations where there are no causal connections at all linking two variables, in which case we expect no persistent association. One goal of building causal knowledge is to understand the network of mechanisms leading to non-causal associations as well as those forming causal relationships. The process of building causal knowledge is expected to involve a series of approximations.

CAUSAL KNOWLEDGE ANALYSIS

If we accept that preexisting causal knowledge should be considered when engaging in causal statistical analyses, it implies the need for a pre-analysis step, which I refer to as *Causal Knowledge Analysis*. Causal Knowledge Analysis can be defined as an evaluation of existing knowledge related to some relationship or model that considers the properties listed in Box 2. As described above, evidence of causal knowledge can come from observed responses to perturbations, patterns of association that are indicative of an underlying mechanism, and direct evidence of mechanism itself. In Causal Knowledge Analysis, our long-term goal is to understand mechanisms so that we can understand when associations will manifest to various degrees in various situations. Our short-term goal is to inform decisions related to the statistical procedures to use when analyzing additional data (considered in the Implications section). In the demonstrations of Causal Knowledge Analysis that follow, I will focus on questions that relate to evidence indicating that some mechanism is in operation and on the details regarding the nature of the mechanism.

Because mechanistic knowledge lies in the domain of subject matter experts, Causal Knowledge Analysis is fundamentally different from statistical causal analysis, both in terms of approach but also in terms of the

expertise required to support or refute claims and assess evidence. This is a fundamental outcome of adopting the principles of the Integrative Paradigm.

In the following section, I consider three tangible examples. As the objective of this paper is to present foundational ideas for an integrative pluralistic perspective, my discussions of individual examples are deliberately brief and limited in scope. I anticipate that fully developed Knowledge Analyses will often resemble substantial reviews of evidence and evaluations of their general features, though certainly this will not always be necessary. For each example that follows I consider the question, “What are our expectations based on existing causal knowledge and what is the basis for those?” Box 3 provides a progression of questions we might ask when evaluating existing mechanistic causal knowledge. In the examples in the next section, I follow consideration of preexisting evidence with a brief consideration of a sample of data relevant to that question.

EXAMPLES

Example 1: Aviation fatalities

The first example I consider is a non-ecological one that should be accessible to readers regardless of background.

BOX 3 Possible progression of questions for a Causal Knowledge Analysis

1. What is the nature of the cause(s) of interest? the response(s) of interest?
2. Are there observed manifestations/observational patterns that suggest the existence of a mechanism connecting causes to responses?
3. Are there known or plausible mechanisms connecting the variables of interest?
4. Are there plausible competing explanations or mechanisms that can counteract the focal mechanism of interest?
5. How sufficient is our knowledge of conditional influences that may affect expressions of the mechanisms?
6. How reliable are the mechanisms and conditional influences based on available knowledge?
7. How exact is our knowledge of processes and associated parameters?
8. How transportable are the mechanisms to other cases or situations?

For this exercise, assume we are interested in understanding factors influencing aviation fatalities. Data are readily available for the relationship between the numbers of accidents and numbers of fatalities, so we will consider this relationship for our exposition of Causal Knowledge Analysis. For a fully developed Causal Knowledge Analysis we would conduct an in-depth review of the literature to compile relevant evidence. For this illustration I will simply rely on general knowledge and a few review articles to demonstrate the process.

Referring to Table 1, the most relevant types of knowledge for understanding this problem come from direct observation and from established knowledge from physics, chemistry, and biology. Applying this knowledge to the problem allows us to consider the questions posed in Box 3.

Regarding question 1 (the nature of cause and response)

The cause of interest for this example is aircraft accidents. Accidents are defined by the Bureau of Aviation Accidents Archive (baaa.com) as, “Any event where an aircraft suffered such damage from a crash or hard landing that it is not in a position to be used anymore and that it is removed from service.” The response of interest in this example is passenger deaths (including crew).

Regarding question 2 (manifestations indicative of a mechanism)

Our immediate confidence that there is a causal relationship comes from accidents being repeatedly followed by observable consequences. I classify this information as manifestations indicative of a mechanism.

Regarding question 3 (support for existence of a mechanistic causal chain)

In this presentation I introduce a novel graph type I describe as a *Causal Knowledge Diagram* (Figure 5a). A causal knowledge diagram can be considered to be a type of diagram for which there is a mechanistic basis for the links instead of a statistical/probabilistic basis. Note that double-line arrows are used instead of standard single-line arrows to signify this feature.

I consider here accidents that begin with an unsafe descent involving an intact aircraft. There could be accidents that begin with explosions or other events leading immediately to fatalities, which would suggest a modification of Figure 5a. Because of its role in survival of the

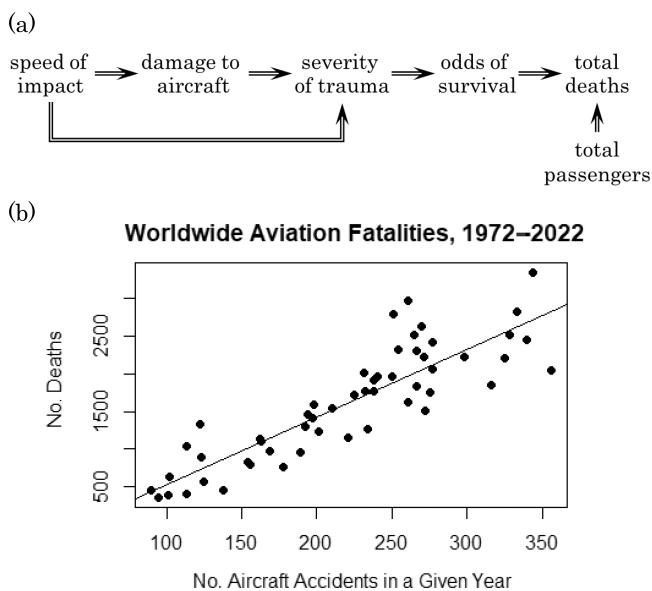


FIGURE 5 (a) Causal knowledge diagram representing core causal chain connecting the speed of an airplane crash to the number of fatalities that result. Note that the use of a double-line arrow is meant to signify that there is an established mechanistic basis for a relationship. (b) Annual summary data from the Bureau of Aviation Accidents Archive can be found in Broussard (2024).

type of accident I describe here, the first variable quantity of importance in the causal chain of events is the speed of impact (Figure 5a). The speed of impact has a direct influence on the resulting damage to aircraft and, in turn, will have direct consequences for the occupants of the aircraft, the physical trauma they experience, and their ultimate odds of survival. Other factors besides speed such as the type of aircraft and the terrain where the crash takes place can be of importance but are treated as a conditional factor and not shown in the key causal chain. Total deaths will then be a function of the odds of survival and total number of passengers involved in accidents.

As mentioned earlier, not all of the available variables that can explain statistical variance in parts of the causal chain will necessarily be characterized as representing causal knowledge. For example, odds of survival may vary depending on state or nation, which raises interesting questions about the reasons behind such associations. However, for such questions, further investigation can help to sort out the causal factors. Non-causal and complex causal information can be useful, as they can help us to begin to isolate individual causal influences.

Regarding question 4 (competing explanations)

The two general categories of competing explanations are alternative causal chains and common-cause explanations.

Opposing mechanisms are also important to consider in some situations. In the context of this example, the first alternative to consider is whether there is some other reason independent of physical trauma that could lead to death? Possibilities might include heart attack and other consequences of the stress of the situation. The second type of alternative explanation to consider is whether there is some common-cause variable which, for this example, would be some factor that influenced the speed of impact and the odds of death. Finally, there is, of course, a background death rate from natural causes unrelated to the event as well. This finer scale of explanation would require additional data, and while these are not expected to cause a substantial fraction of the deaths, a thorough analysis would perhaps consider such alternatives.

Regarding question 5 (sufficiency of knowledge of conditional influences)

All of the factors in the causal chain in Figure 5a are subject to various influences not shown in the diagram (e.g., Boyd, 2017; Ekman & Debacker, 2018; Toyama, 2006). For example, speed of impact is known to be influenced by the cause of the accident and at what altitude uncontrolled descent begins. Additional influences on aircraft damage include the physical features of the aircraft and the terrain at impact. Trauma experienced by individuals has been shown to be influenced by the precise features of the crash, especially including post-crash fires and explosions. Finally, odds of survival are additionally influenced by the responsiveness of rescue crews and medical personnel.

Regarding question 6 (anticipated reliability of the mechanism)

Reliability in this context refers to the consistency of outcome. Strictly speaking, reliability of mechanism is not required for the relationship to qualify as “causal” though it may influence our ability to arrive at confident determinations and precise estimates. Rather, we can see reliability as a useful property of our knowledge. Reliability as used here does not necessarily equate to exactness (considered in question 6) with the distinction having to do with long-term probabilities versus consistent short-term predictiveness.

Considering the related but separate topic of aviation safety, the reliability of safeguards against accidents is much lower for civilian non-commercial flights than for commercial aviation. Numerous studies have shown commercial aviation to be the safest mode of transportation in terms of accidents per miles traveled (Boyd, 2017).

However, the reliability for surviving an accident once one occurs is approximately 15%–20% for both types of aviation. Boyd (2017) reports accident and survival data for the United States and estimates that approximately 20% of the accidents involving civilian non-commercial aircraft result in fatalities. Ekman and Debacker (2018) report for US commercial accidents (1983 through 2000) an overall fatality rate of 14% and a fatality rate of 44% for serious accidents (those involving fire and substantial or total aircraft damage).

Regarding question 7 (exactness of processes and parameters)

There is tremendous variation in the proportion of deaths resulting from aircraft accidents across all situations. At this simple scale (accident = Yes or No), predicted survival rate is very inexact. Ekman and Debacker (2018) report, however, that as the severity of accidents increase from those with no damage to those where the aircraft is destroyed in the crash, risk of death becomes much more inevitable. It would be fair to say that survival rates are reasonably exact on the low and high ends of the severity of crash scale, with greatest variation when crashes involve substantial but not complete damage to aircraft.

Regarding question 8 (transportability of mechanism)

Transportability refers to the degree to which knowledge of mechanisms can be extrapolated from one accident to the next. The term, as used here, is meant to include extrapolation to another situation that differs in significant ways from the source of the knowledge. For example, we might ask how transportable the rate of survival estimate is from airline to airline, but also from commercial flight to military support operations, to ultra-lights, where aircraft and flight conditions are substantially different. There are of course many variables, but our focus might be on the core causal chain presented in Figure 5a since these properties would be common to many situations (while, for example, the factors leading to accidents or recovery operations would vary substantially). Upon consideration, it will be apparent that certain physical/mechanical processes are inherently transportable as are certain biological limits. These features provide our strongest opportunities for reliable predictive forecasting. Both transportable and non-transportable knowledge deserves to be highlighted in Causal Knowledge Analysis investigations.

Example data

Figure 5b plots data relating the number of worldwide fatalities from aviation incidences as a function of the number of accidents reported in the Bureau of Aviation Accidents Archives (2022) for the period of 1972–2022. Note that this relationship ignores counterfactual requirements of the sort expected in causal statistical analyses. In the immediately preceding presentation, I have considered the expectations for this relationship based on mechanistic knowledge. From that, we anticipate an increasing function, which we observe. A simple linear regression yields an estimate of approximately 9 deaths per accident. This summary represents an average over the sample and has no mechanistic or transportable meaning. Such is commonly the case for causal effect estimates. Considering individual components of the mechanisms involved can lead to more meaningful and transportable parameters, such as maximum speed of impact permitting aircraft integrity and speed of impact beyond which survival is unlikely for specific aircraft.

Example 2: Effects of grazer body size on nutrient return ratios in African savannas

In African savannas and other grazer-dominated systems, animals play an important role in regulating ecosystem biogeochemistry through their differential use of nutrients. This differential use varies with animal body size, which has important influences on plant and soil communities (Sitters et al., 2017). The second example presented considers the mechanistic causal knowledge underpinning the presumed effect of grazer body size on the ratios of nutrients returned to the soil. Unlike the aviation accidents example, here we rely on more specialized knowledge possessed by subject matter specialists.

Regarding question 1 (the nature of cause and response)

For this example the cause of interest is the average body size of animals that are grazing at a specific location, while the response of interest is the nitrogen to phosphorus ratio of their feces. Measured N:P of feces will be expected to be influenced by both the N:P ratio of the forage that animals consume as well as the animals' differential retention of phosphorus over nitrogen.

Regarding question 2 (manifestations indicative of a mechanism)

Literature on this topic proposes a general relationship for vertebrates based on mechanistic knowledge of animal physiology (Reynolds, 1977). Subsequent work related to our example has primarily sought to characterize manifestations based on the known mechanisms (Sitters et al., 2017). So, in this case, work has progressed from knowledge of mechanism to looking for evidence of manifestations consistent with presumed mechanism.

Regarding question 3 (support for existence of a mechanistic causal chain)

The stoichiometric ratios required by vertebrate animals can vary for a variety of reasons, including body size. Nitrogen is important for protein synthesis and its metabolic demand is expected to scale to body size with an allometric exponent of 0.75 (Darveau et al., 2002). However, bone is phosphorus rich and since skeletal investment must increase disproportionately with body size due to biophysical requirements, phosphorus requirements scale to body size with an exponent greater than 1.0 (Reynolds, 1977). The result is that larger animals will disproportionately retain more P, which will then be depleted in their excrement (causing N:P ratios to be higher). Panel (b) in Figure 6 provides a causal knowledge diagram representing the causal chain connecting body size to the N:P ratio of their excrement. The causal chain in Figure 6b represents a reasonably sufficient mechanism to convey an effect of body size on N:P output. That does not mean the operation of the mechanism is without variation. However, there is a very plausible basis for thinking that we will observe N:P ratios in feces to be causally influenced by animal body size.

Regarding question 4 (competing explanations)

No other directed mechanism by which animal body size would influence excrement N:P was found in my search of the literature, though the consideration of such may be a normal part of further scientific investigations. However, there is a real possibility that the average size of animals foraging in a location might be a response to vegetation type and indirectly to forage N:P. A complete analysis of the problem would include an examination of the associated data to see if these properties are correlated. If a correlation does exist, it can be controlled for

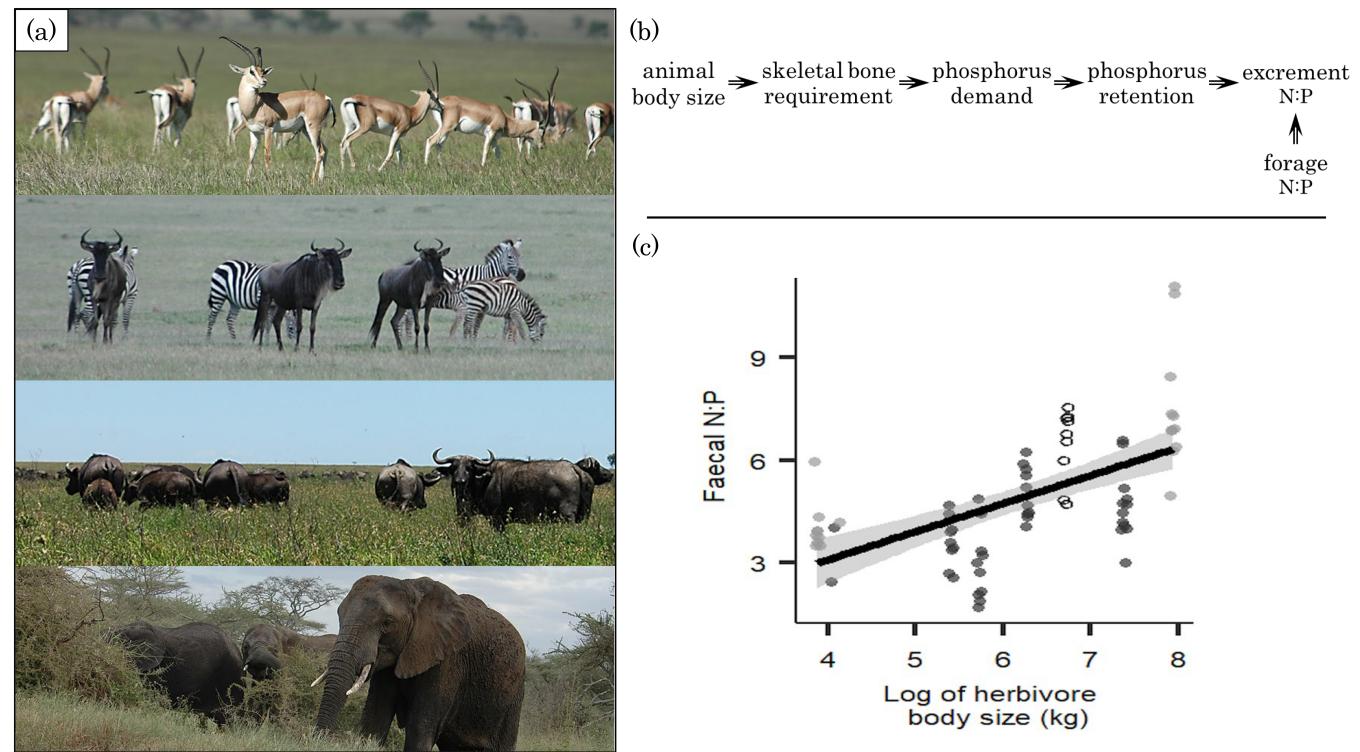


FIGURE 6 (a) Photos of grazers of various body sizes common to African savannas (photos by James B. Grace). (b) Causal Knowledge Diagram for the example. (c) Data plot (from Le Roux et al., 2020, fig. 2, with permission).

in any new analyses conducted. This does not guarantee that there are no other common-cause explanations. That said, within the philosophy of the Integrative Paradigm, the most important question is whether omitted common-cause factors can fully explain the observed relationship between body size and excrement N:P (refer back to Figure 1 for context).

Regarding question 5 (sufficiency of knowledge of conditional influences)

Current knowledge of the system suggests that for Serengeti grazers the conditional influences are not as well understood or quantified as is the core causal chain. An obvious conditional variable of substantial importance is the N:P of the food consumed. African savannas exhibit substantial spatial variation in plant communities for a variety of reasons. Major gradients in precipitation, soils, and topography are prominent features of the ecosystem (Anderson et al., 2007). Wildfires also play an important role in regulating nutrients and plant species. Finally, individual animal species have different feeding preferences, which further influences the quantities and ratios of nutrients consumed. As a result, a key variable serving as a conditional influence is the N:P of

forage consumed. Aside from that, it is not clear whether there is a great deal of precise knowledge available on other conditional influences on the core causal chain, namely, animal age and health.

Regarding question 6 (anticipated reliability of the mechanism)

All of the links in the causal chain in Figure 6b are strongly controlled by animal biology. The increasing requirement for skeletal bone with body size is based on fundamental principles of biophysics while the demand for phosphorus, a key component of bone, and its retention are controlled by fundamental principles of metabolism. The biological processes appear to be reliable, though the precise N:P found in feces under field conditions will be less reliable.

Regarding question 7 (exactness of processes and parameters)

Exactness of parameters seems to be largely unknown, though a thorough investigation of the literature might shed more light on this matter.

Regarding question 8 (transportability of mechanism)

Strong transportability for the discussed mechanism has been demonstrated for a wide range of animals and situations by Sitters et al. (2017), Subalusky and Post (2019), and Veldhuis et al. (2018). This confirms the general principle that biophysical traits of organisms provide highly transportable causal knowledge.

Example data

Figure 6c reproduces one of the figures published by Le Roux et al. (2020), who examined study plots in savanna vegetation in a protected park in South Africa frequented by a wide variety of ungulate grazer species varying in size. The data presented represent the nitrogen to phosphorus ratio of ungulate feces deposited and the estimated average body size of ungulates that visited those plots (based on automatic cameras) over a 10-month period. A general trend consistent with mechanistic expectation was reported. The slope of the relationship appears to fall within the range of expectations as well. Substantial scatter was observed, which suggests merit for additional studies to determine how much of the unexplained variation is due to variations in the N:P ratio of forage versus animal processing versus measurement error.

Example 3: Effects of community biomass on plant species richness in grasslands

With this example we transition to a more complex situation where we consider how the diversity of plant species per unit area in a grassland may be influenced by the amount of total accumulated biomass. Complexity results from several factors; (1) there are thousands of grassland plant species worldwide, (2) virtually, there is an infinite number of species combinations in space and time around the globe, and (3) both community biomass and species richness are community summary variables influenced by a great number of processes, including other trophic levels. Either despite or because of this complexity, ecologists have been fascinated by this challenge for the past century or more. There exists a vast and sprawling empirical and experimental literature but also a large number of contrasting theoretical models, which collectively make a thorough Causal Knowledge Analysis a very large task. Only a superficial attempt can be made here, and as before, the purpose is simply to demonstrate Causal Knowledge Analysis by example.

That said, I think this kind of example illustrates the relevance of building causal knowledge in order to pursue a causal understanding as opposed to estimating a single statistical causal effect that ignores the tremendous conditionality caused by the known processes.

The causal knowledge diagram presented in Figure 7a is adapted from a dynamic model developed in Grace (1999). It is also informed by numerous data-modeling exercises, including one that sought to reconcile a number of competing hypotheses based on theoretical models (Grace et al., 2016).

The processes in Figure 7a leading from biomass to richness predominantly reflect various forms of resource competition. Generalizations about those processes that follow are oversimplifications; nonetheless they reflect mechanisms common to many grassland communities, and their influence is often observed following perturbations and in experiments.

The arrows emanating from community biomass represent known mechanisms of resource reduction. For higher plants these include (1) shading effects that block sunlight, change light spectral quality, influence moisture retention, and dampen soil temperature fluctuations as well as (2) uptake processes that remove water and nutrients from the soil. There are many other possible types of effects of plants on their above- and belowground environments and a vast literature describing mechanistic elements. Here, I treat those additional processes as conditional influences outside of the realm of our discussion. Resource reductions connect to species richness through influences on the recruitment of new species and loss of established species. Substantial recognition has been given to species local extinction due to light reduction from neighbors (e.g., Borer et al., 2014; Hautier et al., 2009). Less attention has been given to the role of reduced light penetration on the maintenance of dormancy of live seeds (i.e., “cryptic competition”—Jutila & Grace, 2002), though this is a well-known process. Species represented in the seed bank, although they are alive, do not get counted as part of the diversity until they have germinated and have survived long enough to be observed. A great fraction of the dynamics of species diversity in grasslands is regulated by interstitial species whose persistence is aided by storage in the seedbank for substantial periods.

Regarding question 1 (the nature of cause and response)

For this example the cause of interest is the standing biomass (including plant litter). This is but one of several causes of interest to investigators and is selected here for

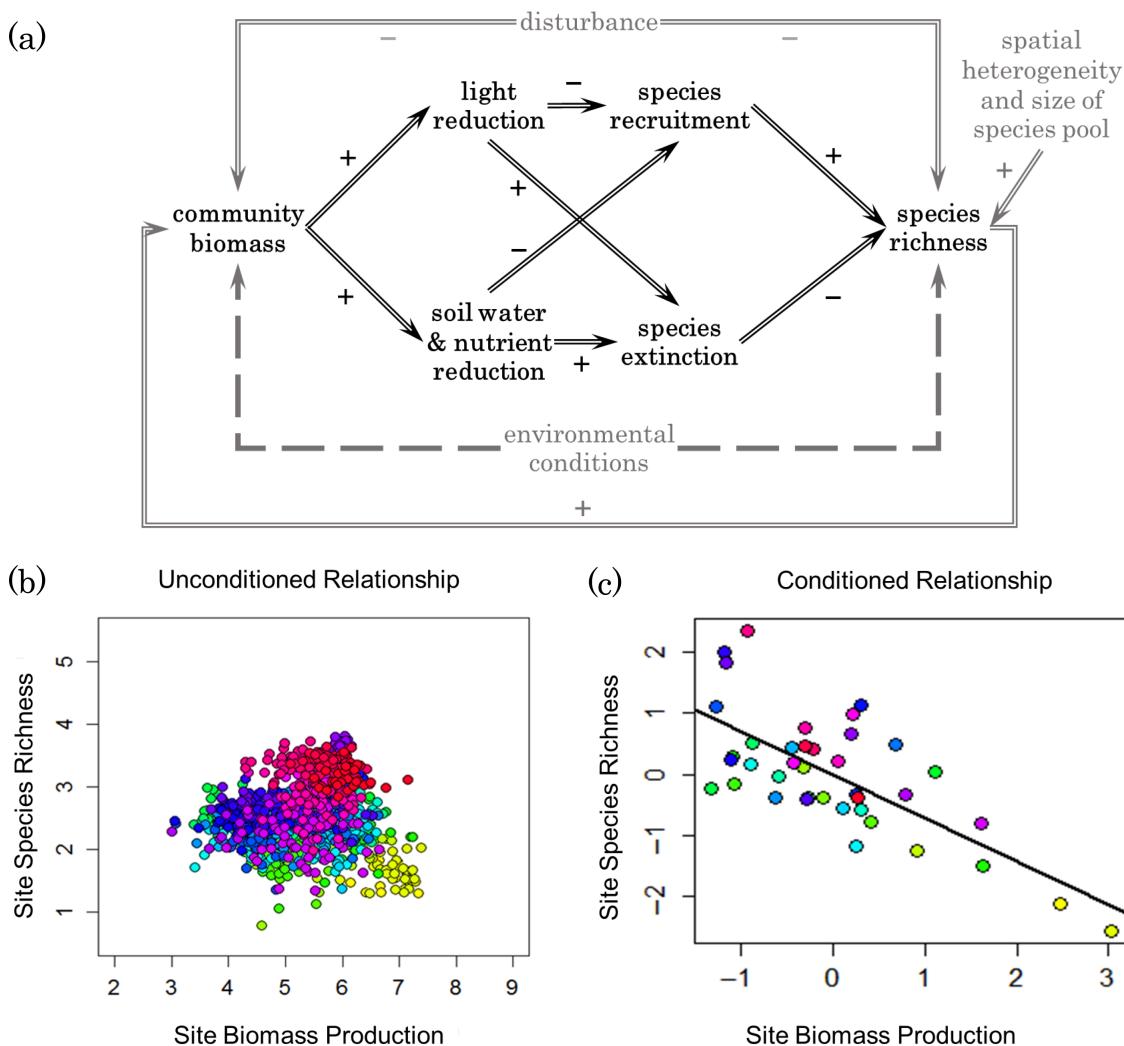


FIGURE 7 (a) Causal Knowledge Diagram representing known connections between community biomass and species richness in grasslands. Note that the long-dashed arrows emanating from environmental conditions signify complex predictive effects as defined in this paper (refer to Box 3). Gray text and arrows represent pathways that had to be controlled for in order to produce the plot in subfigure (c). (b) Unconditioned (raw) plot of annual biomass accumulation (log scale) versus species richness (also log scale) from Grace et al. (2016). (c) Isolated relationship for the same variables from the same study.

practical reasons. Aside from the sources of complexity listed above, it is known that causal effects can emanate from the shading produced by above-ground living and dead biomass, the abundance of fine roots in the soil, the rate of update of water and nutrients, physical and chemical effects from both of those sources, a wide variety of indirect effects mediated through other trophic levels, and seasonal/temporal variations in all of those. The importance of these several sources of causal influence can vary from situation to situation as well. The response of interest selected for this example is the number of plant species observed during an observation period per unit area of ground. Other components of diversity have also been considered in the literature.

Regarding question 2 (manifestations indicative of a mechanism)

In a review of the literature, Grace et al. (2016) summarized results from 30 published experimental field reductions of biomass. The results indicate that the great majority of results have shown increases in species richness as a result of biomass reductions. They further show that studies that have added dead plant material have observed reductions in species richness consistent with the mechanism. Considering preexisting evidence more generally, conservation management in grasslands worldwide relies heavily on the management of biomass through periodic grazing, mowing, and burning so as to reduce competitive pressures. This perturbation evidence

provides convincing evidence for a responsive mechanism (see also Borer et al., 2014).

Regarding question 3 (support for existence of a mechanistic causal chain)

In addition to ecological studies, agronomists and other plant scientists have extensively studied competitive and other interactions among plants for more than a century because of its importance for crop production. Competitive effects of community biomass on species richness can occur and for select species a great deal is known about mechanisms of interaction. In ecology, however, sufficient information to distinguish between the various mechanisms is rarely achieved except in tightly controlled experimental setting. As a result, we can expect that only strong and reliable mechanisms will be consistently observed across a broad sample of grasslands.

Regarding question 4 (competing explanations)

The complexity of this problem is such that evidence suggesting competing explanations deserves serious consideration. The mechanism represented in Figure 7a is certainly incomplete, and evidence for other directed mechanisms could be enumerated. The same can be said for the possibility of additional common-cause influences. If one were to adopt the perfection standard, it would be difficult to proceed with this problem. However, if the standard of reasonable approximation based on weight of evidence is adopted, the question becomes whether there is sufficient evidence to support some other explanation for the results in Figure 7c involving unmeasured common causes. Because our goal is to build causal knowledge and our confidence in explanations across studies, fear of some undescribed potential confounding is replaced by assessing potential models based on internal and external consistency with the full body of evidence.

Regarding question 5 (sufficiency of knowledge of conditional influences)

Following from the narrative for question 2, there are a great number of conditional factors stemming from variations in community composition and environmental conditions as well as the history of disturbances for individual sites. Any general patterns will have to be

enforced by the most consistent processes, which may well be the effects of shading on the survival of subordinate species and on impaired recruitment due to enforced dormancy.

Regarding question 6 (anticipated reliability of the mechanism)

The literature supports that at the qualitative level, competitive mechanisms are reliable. There have been some suggestions for European grasslands that there are specific ranges of standing biomass where competitive exclusion starts to be observed (Grime, 2006). Such observations are still somewhat disconnected from thresholds in resource levels that would represent truly mechanistic knowledge. Aside from that, the great variety of types of grassland and environmental settings means effects vary. In particular, it is likely that arid grasslands that are strongly limited by water availability and desiccation may involve more below-ground interactions and even offsetting facilitation effects. Nonetheless, there is sufficient preexisting evidence to support the general expectation that the abundance of community biomass can have a negative causal effect on species richness in many cases.

Regarding question 7 (exactness of processes and parameters)

The above discussion suggests that there will be substantial variation in the magnitudes of processes connecting biomass to richness. However, there are certain physiological limits that are common to many grassland species, such as minimum survival and germination resource requirements. These biological requirements suggest that in principle certain effects may be relatively predictable for portions of the total range of grasslands, but across all grassland only general patterns are likely to manifest.

Regarding question 8 (transportability of mechanism)

If we do not focus on the great variety of circumstances, the biological requirements that participate in a competitive effect of biomass on richness should be fairly general. Transportability is likely not the key factor controlling the predictability of this system, but instead the complexity of the problem and the difficulties associated with obtaining sufficient data.

Example data

Figure 7b,c reproduce results published by Grace et al. (2016). This study was inspired by a long ongoing debate over competing theoretical ideas about community assembly. Resolution of the debate was at a standstill at the time because an attempt to reason about the importance of various processes in natural systems while relying largely on bivariate relationships (Figure 7b). Grace et al. (2016) used a global dataset and structural equation modeling to control for known and suspected confounding, which yielded the relationship shown in Figure 7c. This isolated relationship corresponds qualitatively to expectations derived from the causal knowledge chain. More detailed quantitative comparisons to other studies could help to ascertain the degree of reliability of the shape of the relationship. Compared with the first two examples presented, this example is far more complex and the business of Causal Knowledge Analysis is clearly unfinished.

IMPLICATIONS OF THE INTEGRATIVE CAUSAL INVESTIGATION PARADIGM

Potential for advancing ecology

Ecologists have a long tradition of experimentation (Scheiner & Gurevitch, 2001). With a few exceptions the approaches used have been adopted from other fields, especially agriculture, and reflect the norms from experimental statistics. The inadequacy of these standard approaches for investigating systems of variables has been noted in a few cases (Cottingham et al., 2005; Grace et al., 2009). Nonetheless, a great deal of our accumulated causal knowledge is derived either directly or indirectly from experiments. The natural sciences have had a great advantage over the human sciences with regard to the ability to implement random assignment. Modern causal statistics (e.g., Imbens & Rubin, 2015) was primarily born out of the challenges faced in human randomized trials, where individual subjects are both seldom representative of the general population and comparability in outcomes among treatment groups is frequently problematic. There is important knowledge from that source ecologists would benefit from knowing (e.g., Kimmel et al., 2021).

Ecologists also have a long history of pursuing causal understanding by invoking mechanistic connections in ecological systems. Pickett et al. (2007) have argued for causal explanation as a general aspiration. Holt (2015) succinctly summarizes an approach known as “inference

towards the best explanation” that seeks coherent causal explanations based on weighing all forms of evidence. Scheiner and Willig (2008) echo a similar sentiment while describing a theory as, “A framework or system of concepts and propositions that provides causal explanations of phenomena within a particular domain.” DeAngelis and Mooij (2003) argue generally for the merits of mechanistic models, while Hone et al. (2023) provide a specific context for arguing for reliance on a combination of evidence types as causal criteria for wildlife management. Dade et al. (2019) have argued for explicit identification of the drivers of trade-offs and synergies in the controls of ecosystem services through the use of causal inference and process-based models. Perhaps one of the most promising approaches to causal understanding is reliance on organismal traits as stable (and thereby transportable) mechanisms supporting causal interpretations (e.g., Funk et al., 2017; Laughlin, 2023; McGill et al., 2006). While ecologists have indeed articulated rationale for arriving at causal inferences, the form of the arguments may be insufficient to dispute the claims of causal statistics that there can be no omitted confounding variables that can contribute bias to estimates. Thus, these prior efforts leave unresolved the age-old conflict between statistics and the natural sciences over requirements for causal inference.

It may be that some ecologists are comfortable with continued reliance on expert knowledge to justify the use of surrogate causal language and de facto make causal inferences. However, advocates of causal statistics are now challenging such approaches in the field of ecology (e.g., Arif & MacNeil, 2022; Ferraro et al., 2019). It may also be that some ecologists will not consider the complex set of rules and restrictions of causal statistics. The goal of this paper is to clarify certain issues and provide some capacity for broader conversations about what constitutes evidence of causal knowledge in the natural sciences. Additionally, this work can support ecologists’ central role in building explicitly causal knowledge by providing some ideas for how this might be approached.

Potential for refining causal investigation methodology

This paper provides a very distilled and incomplete characterization of causal statistics and focuses more on what it does not do well than on its strengths. A great deal of progress has been made in this area in the past 25 years. The goal of this paper is to motivate improvements rather than criticize past efforts. The work presented is in response to an influx of ideas developed in other fields, as well as an explosion in quantitative

approaches to causal inference, which in turn is motivated by rapid developments in artificial intelligence. The arguments and examples presented in this paper suggest areas for refinement of the existing advice given for causal inference.

Shifting our attention to the long-term goal of building causal knowledge demands that we recognize the preexisting causal knowledge and consider what is known in subsequent analyses. This paper presents numerous examples where it can be argued that causal inferences can be made based on mechanistic knowledge. This will not be found to be some rare situation in ecology, but simply a product of centuries of scientific investigation in the natural sciences. The mechanistic support for interpreting new evidence will be of various sorts, however, which suggests adoption of a pluralistic perspective. There has been a persistent interest within causal statistics in proposing universal guidance for causal inference, resulting in oversimplifications that may impede progress. I believe that developing more extensive terminology and adopting a pluralistic perspective can help to clarify the path forward.

Potential for advancing causal knowledge

I believe Causal Knowledge Analysis can serve as an important step in causal investigations. Such a practice can have numerous benefits for refining our understanding of existing knowledge and for strengthening the process of causal inquiry. This presumption has relevance for causal statistical analysis, structural equation modeling (e.g., Grace, 2006; Shipley, 2000, 2016), and mechanistic modeling. At present, if an assessment of preexisting causal knowledge is conducted, this is done informally and without thorough consideration of the properties of manifestations or of mechanistic elements. A more thorough evaluation has the potential benefit of steering us to avoid ignoring what we (collectively) do know because of a preoccupation with what the analyst does not know (another consequence of accepting the perfection standard). Going further, the possibility that a relationship is already known to be causal should be taken into account, though it is typically not mentioned. Knowing that a relationship of interest is causal can shift our objective to refining that knowledge. Clearly, wildfires have a causal effect on forests, but there is much to learn about the mechanistic elements of that process. One may wish to obtain a pure estimate of the net effect, but often we will wish to advance our understanding of the structures and functions that comprise the mechanisms in operation. How much a pure causal effect estimate actually tells us depends on the nature of

the associated mechanism. If the mechanism is complex and highly conditional, a pure causal effect estimate only serves as an observation. Perhaps many such observations can lead us to a greater understanding, but study of the underlying properties and processes controlling the forms of expression may be the greater prize. Beyond that, preexisting causal knowledge can allow investigators to relax restrictive requirements as appropriate to the situation and avoid proposing invariant standards that restrict the evidence to be considered. The business of evaluating causal models based on their consistency with empirical data can often be justified, especially for the case where one is working towards a deeper understanding.

The Integrative Paradigm has another implication, which is that scientific experts can play a more central and active role in judging the standards for causal inferences. The current situation is one where causal statisticians (or their equivalent) propose to be authorities for defining the limits of defensible causal interpretations. This has produced a long history of critique that is only defensible if one presumes that there is no mechanistic understanding of the phenomenon under investigation. Focus on select examples involving human behavior and especially challenging problems along with the lack of a pluralistic perspective such as the Integrative Paradigm may have contributed to the situation. Accepting the Integrative Paradigm as a useful framework represents a shift in the current status quo that affords the opportunity to move to a position that recognizes that all available approaches may have something important to contribute, but none have a monopoly on the rules of evidence and standards for inference. Debates over the superiority of individual approaches could dissipate and the potential for progress towards causal understanding could advance as a consequence. One side effect may be that the routes to causal knowledge will increase in proportion to the variety of approaches applied in scientific investigations—one set of guidelines will not apply for all situations.

Potential for contributing to predictive forecasting

A final implication for adopting the Integrative Paradigm relates to the emerging interest in predictive ecology (Clark et al., 2001; Dietze, 2017; Houlahan et al., 2017; Mouquet et al., 2015). Interest in predictive ecology has shed light on the issue of transportability (Yates et al., 2018), which is a fundamental element of causal knowledge dependent on mechanistic understanding. At present the issue of prediction is often discussed without

explicit reference to causal criteria. Causal Knowledge Analysis is particularly relevant for forecasting because of the potential for a deeper understanding of the mechanistic elements controlling predictability of phenomena. The prospects for successful prediction are heavily influenced by the nature of the mechanisms involved. Predictive ecologists have begun to discuss more thoroughly the issue of transportability (Yates et al., 2018). As this paper suggests, invariant mechanisms, including constraints, provide fertile bases for research to support forecasting. This paper seeks to encourage a more systematic accounting of evidence in support of documenting established causal knowledge. This documentation can serve multiple purposes, including as an aid to causal statistical analyses but also as an advent to understanding the predictability of various phenomena and the needed information to enhance prediction.

ON PARADIGMS, EPISTEMIC SYSTEMS, EXCEPTIONS, AND THE ROLE OF EXAMPLES

This paper refers to integrative causal investigation as a *paradigm*, which implies certain things. The use of this term is meant to conform to Kuhn's (2012) treatment of the subject and equates to a general worldview. The Integrative Paradigm as a distinct concept proposes the general worldview that causal understanding can be arrived at from statistical and mechanistic investigations. Aside from generality, another property of scientific paradigms described by Kuhn is the expectation that the concept will promote further evolution and refinement and is not an immutable prescription, which can of course be replaced by a slightly different prescription. Certainly, the general idea is ancient though it has taken the work of philosophers to remind us of the role of mechanisms in causal understanding. Causal Knowledge Analysis as presented has more of the properties of a prescribed list of criteria (e.g., Table 1 and Box 3) which should not be referred to as a paradigm.

Philosophical discussions of causality are importantly influenced by counter-examples. A motivation for emphasizing that the Integrative Paradigm is a pluralistic system is that it attempts to avoid dogmatism. The possibility that functional causal knowledge can be constructed without mechanistic understanding is certainly an accepted possibility within the Integrative Paradigm. As the capabilities of causal search methods and artificial intelligence develop, it is entirely possible that sufficient empirical search may lead to a level of prediction that goes beyond that possible by mechanistic explanation. The question remains as to whether such capabilities

will enable diagnostic evaluations and novel interventions, but at this point such possibilities cannot be ruled out. What we can be sure of is that a variety of methods will ultimately contribute to our progress in developing a causal understanding of ecological systems.

This paper represents a bare beginning towards advancing causal inquiry. One potential outcome is for closer attention to be paid to the enterprise of building causal knowledge and understanding. Future development might focus on analysis of a wide range of specific examples. General commonalities may be identified while new types of evidence and interpretations are likely to arise as more examples are evaluated.

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CONFLICT OF INTEREST STATEMENT

The author declares no conflicts of interest.

DATA AVAILABILITY STATEMENT

Data from Broussard (2024) were used for Figure 5 and these data are available in figshare at <https://doi.org/10.6084/m9.figshare.25999003.v1>.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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