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Assessing Interactive Causal Influence

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The discovery of conjunctive causes—factors that act in concert to produce or prevent an effect—has been explained by purely covariational theories. Such theories assume that concomitant variations in observable events directly license causal inferences, without postulating the existence of unobservable causal relations. This article discusses problems with these theories, proposes a causal-power theory that overcomes the problems, and reports empirical evidence favoring the new theory. Unlike earlier models, the new theory derives (a) the conditions under which covariation implies conjunctive causation and (b) functions relating observable events to unobservable conjunctive causal strength. This psychological theory, which concerns simple cases involving 2 binary candidate causes and a binary effect, raises questions about normative statistics for testing causal hypotheses regarding categorical data resulting from discrete variables.

A single causal factor is often perceived as contributing toward producing an effect yet insufficient to produce it on its own. Low body resistance in the absence of a flu virus is not by itself sufficient to cause one to have the flu; neither, typically, is the presence of a flu virus per se. The two in conjunction, however, often do cause one to come down with the flu. Likewise, striking a match per se does not cause it to light—there must be oxygen in the environment, the match must be combustible, and so forth. Cigarette smoke per se does not cause lung cancer—the smoke must be inhaled over a relatively long interval, the smoker must be susceptible to the disease, and so forth. The susceptibility itself is probably in turn specified by multiple genetic factors. Hard work alone typically does not produce success; it must be combined with talent and opportunity. Most causes in the real world, like these examples, are complex, involving a *conjunction* of factors acting in concert, rather than *simple*, involving a single factor acting alone. How do reasoners come to know that there is something special about the conjunction of several factors such that it can produce or prevent an effect?

We first present some phenomena that are inexplicable by previous psychological accounts of conjunctive causation. To explain these phenomena, as well as to solve other problems that beset previous accounts, we propose our *causal-power* theory of the assessment of interactive causal influence. We then review previous findings in the literature in light of our new theory and report new empirical evidence in support of the theory. Finally, we discuss some implications of our approach for the normative testing of causal hypotheses regarding data resulting from discrete variables.

Our new theory, like many previous psychological accounts of conjunctive causation (Cheng & Novick, 1990, 1992; Forsterling, 1989; Hewstone & Jaspars, 1987; Hilton & Slugoski, 1986; Kelley, 1967), is covariational in that it bases causal inferences on concomitant variations in observed events as well as on other observable features such as temporal ordering. Previous accounts, however, are *purely covariational* in that they do not consider the possible existence of unobservable causal structures to arrive at their output. In contrast, our theory explicitly incorporates into its inference procedure the possible existence of *distal* causal structures: Structures in the world that exist independently of one's observations. More specifically, in our theory we hypothesized that reasoners estimate the theoretical probability with which candidate causes both individually and in conjunction influence the effect in question, by producing it or preventing it. We call this theoretical probability the *causal power* of the (simple or conjunctive) candidate (after Cartwright, 1989). Our new approach allowed us to derive the conditions under which observed covariation implies conjunctive causation as well as the functions relating covariation to *conjunctive causal power*.

Scope of Our Theory

This article concerns the discovery of conjunctive causal relations in which (a) candidate causes and effects are represented by binary variables or by other types of discrete variables that can be recoded into that form, (b) the binary effects occur in a set of distinct entities (e.g., lung cancer occurs or not in each patient in a group, college admission is granted or not to each student who has applied, pregnancy occurs or not in a woman on each of a

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number of occasions), and (c) the binary candidate causes have two values (the presence and absence of a factor) that respectively indicate a potentially causal state and a noncausal state (e.g., the presence of asbestos in the air potentially causing lung cancer but the absence of asbestos not causing lung cancer or anything else). One might note that people often speak of the absence of something as a cause. Our interpretation of a statement such as "the absence of nicotine causes John to have withdrawal symptoms" is that it is the presence of nicotine that prevents John from having withdrawal symptoms that otherwise would occur; the absence of nicotine merely fails to prevent those symptoms, but it does not cause them.

For simplicity, we limit our analysis to (a) conjunctive causes that are composed of only two factors (e.g., presence of a flu virus and a weakened immune system) and (b) situations in which all unobserved causes of a target effect other than the candidate causes may produce or generate (we use these terms interchangeably) the effect but do not prevent it. The estimate of the simple generative power of a candidate is overly conservative if an unobserved preventive cause is occurring in the context or background (Cheng, 2000; Glymour, 1998, 2001), complicating the evaluation of interactive influence. All analyses presented here address the estimation of causal strength (i.e., the magnitude of a causal relation, including a magnitude of zero for a noncausal relation). We separate this issue from the assessment of confidence in an estimate (i.e., statistical reliability) because the latter is not specific to causal judgments. To avoid a conflation of the two concepts, we restrict our discussion to the assessment of causal strength in situations in which confidence can be assumed and is therefore not an issue. This article is consistent with, but does not address, the influence of prior causal knowledge on subsequent causal judgments (for normative accounts of this process, see Glymour, 2000, 2001; Pearl, 2000; Spirtes, Glymour, & Scheines, 2000; Tenenbaum & Griffiths, 2001, 2003; for recent psychological studies, see Carmichael & Hayes, 2001; Lien & Cheng, 2000). That is, we are concerned with the issue of how an acquired conjunctive causal relation is discovered.

Our causal-power approach makes explicit some issues regarding causal induction that have not been recognized before: Is the presence of something conceived as causal in a way that its absence is not, with the latter being causal only as a way of speaking? When there is an unobserved preventive cause of the target effect in the context, is the assessment of interactive influence possible? Should measures of conjunctive causation, unlike their current statistical renditions, differ depending on (a) the causal direction (generative vs. preventive) of the constituent simple causes and (b) the causal direction of the interactive influence being assessed? Moreover, should such measures differ depending on whether unobserved causes of the same effect interact with the simple causes? If so, are there ever circumstances under which interactive influence can be assessed?

Conventional statistical measures of independence for categorical data—those covered in popular textbooks (e.g., Freedman, Pisani, & Purves, 1998; Hays, 1994) and reported in typical psychology journal articles—are purely covariational, as we discuss in the final section of this article (see The Normative Assessment of Conjunctive Causation). These measures rely on the principles of experimental design to justify causal conclusions. Intuitive causal inference has sometimes been regarded as qualitative statistics (Cheng & Holyoak, 1995; Kelley, 1972; Peterson & Beach, 1967). Is it purely covariational, like conventional statistics, or does it incorporate the postulation of distal causal relations and bear a different relation to the principles of experimental design, as in our new theory? In this article we address some of these issues, while leaving others for future study.

Outcomes Illustrating the Independent Influence of Two Causes of an Effect

Let us consider the evaluation of the *independent influence* of two causes on an effect, assuming that no principle of experimental design is violated. The concept of independent causal influence is critical for assessing conjunctive causation in the typical situation in which the conjunctive cause, if it exists as a separate entity, is observable only indirectly through observations of its components (as in our flu, fire, lung cancer, and success examples). In this situation, deviation from independent causal influence provides the only basis for the assessment of conjunctive causation.

Figures 1 and 2 illustrate this concept in the simplest of possible

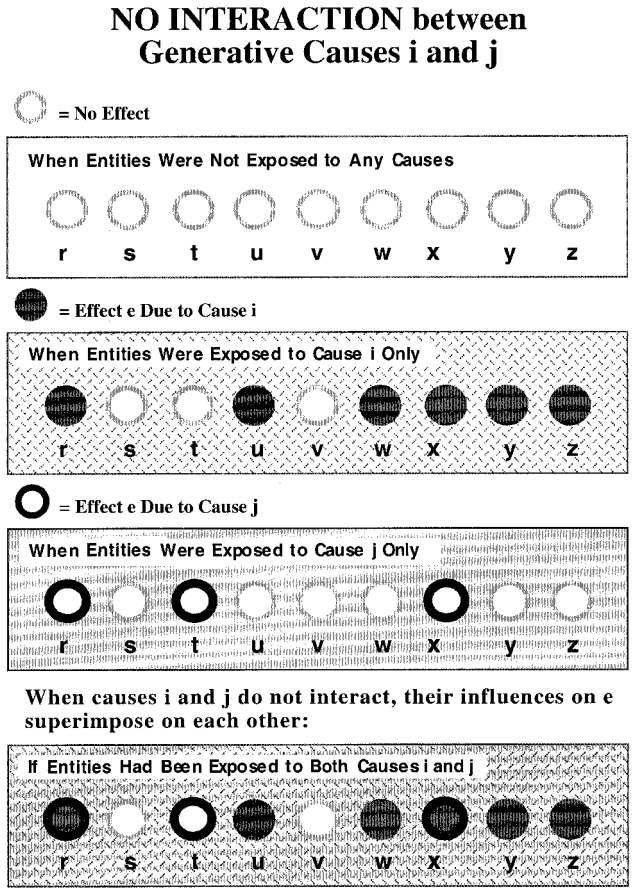

Figure 1. An illustration of the independent influences of two generative causes on an effect occurring in entities that are presumed to recover completely from the influence of one cause before being exposed to a second one. This illustration adopts the perspective of an omniscient being to whom causal relations are visible. Letters r, s, . . ., z label individual entities.

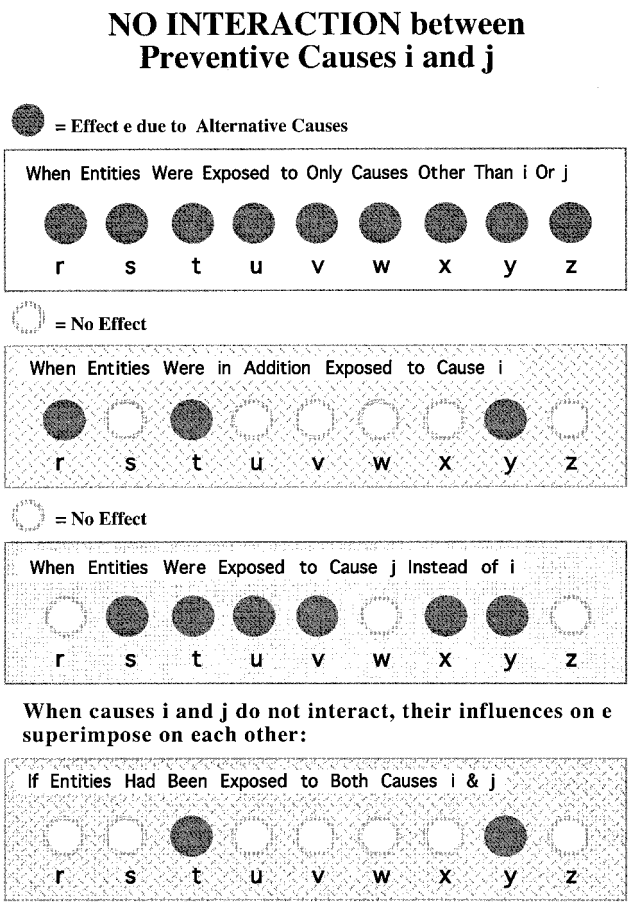

Figure 2. An illustration of the independent influences of two preventive causes on an effect occurring in entities that are presumed to recover completely from the influence of one cause before being exposed to a second one. This illustration adopts the perspective of a mere mortal who must infer causal relations from observations. Letters r, s, . . ., z label individual entities.

situations: There are two causes of effect *e* that do not interact with each other, nor with other causes of *e.* These figures depict idealized situations in which the relative frequencies of events (e.g., "*e* is not produced by any cause") accurately reflect the probabilities of those events, and the entities (represented by circles) are assumed to recover completely from the influence of the first cause, *i,* before being exposed to the second cause, *j.*[1] Figure 1 illustrates independent causal influence for the situation in which *i* and *j* each produce *e* (with some probability), seen from the perspective of an omniscient being to whom causal relations are visible (visibility is represented in the figure by marking causes of the effect in an entity). Figure 2 illustrates the situation in which these causes each prevent *e* (with some probability), seen from the perspective of a mere mortal who must infer causal relations from observations. The independent influence of *i* and *j* on *e* illustrated in these figures should be compellingly intuitive; yet, no previously proposed account of conjunctive causation, psychological or normative, concurs with these intuitions.

Let us examine Figure 1 more closely. For simplicity, assume that all candidate causes alternative to *i, j,* and their potential interaction have no power to influence *e.* The outcome for a set of entities that is consistent with this assumption is shown in the first panel: When the entities were exposed to neither *i* nor *j,* none of them showed *e.* Here and elsewhere, a light gray ring indicates the absence of *e* in an entity. The second panel shows that *i* produces *e* in two thirds of the entities. Analogously, the third panel shows that *j* produces *e* in one third of the entities. As the figure shows, this one-third probability (of having black rings in the third panel) applies both to the entities that did not show *e* when *i* was present (the light gray rings in the second panel) and to those that did (the dark gray filled circles in the same panel), which is consistent with the notion of the independent causal influences of *i* and *j* on *e.*[2]

Now, if *i* and *j* indeed independently produce *e,* then if they had been both present, their individual influences would have simply superimposed on each other, as shown by the superimposition of the second and third panels in the bottom panel. In accord with intuition, for *e* to fail to occur in an entity in this case, *e* must fail to be produced by *i* and fail to be produced by *j.* In this figure, not-*e* is the observable outcome that, in the bottom panel, exhibits a conjunction of events from the earlier panels. Thus, applying probability theory to the situation depicted, the probability of *e* not being produced in the bottom panel of Figure 1 (two ninths, as shown) is the product of the probability that *e* is not produced by *i* when *i* is present (one third, as shown in the second panel) and the probability that *e* is not produced by *j* when *j* is present (two thirds, as shown in the third panel). Note that this product definition of independence does not, and intuitively should not, apply to the event that *e* occurs in the presence of *i* and *j,* because *e* occurs when it is produced by *i, j,* or both. Rather, it applies to the "nonevents," the entities that are untouched by the causal processes in question. In this situation, the nonevent is the nonoccurrence of *e.* (The product definition also applies to the conjunction of "*e* produced by *i*" and "*e* produced by *j*" in Figure 1, indicated by the black rings superimposed on the dark gray circles in the bottom panel. But, this conjunction is observable to an omniscient being only, as assumed in the figure.)

Let us now turn to Figure 2, which illustrates the independent influence of two preventive causes on *e.* For simplicity in this case, assume that candidate causes alternative to *i, j,* and their potential interaction always produce *e.* Thus, *e* occurred in every entity that was exposed to these alternative causes, as shown by the dark gray filled circles in the first panel. As should be clear, the preventive situation in this figure is a direct analogue of the generative one in Figure 1. Instead of *i* producing *e* in two thirds of the entities, *i* now prevents *e* in two thirds of the entities. Instead of *j* producing *e* in one third of the entities, *j* now prevents *e* in one third of the entities. And, as before, if *i* and *j* indeed independently prevent *e,* then if they had been both present, their individual preventive influences would have superimposed on each other, as shown in the bottom panel.

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[1] We thank Terry Au for inspiring the counterfactual wording in these figures.

[2] In probability theory, if two events are independent, the probability of their conjunction is the product of the probabilities of the constituent events (Feller, 1957). This *product* definition implies that the probability of one event remains constant regardless of whether the other event occurs. We apply this definition to causal events here.

In this case, however, for *e* to occur, it must not be prevented by *i* and not be prevented by *j*. Thus, the probability of *e* in the bottom panel of Figure 2 (two ninths, as shown) is the product of the probability that *e* is not prevented by *i* when *i* is present (one third, as inferred from the first two panels) and the probability that *e* is not prevented by *j* when *j* is present (two thirds, as inferred from the first and third panels). Notice that the product definition of independence now applies to the occurrence of *e*, which is the nonevent in this preventive situation.

How would a formal account of conjunctive causation explain and predict the compelling intuitions of independent causal influence illustrated in these figures? We first review the predictions of previous psychological models of this process. Near the end of the article (see The Normative Assessment of Conjunctive Causation), we review what is predicted by the *cross-product ratio* (see Equation 26 for its definition), the criterion of independence underlying all conventional statistical measures for categorical data that would be applicable to the causal hypotheses in question.

Purely Covariational Models Fail: The Case of the Probabilistic Contrast Model

Cheng and Novick's (1990, 1992) probabilistic contrast model, which explains conjunctive causal attributions better than any alternative explicit psychological model of conjunctive causes (e.g., Forsterling, 1989; Hewstone & Jaspars, 1987; Hilton & Slugoski, 1986; Kelley, 1967), nonetheless fails to account for the compelling intuitions of independent influence in Figures 1 and 2, as we show in the following paragraphs. The other models, because they do not accept quantitative input, are not applicable to these figures.

The probabilistic contrast model attempts to explain how people induce both simple and conjunctive causes. According to this model, reasoners evaluate whether a single causal factor, which is perceived to occur before an effect, is a simple cause of that effect by using a *main-effect contrast* within a *focal set.* The focal set is a set of events that the reasoner uses as input to the causal discovery process. Experimental evidence independent of subjects' causal inferences shows that their focal sets may be either larger or smaller than the set of events specified by an experimenter (Cheng & Novick, 1990; Novick, Fratianne, & Cheng, 1992). The main-effect, or simple, contrast for candidate cause *i* with respect to effect *e* is defined as

$$\Delta P_i = P(e|i) - P(e|\bar{i}), \tag{1}$$

where $P(e|i)$ and $P(e|\bar{i})$ are, respectively, the probability of *e* given the presence of *i* and that probability given the absence of *i.* Both conditional probabilities are directly estimable by observable frequencies, and ΔP_i is a measure of covariation between *i* and *e.* Cheng and Holyoak (1995) and Melz, Cheng, Holyoak, and Waldmann (1993) modified the model by specifying that the focal sets reasoners prefer for computing simple contrasts are those in which every plausible alternative cause remains constant.

When applied to the focal set consisting of the first two panels of Figure 1, Equation 1 predicts that *i* causes *e.* Likewise, this equation predicts that *j* causes *e* in the same figure and that *i* and *j* each prevents *e* in Figure 2. So far, the figures are not problematic for the model.

Continuing with the description of the model, a two-way *interaction contrast* evaluates a pair of candidate causes, which are perceived to occur before *e*, as a conjunctive cause of *e* within a focal set. This contrast for candidate causes *i* and *j* with respect to *e* is defined as

$$\Delta P_{ij} = [P(e|ij) - P(e|\bar{i}j)] - [P(e|i\bar{j}) - P(e|\bar{i}\bar{j})]. \tag{2}$$

This contrast is thus a difference between differences—the simple contrast for *i* when *j* is present minus the simple contrast for *i* when *j* is absent. If ΔP_{ij} is noticeably greater than zero, then *i* and *j* interact to produce *e*; if it is noticeably less than zero, the two candidates interact to prevent *e.* Otherwise, the influences of the two candidates on *e* are independent. Equation 2 coincides exactly with a primary measure of interactive influence in epidemiology discussed in a well-regarded textbook (Rothman & Greenland, 1998).

When applied to the four panels in each figure, Equation 2 yields an interaction contrast value of –2/9 for Figure 1 (implying that *i* and *j* interact to prevent *e*) and 2/9 for Figure 2 (implying that *i* and *j* interact to produce *e*). This equation therefore fails to explain the intuition that the figures depict independent causal influence.[3]

As we hope to show in this article, this failure stems from the lack of explicit representation of distal causal relations in purely covariational models of (simple and conjunctive) causation. Another shortcoming of such models traceable to the same source is the failure to distinguish between covariation and causation. Consider an example in which the covariation between the conjunction of two events and an effect in question does not indicate causation: Daffodils blooming and morning school bells ringing, in combination, covary with flattened snakes on the road (the effect in question), even though each event individually is only very weakly associated (if at all) with flattened snakes. From the covariation between that combination of events and flattened snakes, would a reasoner conclude that the conjunction of blooming daffodils and morning school bells causes dead snakes on roads? We think not. (The pattern is due to snakes being especially likely to sun themselves on the warm asphalt on spring mornings.)

In our view, the remedy for both shortcomings of purely covariational models is to treat observable frequencies as manifestations of an underlying causal process, as in our theory of conjunctive causal power. Our theory is an extension of Cheng's (1997) theory of simple causal power, reviewed in the next section.

A Causal-Power Theory of Covariation for Simple Causes

Cheng (1997) proposed a theory of causal discovery with respect to simple causes involving a single factor. According to her theory, reasoners interpret their observations of covariation between two variables, a candidate cause and an effect, in which the candidate cause is perceived to occur before the effect, in terms of

[3] Cheng and Holyoak's (1995) modification for simple causes does not help here, as it is not obvious how the modification could apply. With respect to the conjunctive candidate, the two constituent simple causes are alternative causes; a moment's reflection will reveal that it is impossible to hold both of these alternative causes constant while varying the presence versus absence of the conjunctive cause.

a (probably innate) a priori framework that postulates the possible existence of cause-and-effect relations (cf. Kant, 1781/1965). This framework specifies that causes influence the occurrence of an effect, either producing or preventing it, with certain theoretical probabilities, termed *causal powers* (Cartwright, 1989). These are distal properties of causal relations that give rise to one's observations but exist independently of them. The *generative power* of candidate cause x with respect to effect e, denoted by q_x, is the probability with which x produces e when x occurs; the *preventive power* of x with respect to e, denoted by p_x, is the probability with which x prevents (an otherwise occurring) e when x occurs. The reasoner has a tacit goal of estimating the magnitude of these powers.

Motivation

Cheng's (1997) theory specifies, under a set of assumptions, how a reasoner can discover a causal relation, rather than merely a covariational relation, from observations in the input. Of the two previous approaches to causal induction, the covariation approach (e.g., Cheng & Novick, 1990, 1992; Rescorla & Wagner, 1972) has been unable to either specify or justify the conditions under which covariation reveals causation, and the power or mechanism approach (e.g., Ahn, Kalish, Medin, & Gelman, 1995; Shultz, 1982) has been unable to specify the process that transforms information from the available noncausal input to a causal judgment (for discussions, see Cheng, 1993; Glymour & Cheng, 1998).

To understand these problems with the two previous approaches, one should note that causal relations (unless innately known) must be discovered on the basis of (perceptually or introspectively) observable events—the ultimate source of information about the world (Hume, 1739/1987). This constraint poses a problem for all accounts of causal discovery: Observable characteristics typical of causation do not always imply causation. As we demonstrated in our snake example, covariation need not imply causation. Nor does the absence of covariation between a candidate cause and an effect imply the lack of a causal relation between them, even when alternative causes are controlled. Consider an experiment testing whether a candidate cause i prevents an effect e. Suppose alternative causes of e are controlled, and e occurs neither in the presence of i nor in its absence, so that there is no covariation between i and e. For example, suppose headaches (the effect) never occur in patients who are given a potential drug for relieving headaches (the candidate) or in patients who are given a placebo. The reasoner would not be able to conclude from this absence of covariation that the drug does not relieve headaches—there is no room for the drug to reveal its preventive power.

A Synopsis

According to Cheng's (1997) power PC theory (short for "causal-power theory of the probabilistic contrast model"), to evaluate the power of a candidate cause i to influence effect e, reasoners partition all causes of e into i and the composite of (known and unknown) causes of e alternative to i, labeled a, and they explain covariation defined in terms of observable frequencies by the hypothetical unobservable powers of i and a. The partitioning of all causes of e into i and a is the simplest possible conception of the causes of e that would still explain covariation between i and e.

The assumptions underlying the derivation of simple causal power are as follows:

1. i and a influence e independently,
2. causes in a could produce e but not prevent it,[4]
3. the causal powers of i and a are independent of their occurrences (e.g., the probability of a both occurring and producing e is the product of the probability of a occurring and the power of a), and
4. e does not occur unless it is caused.

As we show in the following paragraphs, from these four assumptions one can derive a necessary condition for inferring the causal power of i. This condition, which we call the *no-confounding condition*, concerns the independent occurrence of i and a.[5] Assumptions 1 and 2, like the no-confounding condition, are merely working hypotheses, and each is adopted or not depending on perceived evidence for or against it.

When e occurs at least as often after i occurs as when i does not occur (a condition that can be determined by observation alone), so that $\Delta P_i > 0$, reasoners evaluate the hypothesis that i produces e, and they estimate the generative causal power of i. To do so, they allow the possibility that i produces e and explain $P(e|i)$ by the probability of the union of two events: (a) e produced by i and (b) e produced by a if a occurs in the presence of i. That is, they reason that when i is present, e can be produced by i or by a if a occurs in the presence of i. Likewise, they explain $P(e|\bar{i})$ by how often e is produced by a alone when a occurs in the absence of i.

Figure 3 illustrates these explanations of the two conditional probabilities by Euler circles. The dashed and undashed circles in the figure, respectively representing e produced by i and e produced by a, are both unobservable; they are theoretical constructs. Only the white and shaded areas—respectively representing e occurring and e not occurring—and in which of the two boxes these events occur—the two boxes respectively representing exposure to i and no exposure to i—can be observed. Now, e occurring in the presence of i—the white area in the left box representing the union of the dashed and undashed circles—can be decomposed theoretically into the sum of the area of the dashed circle and that of the undashed circle, minus their overlap. The dashed circle in this box (i.e., how often e is produced by i when i is present) depends on how often i occurs (always in this situation) and its causal power, q_i (i.e., how often i produces e), a theoretical unknown. The undashed circle can be similarly ex-

[4] Glymour (1998, 2000) notes that a weaker assumption—that no unobserved causes in the composite a prevent e—would suffice for inferring the power of i. On this assumption, if the reasoner conditionalizes on the absence of any observed preventive causes of e (i.e., restricts the focal set to events in which observed preventive causes of e are absent), then the "explanations" of $P(e|i)$ and $P(e|\bar{i})$ in Cheng's (1997) theory (sketched in the paragraphs that follow these assumptions) would apply.

[5] We thank Clark Glymour and Patricia Kitcher for suggesting the terminology that we adopt here for some of Cheng's (1997) assumptions.

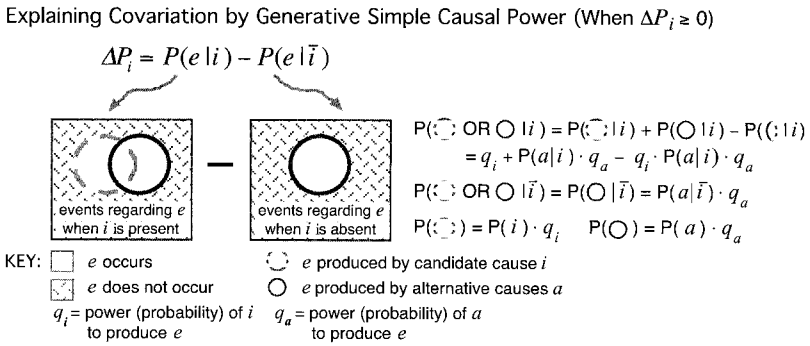

Figure 3. Euler diagrams illustrating the explanation of covariation in Cheng's (1997) theory of simple causal power.

plained. Likewise, the white area in the right box can be analogously explained, in this case by how often *e* is produced by *a* in the absence of *i* (see the equations in Figure 3). These explanations of *e* set up equations relating the observable quantities to the various theoretical variables.

For situations in which $\Delta P_i < 0$ (*e* occurs at most as often after *i* occurs as when *i* does not occur, as can be determined by observation alone), there are analogous explanations for evaluating preventive causal power. The only difference in the preventive case is that reasoners evaluate the hypothesis that *i* prevents *e* rather than *i* produces *e*. This difference implies that when reasoners evaluate whether *i* prevents *e*, they explain $P(e|i)$ by the probability of the intersection of two events: (a) *e* produced by *a* if *a* occurs in the presence of *i* and (b) *e* not stopped by *i*. That is, they reason that when *i* is present, *e* occurs only if it is both produced by *a* and not prevented by *i*. The explanation of $P(e|\bar{i})$ remains as before.

The goal of these explanations of $P(e|i)$ and $P(e|\bar{i})$ is to yield an estimate of the (generative or preventive) power of *i* from observable frequencies alone, even though it may never be possible to observe the influence of *i* in isolation. (For Figure 3, this goal would correspond to estimating the whole of the dashed circle when *i* is present, even though there may never be a diagram showing that circle by itself.) These explanations show that, under some conditions but not others, covariation implies causation. In particular, when *a* and *i* do not occur independently—that is, when there is confounding (e.g., between a rooster's crowing and the true cause of sunrise)—one equation in four unknowns results. In this case, therefore, there is no unique solution for the desired unknown. That is, the observed value of ΔP_i does not allow any inference about the causal efficacy of *i*: ΔP_i may be greater or less than the causal power of *i* (Cheng, 2000).[6]

But, consider the special case in which *a* occurs independently of *i* (i.e., when there is no confounding). Under this condition, these explanations yield equations with only one unknown, the causal power of the candidate.[7] Equation 3 gives an estimate of q_i, the *generative* simple power of *i*, when $\Delta P_i \geq 0$:

$$q_i = \frac{\Delta P_i}{1 - P(e|\bar{i})};^{8} \tag{3}$$

and, Equation 4 gives an estimate of p_i, the *preventive* simple power of *i*, when $\Delta P_i \leq 0$:

$$p_i = \frac{-\Delta P_i}{P(e|\bar{i})}. \tag{4}$$

Note that the righthand sides (RHSs) of Equations 3 and 4 require observations regarding *i* and *e* only, implying that q_i and p_i can be estimated without observing *a*.

Intuitively, Equations 3 and 4 make sense as respective estimates for simple generative and preventive power. Recall that q_i is the distal probability with which *i* produces *e*. In Equation 3, this probability is estimated by how often *e* occurs in the presence of *i* among entities in which *e* is not already produced by alternative causes. To see this interpretation, note that when *a* occurs independently of *i*, *e* is produced by *a* just as often whether or not *i* is present. Therefore, $P(e|\bar{i})$ estimates how often *e* is produced by *a* when *i* is present (as well as when *i* is absent). Accordingly, $1 - P(e|\bar{i})$, the denominator on the RHS of Equation 3, provides an estimate of how often *e* is not already produced by *a* when *i* is present.

As should be clear, at one extreme, $q_i = 1$ means that *i* is estimated to produce *e* in every entity. At the other extreme, $q_i =$

[6] Generalizations of Cheng's (1997) theory have been shown to allow generative causal power to be estimated normatively in certain cases even when there is confounding (Glymour, 2001). For the rooster example, although no inference results from applying a causal-power analysis to "crowing" with respect to "sunrise," a typical reasoner would judge that crowing does not cause sunrise. Cheng gave an explanation of how this definite answer might be due to previously inferred causal knowledge (see also Lien & Cheng, 2000).

[7] A nice property of causal powers is that the solutions are independent of the particular covariation measure "explained" by such powers whenever there is a solution according to the explanations. For example, explaining the ratio of $P(e|i)$ to $P(e|\bar{i})$ instead of the difference between these conditional probabilities yields identical values for the candidate causal power except for cases in which the ratio explanation gives an undefined value.

[8] Equations 3 and 4 appear in Sheps's (1958) article, in which Sheps refers to them as estimates of *harmful effects* and *beneficial effects*, respectively. Equation 3 was subsequently derived from assumptions similar to those in Cheng (1997) and proposed as a measure of susceptibility by Khoury, Flanders, Greenland, and Adams (1989). It was also derived by Pearl (2000) under a weaker set of assumptions, with a different interpretation.

0 means that *i* is estimated to never produce *e* in any entity (i.e., to be noncausal). Of course, if *e* occurs all the time due to *a* (i.e., $P(e|\bar{i}) = 1$), q_i cannot be assessed (i.e., it has the undefined value of 0/0), as there are no remaining entities in which *i* can possibly manifest its generative power.[9] Equation 3 therefore explains this intuition and its analogue in experimental design—the principle of avoiding a ceiling effect. Despite the observation that $\Delta P_i = 0$ in this case, on the basis of this pattern of observations one would not make any judgments about the causal relation in the world between *i* and *e*, which is assumed to exist regardless of a reasoner's observations; in particular, one would not infer that *i* does not cause *e*. In contrast, if *e* never occurs due to *a* (i.e., nothing other than *i* produces *e*, so that $P(e|\bar{i}) = 0$), then $P(e|i)$, which in this case equals the simple contrast (ΔP_i), provides a good estimate of q_i: In this context, whenever *e* occurs in the presence of *i*, it is produced by *i*. Note that, unlike purely covariational measures (e.g., ΔP_i), whenever q_i has a defined value, that value is a property of the causal relation between *i* and *e* and is independent of how often alternative causes in the context produce *e* (for an illustration of this feature of simple power, see Glymour & Cheng, 1998).

Analogously for Equation 4, the simple preventive power of *i* is estimated by how often *e* does not occur in the presence of *i* among entities in which *e* is produced by *a*. As before, $P(e|\bar{i})$ is an estimate of how often *e* is produced by *a*. Thus, $p_i = 0$ means that *i* is estimated to never prevent *e* in any entity, and $p_i = 1$ means that *i* is estimated to prevent *e* in every entity. Of course, if *e* never occurs due to *a* (i.e., $P(e|\bar{i}) = 0$), p_i cannot be assessed (i.e., it has the undefined value of 0/0), as there are no entities in which *i* can possibly manifest its preventive power. Equation 4 therefore explains this intuition and the tacit preventive analogue of the ceiling effect, as illustrated in our headache-relieving-drug example.[10] This unspoken principle of avoiding situations in which *e* never occurs in the absence of a candidate in tests of its preventive power is uniformly obeyed in experimental design (for the use of the last two principles by untutored reasoners, see Wu & Cheng, 1999). In contrast, if *e* occurs all the time due to *a* (i.e., $P(e|\bar{i}) = 1$), then $-\Delta P_i$, which now equals $1 - P(e|i)$, provides a good estimate of p_i: In this context, whenever *e* does not occur in the presence of *i*, it is prevented by *i*.

Some Implications

The power PC theory provides a qualitative description of untutored human causal discovery involving direct simple causes. As described in Cheng (1997), it accounts for a diverse range of psychological experimental findings and intuitions that are inexplicable by previous accounts of causal inference.

A critical aspect of the power PC theory is that it "deduces" when to induce. One consequence of this is that the theory provides a normative justification for when and why covariation implies causation. In particular, it explains some principles of experimental design. As we just noted, it explains the ceiling effect and its preventive analogue. In addition, it explains the principle of control. To enable causal inference, scientists use methods such as randomly assigning subjects to conditions in a study and holding alternative causes at a constant value across conditions. The principle of controlling for alternative causes is also used in observational studies, for example, by matching as much as possible the relevant properties of subjects in every pair (e.g., identical twins raised in the same family) except for a candidate feature (e.g., oxygen deprivation during delivery from mother) to study its influence on some outcome (e.g., schizophrenia). Because the derivations of Equations 3 and 4 show that those equations result if and only if there is no confounding (Cheng, 1997, 2000), they explain why *no confounding* is a boundary condition for causal inference according to experimental design. Thus, whereas these basic principles of design are extrinsic to purely covariational measures, and either augment or overrule their interpretation, they arise from the same assumptions as do the measures under our causal-power approach and are intrinsically incorporated into these measures.

Cheng's (1997) theory can be regarded as a particular parameterization of directed graphical models, sometimes called *Bayesian networks* (Glymour, 1998, 2000, 2001; Pearl, 2000; Spirtes et al., 2000; Tenenbaum & Griffiths, 2001; see Gopnik et al., 2004, for a general proposal of such networks as a psychological model). In this parameterization, each causal-power variable is a parameter associated with an arrow in the acyclic graph representing the assumptions underlying the theory, a parameter that represents the influence of (a) an intervention on the node at the tail of an arrow on (b) the node at the head of the arrow.

A Causal-Power Analysis of Two-Way Interactions

Overview and Assumptions

In this section, we extend Cheng's (1997) power PC theory of simple causation to address the assessment of conjunctive causation. We call our new theory the *conjunctive power PC theory.*[11] Both of these theories propose that reasoners implicitly follow a qualitative version of the mathematical analyses. Our predictions concerning their judgments about causal power are therefore ordinal.

We present an analysis of how a reasoner might evaluate whether two candidate causes, *i* and *j*, interact to influence an effect *e* (i.e., whether they do not influence *e* independently). Just as in the case of simple causes, the reasoner explains observable covariation by the possible causal powers. Whereas Cheng (1997) explained the occurrence of *e* in the presence of a single causal candidate, *i*, by considering the possible existence of two causes of *e*, namely, *i* and *a* (the composite of causes other than *i*), we must consider the possible existence of four causes to explain the occurrence of *e* in the presence of two causal candidates, *i* and *j*: *i*, *j*, the combination of *i* and *j* as a conjunctive cause, and *a* (here, *a* is the composite of all causes other than the candidate causes *i*, *j*, and their conjunction). We refer to the power of the combination of *i* and *j* as their *conjunctive power*. Table 1 enumerates which of these four candidate causes can exert their influence in the various possible situations formed by varying the presence versus absence

[9] Because ΔP_i is nonnegative for q_i, $P(e|\bar{i})$ being 1 implies that $\Delta P_i = 0$; thus, $q_i = 0/0$.

[10] It is not the typical floor effect, which concerns situations in which some enabling conditions of a candidate for generative—rather than preventive—power are missing so that *i* does not increase the frequency of *e*.

[11] PC again stands for *probabilistic contrast*, but in this case the contrast is the conjunctive contrast specified by our new theory (Equation 5) rather than the one specified by our old probabilistic contrast model (Equation 2).

Table 1
Possible Causes of an Effect, e, in the Various Possible Situations Involving Observable Candidate Causes i and j When All Alternative Candidate Causes (Known and Unknown), Denoted by the Composite a, Influence e Independently of i and j

	j	
i	Occurs	Does not occur
Occurs	$i \wedge j$, *i*, *j*, *a*	*i*, *a*
Does not occur	*j*, *a*	*a*

Note. $i \wedge j$ denotes the conjunctive cause composed of both *i* and *j*.

of *i* and *j*. The partitioning of all causes of *e* into these four yields the simplest possible conception of the causes of *e* that would explain any covariation between *e* and the candidate causes *i*, *j*, and their conjunction.

Estimating the simple and conjunctive powers of *i* and *j* requires that *i*, *j*, and *e* be observable. Our analyses concern the typical situation in which the conjunctive cause is observable only indirectly through observations of *i* and *j* (as in all of our examples of conjunctive causation so far). If the conjunctive cause is a new entity that is directly observable (e.g., a new chemical compound that is produced as the result of mixing two drugs), it can potentially be evaluated as a simple cause on its own, rendering the present analyses unnecessary. In contrast, in the situation under investigation here, whether *i* and *j* interact to influence *e*, as we noted earlier, can only be inferred from the *deviation* of the observed probability of *e* from that expected if *i* and *j*, possibly counterfactually, exerted only their simple powers on *e*. The estimation of this *expected probability* is therefore a critical step in our analyses.

The role of this expected probability in the estimation of conjunctive power has a direct analogue in the estimation of simple power. Recall that, with respect to simple powers, $P(e|\bar{i})$ estimates how often *e* is produced by alternative causes. In other words, $P(e|\bar{i})$ estimates the expected probability of *e* in the presence of *i*, assuming that only causes other than *i* exerted an influence on *e*. A deviation from this counterfactual probability indicates that *i* is a simple cause of *e*. Likewise, we assume that to evaluate the conjunctive power of two candidate causes *i* and *j*, the reasoner estimates the expected probability of *e* in the presence of *i* and *j*, assuming that only causes other than the conjunction of *i* and *j* exerted an influence on *e*.

Thus, analogous to the simple contrast in Equation 1, a conjunctive contrast has the following general form:

$$\Delta P_{ij} = P(e|ij) - \tilde{P}(e|ij), \tag{5}$$

where $P(e|ij)$ is the actual (i.e., observed) probability of *e* in the presence of *i* and *j*, and $\tilde{P}(e|ij)$ is the expected probability if *i* and *j* exerted only their simple powers on *e*. Just as reasoners explain the two conditional probabilities in a simple contrast to "deduce" simple causal power in Cheng's (1997) theory, they explain the analogous conditional probabilities in Equation 5 to "deduce" conjunctive power in our extension of that theory (see Figure 4). Now, instead of partitioning the causes of *e* into the candidate simple cause and everything else, reasoners partition them into the candidate conjunctive cause corresponding to the combination of *i* and *j* (the dashed circle in Figure 4) and everything else (the simple causes *i* and *j*, represented by two lighter undashed circles in the figure, and a composite of all other causes of *e*, represented by a darker undashed circle). Reasoners explain $P(e|ij)$ by the potential causal powers of *i*, *j*, their conjunction, and *a*; they explain $\tilde{P}(e|ij)$ by the same set of potential causal powers without the conjunctive power of *i* and *j*. Thus, as for simple causes, everything other than the candidate is common across the two conditional probabilities. Analogous to Figure 3, the goal here is to estimate the relative area of the whole dashed circle even though it may never appear on its own.

Recall that in Cheng's (1997) theory, depending on the sign of ΔP_i, reasoners evaluate *i* as either a generative or preventive cause, situations that call for different measures of simple causal power. The measures of conjunctive power similarly differ depending on the sign of ΔP_{ij}, as well as of ΔP_i and ΔP_j. Allowing *i* and *j* to be either generative or preventive causes of *e*, and treating *i* and *j* as interchangeable (so that *i* being generative and *j* being preventive is equivalent to *i* being preventive and *j* being generative), there are three possible combinations of simple candidate cause types: Both simple candidate causes may be potentially generative, both may be potentially preventive, and one may be of each type. For each of these three combinations of simple cause types, the conjunction of *i* and *j* may either generate *e* or prevent it. There are, therefore, six possible combinations of two simple powers and their conjunctive power.

For a concrete illustration, consider an example of the last combination of candidate cause types. The effect of interest (*e*) is attending (as opposed to not attending) a high school basketball game in a Midwestern state. Snow-covered roads (*i*) reduce attendance (i.e., have a preventive influence), whereas a strong winning record for the home team (*j*) increases attendance (i.e., has a

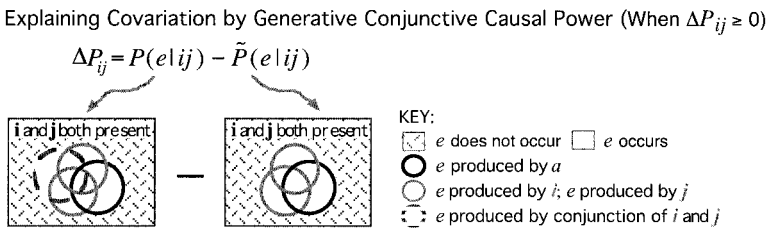

Figure 4. Euler diagrams illustrating the explanation of covariation in our theory of conjunctive causal power.

generative influence). The identification of these simple causes may not provide a sufficient explanation, however, because snowy roads and a strong winning record may interact to influence attendance, such that the combination, for example, increases the probability of attending compared with what would be expected if the candidates exerted only their simple powers.

For each of the three combinations of the types of simple powers of *i* and *j*, we present an explanation of $\tilde{P}(e|ij)$ based on the same assumptions listed earlier for the derivation of simple power, applying them to simple causes only, including the two candidate simple causes, *i* and *j*. The results concerning $\tilde{P}(e|ij)$ therefore follow directly from Cheng's (1997) theory of simple power.

Then, for each of the three combinations of simple powers, we derive equations for q_{ij} and p_{ij}, respectively the generative and preventive powers of the conjunction of *i* and *j*. We do so by introducing explanations of the observed $P(e|ij)$, completing the explanation of the conjunctive contrast (Equation 5). Our derivations make use of Assumption 1 below, which concerns conjunctive causal power, together with the simple-power assumptions, which are generalized when relevant to apply not just to *i* but also to the other two candidate causes, *j* and the conjunction of *i* and *j*. We list all five relevant assumptions below, with the last four mapping, respectively, onto the four simple power assumptions:

1. The influence of the interaction between *i* and *j* can be represented by a separate conjunctive power, q_{ij} or p_{ij}; this conjunctive cause can operate only when *i* and *j* both occur (analogously as for simple causes).

2. All causes, simple and conjunctive, influence *e* independently.

3. Unobserved causes in *a* could produce *e*, but not prevent it.

4. All causes influence the occurrence of *e* with causal powers that are independent of how often the causes occur.

5. *e* does not occur unless it is caused.

The results of our mathematical derivations, detailed in the remainder of this section, are summarized in Figures 5 and 6, for the special case in which *a* occurs independently of *i*, *j*, and their conjunction (i.e., when there is no confounding). Because the explanation of $\tilde{P}(e|ij)$ involves only a subset of the assumptions just listed for conjunctive power, our accounts of $\tilde{P}(e|ij)$ and of conjunctive power should be evaluated separately; our account of conjunctive power could fail to hold even when our account of $\tilde{P}(e|ij)$ is supported. We propose, however, that each of these parts of our theory is both descriptive and normative.[12]

Both Candidate Causes Are Generative

First, we consider situations in which candidate causes *i* and *j* each (potentially) produces *e* (e.g., see Figure 1). That is, these are situations in which $\Delta P_i \geq 0$ and $\Delta P_j \geq 0$. In these situations, we denote the expected probability of *e* when both *i* and *j* are present but exerted only their simple powers on *e* by $\tilde{P}_{++}(e|ij)$, with the subscript indicating that the simple candidate causes are both potentially generative.

Estimating $\tilde{P}_{++}(e|ij)$

If *i* and *j* exerted only their simple powers on *e*, then when *i* and *j* are jointly present, *e* occurs if *i* causes *e*, *j* causes *e*, or *a* is present and causes *e*. Accordingly, the expected probability of *e* in the presence of *i* and *j* can be estimated by the union of these three events. A simple way to calculate this probability makes use of the relevant De Morgan's Law.[13] Specifically, negating both sides of the law that states that the complement of a union is the intersection of the complements of the constituents of the union, we find that $\tilde{P}_{++}(e|ij)$ is the complement of the probability of the intersection of three events in the presence of *i* and *j*: *e* is not produced by *i*, *e* is not produced by *j*, and *e* is not produced by *a* when *a* is present. In other words, when *i* and *j* are both present but they exerted only their simple influences, the expected probability of *e* is the complement of the probability that *e* is not produced by any of the simple candidate causes. Therefore,

$$\tilde{P}_{++}(e|ij) = 1 - (1 - q_i) \cdot (1 - q_j) \cdot [1 - P(a|ij) \cdot q_a]. \quad (6)$$

Equation 6 contains unobservable causal-power variables and the probability $P(a|ij)$, which is typically unobservable because *a* is not knowable. To demonstrate that the equation is solvable through observations alone, and hence there is no circularity in our argument, we replace the unobservable terms with "observable" probabilities, that is, with probabilities that are directly estimable by observable frequencies.

To do so, we first estimate the simple powers, q_i and q_j, according to Cheng's (1997) theory. Recall that the power of a simple candidate cause can be estimated from observable probabilities using Equations 3 or 4 only under the no-confounding condition, namely that all causes alternative to the candidate cause occur independently of it. In the situation of interest here, satisfying this condition would mean that, for example, when *i* is the candidate cause, *j*, *a*, and the conjunctive cause must all occur independently of it. This implies that even for the special case in which *a* occurs independently of *i*, *j*, and their conjunction, to estimate the simple power of *i* or *j*, conditionalizing on the absence of the other simple candidate is the only way one can be sure that the no-confounding prerequisite is satisfied (see Table 1). Without this conditionalization, confounding may occur: For example, with

[12] The independent influence assumption can be met in two ways. One possibility is that the simple causes and the conjunctive cause operate sequentially, with the conjunctive cause exerting its influence, if any, on the outcome produced by the simple causes. The other possibility is that the simple and conjunctive causes operate in parallel. We discuss this distinction more fully later (see *The Assumption That the Conjunctive Cause Operates on the Static Outcome of the Simple Causes*), as it is relevant for only two of the six possible cases of conjunctive causation. We take the sequential version of Assumption 1 to be the default.

[13] De Morgan's Laws state that the complement of a union is the intersection of the complements of the constituents of the union (e.g., not-*a*-or-*b* ≡ not-*a* and not-*b*) and that the complement of an intersection is the union of the complements of the constituents of the intersection (e.g., not-*a*-and-*b* ≡ not-*a* or not-*b*).

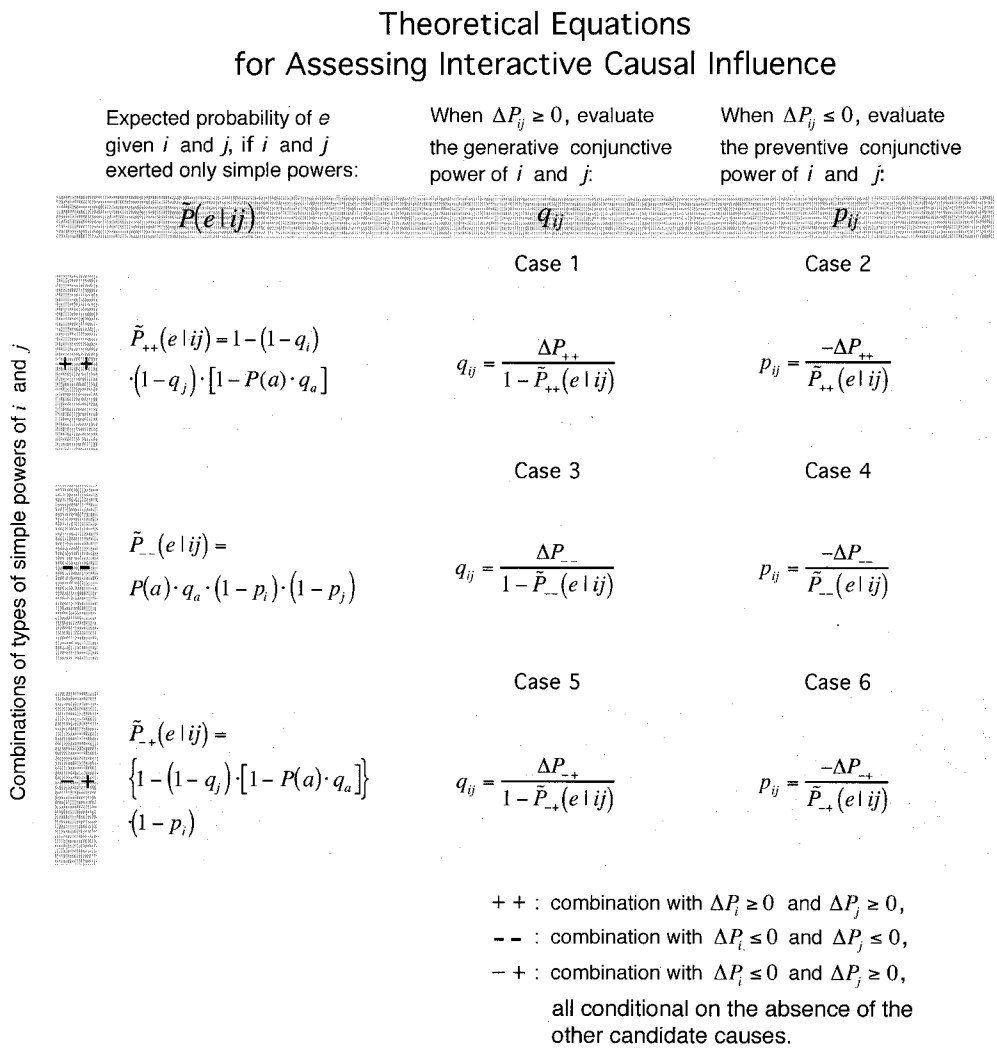

Figure 5. Theoretical equations for assessing interactive causal influence for the special case in which alternative causes occur independently of the candidate causes.

respect to candidate *i*, the conjunctive cause, if it exists, would covary with *i*, occurring in its presence but not in its absence.

Accordingly, for q_i, we instantiate Equation 3 conditional on *j* being absent, yielding

$$q_i = \frac{P(e|i\bar{j}) - P(e|\bar{i}\bar{j})}{1 - P(e|\bar{i}\bar{j})}. \quad (7)$$

Likewise,

$$q_j = \frac{P(e|\bar{i}j) - P(e|\bar{i}\bar{j})}{1 - P(e|\bar{i}\bar{j})}. \quad (8)$$

Because q_i and q_j have undefined values when $P(e|\bar{i}\bar{j}) = 1$, analogously as we showed earlier regarding the ceiling effect for simple causes, $P(e|\bar{i}\bar{j}) < 1$ is a condition for estimating these simple powers.

The last unobservable term in the expression for the expected probability (see Equation 6) is the product $P(a|ij) \cdot q_a$. When *a* occurs independently of the conjunction of *i* and *j* (i.e., when the no-confounding condition holds), $P(a|ij) = P(a|\bar{i}\bar{j})$. Thus, analogous to the explanation of $P(e|\bar{i})$ by $P(a) \cdot q_a$ in Cheng (1997), we obtain

$$P(a|ij) \cdot q_a = P(a|\bar{i}\bar{j}) \cdot q_a = P(e|\bar{i}\bar{j}). \quad (9)$$

Expanding the expression for the expected probability (see Equation 6) using Equations 7–9 and simplifying, we obtain Equation 10, which does not contain any unobservable variables. To show more clearly the analogous forms among the equations we obtain, we express the RHS of this equation in terms of the probability of not-*e* rather than of *e*:

$$\tilde{P}_{++}(e|ij) = 1 - \frac{P(\bar{e}|i\bar{j}) \cdot P(\bar{e}|\bar{i}j)}{P(\bar{e}|\bar{i}\bar{j})}. \quad (10)$$

Note that without the no-confounding condition—that *a* occurs independently of *i*, *j*, and their conjunction—Equations 7–9 would

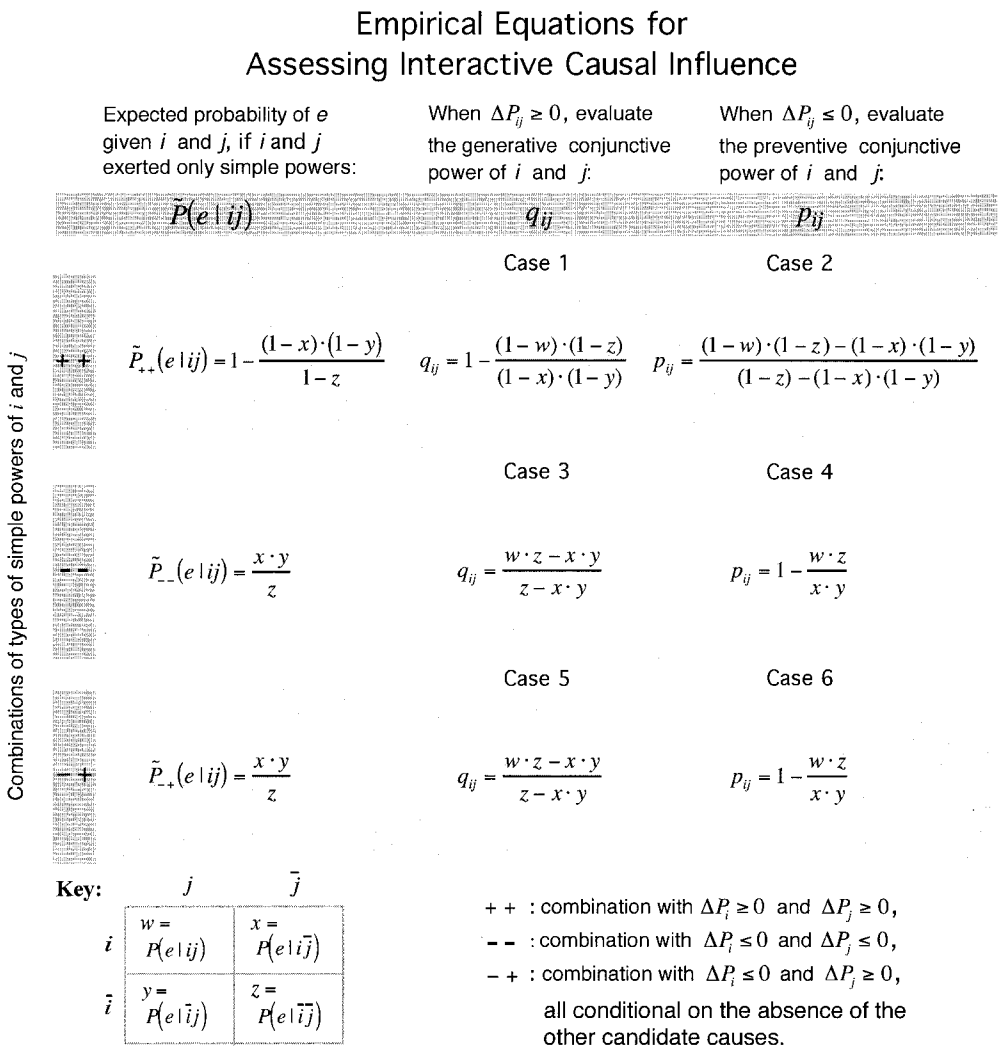

Figure 6. Empirical equations for assessing interactive causal influence for the special case in which alternative causes occur independently of the candidate causes.

not obtain, leaving $\tilde{P}(e|ij)$ in Equation 6 unsolved.[14] Anticipating our subsequent derivations, without a solution for $\tilde{P}(e|ij)$, there would be no solution for conjunctive causal power. In other words, when there is confounding of the causes in question by alternative causes of the effect, our measure indicates that there is no solution for conjunctive power. Thus, as for simple causal power, our derivations explain why no confounding is a boundary condition for inferring conjunctive causal power.

This explanation reveals, as do analogous explanations in Cheng's (1997) theory, a new relation between the principles of experimental design and the mathematical measures of inference. Contrary to current scientific practice, in which the output of statistics is overridden when a principle of experimental design is violated, under our approach the mathematical measure yields an output that corresponds to the intuitively and normatively correct inference, so that there is no such overriding: The design principles that concern causality become part and parcel of the mathematical measures of inference.

We refer to equations that contain unobservable theoretical variables, such as Equation 6, as theoretical equations. Figure 5 summarizes our key theoretical equations. We refer to the corresponding equations in which the theoretical variables are replaced by observable probabilities, such as Equation 10, as empirical equations. Figure 6 summarizes these equations.

Figure 1 illustrates both Equations 6 and 10, the theoretical and empirical equations for $\tilde{P}_{++}(e|ij)$. (As should be clear from the independence assumptions underlying our analysis, these equations are not restricted to the type of data illustrated in this figure, namely, data from repeated measures on the same entities that

[14] When the no-confounding condition holds, the equation for the unconditional expected probability of e, $\tilde{P}_{++}(e)$, the more general version of Equation 6 from which the other three expected conditional probabilities can be derived, is $\tilde{P}_{++}(e) = 1 - [1 - P(i)\cdot q_i] \cdot [1 - P(j)\cdot q_j]\cdot[1 - P(a)\cdot q_a]$.

recover from the influence of each cause. They are also applicable to other types of data for which those independence assumptions hold—for example, data from exposing disjoint sets of entities to the various combinations of candidate causes.) Substituting the causal-power values from Figure 1 into the theoretical equation yields the value of the expected frequency shown in the bottom panel of the figure: $\tilde{P}_{++}(e|ij) = 1 - (1 - 2/3) \cdot (1 - 1/3) \cdot (1 - 0) = 7/9$. Substituting the corresponding relative frequencies from the top three panels of that figure into the empirical equation yields the same value:

$$\tilde{P}_{++}(e|ij) = 1 - \frac{1/3 \cdot 2/3}{1 - 0} = 7/9.$$

(The power values and observed relative frequencies happen to correspond in this simple figure, because *i* and *j* are the only causes of *e*.) As can be seen, our mathematical criterion of independent influence corresponds to the intuitive one illustrated in Figure 1.

As for simple causes, we assume that the direction of the conjunctive causal hypothesis to be evaluated—that the conjunction produces *e* or that it prevents *e*—depends on the sign of the conjunctive contrast (Equation 5). For $\Delta P_{ij} \geq 0$ and $\Delta P_{ij} \leq 0$, respectively, the reasoner assesses generative and preventive conjunctive power. We discuss these two cases below. When $\Delta P_{ij} = 0$, the reasoner may evaluate either type of conjunctive power or both types. (Recall that the goal is to solve for the causal powers, which may be either zero or undefined when $\Delta P_{ij} = 0$.)

Case 1: Evaluating the Conjunctive Power of i and j to Produce e, When i, j, and a May Each Have Simple Generative Power

The derivation of q_{ij}. We first consider the case in which $\Delta P_{ij} \geq 0$, so that the reasoner assesses whether *i* and *j* interact to produce *e* and estimates q_{ij}, the generative power of the conjunction. In this case, the observed $P(e|ij)$ would be explained by how often *e* is produced in the presence of *i* and *j* by the simple causes or by the conjunction of *i* and *j*. Note that including the conjunctive power as a possible explanation for $P(e|ij)$ does not presuppose that the conjunction is in fact a cause, as its power may turn out to be zero. Applying Assumptions 1–5 and the relevant De Morgan's Law, one can see that the probability of this union is the complement of the probability of the intersection of two events in the presence of *i* and *j*: *e* is not produced by the simple causes, and *e* is not produced by the conjunction of *i* and *j*:

$$P(e|ij) = 1 - [1 - \tilde{P}_{++}(e|ij)] \cdot (1 - q_{ij}). \tag{11}$$

Substituting the expression for $P(e|ij)$ from Equation 11 into the conjunctive contrast defining ΔP_{ij} (Equation 5), simplifying, and rearranging terms, we obtain an expression for generative conjunctive power:

$$q_{ij} = \frac{\Delta P_{++}}{1 - \tilde{P}_{++}(e|ij)}. \tag{12}$$

Expanding $\tilde{P}_{++}(e|ij)$ in Equation 12 according to the theoretical equation for the expected probability (Equation 6), simplifying, and making use of the no-confounding condition (independent occurrence) to reduce $P(a|ij)$ to $P(a)$, we obtain

$$q_{ij} = \frac{\Delta P_{++}}{(1 - q_i) \cdot (1 - q_j) \cdot [1 - P(a) \cdot q_a]}. \tag{13}$$

These results show that q_{ij} has a defined value only under certain (observable) conditions. As can be seen from Equation 12, q_{ij} has a defined value only if $\tilde{P}_{++}(e|ij) < 1$. Equation 13 clarifies that this condition obtains only if neither *a* nor *i* nor *j* produces *e* all the time, implying that $P(e|\bar{i}\bar{j}) < 1$, $P(e|i\bar{j}) < 1$, and $P(e|\bar{i}j) < 1$.

The interpretation of q_{ij}. The analogy between the equations for conjunctive and simple power (Equations 12 and 3, respectively) should be clear. Just as $1 - P(e|\bar{i})$ in Equation 3 is an estimate of how often *e* is not already produced by causes other than the one of interest, so too is $1 - \tilde{P}_{++}(e|ij)$ in Equation 12. Other causes consist of *a* only for Equation 3, but consist of the simple causes *i* and *j* as well as *a* for Equation 12. Therefore, the generative power of a candidate cause, both simple and conjunctive, is estimated by how often *e* occurs in the presence of the candidate among entities in which *e* is not already produced by alternative causes.

To visualize generative conjunctive power, one should imagine superimposing the influence of the conjunctive cause on the bottom panel of Figure 1, so that this panel depicts the observed $P(e|ij)$ rather than the expected $\tilde{P}(e|ij)$. Assume that some of the entities (say, a randomly selected set) have sun rays radiating from them, denoting the occurrence of *e* due to the conjunctive cause, as seen by the omniscient being. Some of the entities so marked will be ones in which *e* is not already caused by the simple causes (i.e., the light gray rings); q_{ij} would be estimated by the proportion of shining rings among the light gray ones. As the visualization shows, q_{ij} is a probability (on a ratio scale) that has a well-defined meaning in terms of the relative frequency of an event in the world. As we discuss later (see *On the Interpretability of the Output of Various Measures of Interactive Causal Influence*), this is not always true of the causal estimates generated by purely covariational models.

We interpret our result regarding q_{ij} as a psychological model in two ways. The first, indirect, interpretation is rooted in the expression of conjunctive power in terms of theoretical power variables. According to this interpretation, a reasoner may estimate the numerator on the RHS of Equation 12, ΔP_{++}, according to Equation 5. In turn, $\tilde{P}_{++}(e|ij)$ is estimated according to Equation 6, with q_i, q_j, and $P(a|ij) \cdot q_a$ estimated according to Cheng's (1997) theory (see Equations 7–9).

The second, more direct, interpretation makes use of another analogy to Cheng's (1997) solutions for simple power by considering conjunctive power purely in terms of observable probabilities. To arrive at this interpretation, we expand the theoretical equation for conjunctive power (Equation 12) according to the conjunctive contrast equation and the empirical equation for the expected probability (Equations 5 and 10, respectively). Simplification yields an empirical equation for q_{ij} when *i* and *j* are potentially generative:

$$q_{ij} = 1 - \frac{P(\bar{e}|ij) \cdot P(\bar{e}|\bar{i}\bar{j})}{P(\bar{e}|i\bar{j}) \cdot P(\bar{e}|\bar{i}j)}. \tag{14}$$

The conditions for applicability for Equation 14 are necessarily the same as those for Equation 13—the theoretical expression for q_{ij} in

this situation—because the two equations are algebraically equivalent. When i and j do not interact,

$$\frac{P(\bar{e}|ij) \cdot P(\bar{e}|\bar{i}\bar{j})}{P(\bar{e}|i\bar{j}) \cdot P(\bar{e}|\bar{i}j)} = 1,$$

and $q_{ij} = 0$. We illustrate Equation 14 for this special case by returning to Figure 1. Instantiating this equation with the conditional probabilities estimated from that figure yields

$$q_{ij} = 1 - \frac{2/9 \cdot 9/9}{3/9 \cdot 6/9} = 0.$$

Although the two procedures for estimating conjunctive power give identical estimates of q_{ij}, the indirect procedure seems more plausible to us. We do not empirically differentiate between them in this article, however, because our goal here is to study what reasoners compute, rather than how they compute it (Marr, 1982).

Case 2: Evaluating the Conjunctive Power of i and j to Prevent e, When i, j, and a May Each Have Simple Generative Power

The derivation of p_{ij}. Now consider the case in which $\Delta P_{ij} < 0$, so that the reasoner evaluates whether i and j interact to prevent e; that is, the reasoner estimates p_{ij}, the preventive power of the conjunction. As in Case 1, i, j, and a may each individually generate e; thus, $\tilde{P}_{++}(e|ij)$ remains as given by Equation 6.

If i and j potentially interact to prevent e, then in the presence of i and j, e would occur if it is (a) produced by the simple causes and (b) not prevented by the conjunctive cause. Thus, the observed $P(e|ij)$ would be the probability of the intersection of these two events, yielding

$$P(e|ij) = \tilde{P}_{++}(e|ij) \cdot (1 - p_{ij}). \tag{15}$$

Substituting the expression for $P(e|ij)$ from Equation 15 into the conjunctive contrast (Equation 5), simplifying, and rearranging terms, we obtain a theoretical expression for p_{ij} in this situation:

$$p_{ij} = \frac{-\Delta P_{++}}{\tilde{P}_{++}(e|ij)}. \tag{16}$$

Rewriting the RHS of Equation 16 in terms of observable probabilities as we did for Case 1, we obtain an empirical equation for p_{ij} in this situation:

$$p_{ij} = \frac{P(\bar{e}|ij) \cdot P(\bar{e}|\bar{i}\bar{j}) - P(\bar{e}|\bar{i}j) \cdot P(\bar{e}|i\bar{j})}{P(\bar{e}|\bar{i}\bar{j}) - P(\bar{e}|\bar{i}j) \cdot P(\bar{e}|i\bar{j})}. \tag{17}$$

When i, j, and a are all potentially generative, the conjunctive powers (p_{ij} and q_{ij}) are expressed most directly in terms of the conditional probabilities of not-e, the nonevent. Recall that the nonevent is the outcome showing lack of influence by the simple causes. Figure 1 illustrates the special case for Equations 16 and 17 in which the observed relative frequencies yield $p_{ij} = 0$.

The interpretation of p_{ij}. The analogy between Equation 16 for conjunctive preventive power and Equation 4 for simple preventive power should be clear. Both equations estimate how often e is prevented in the presence of the candidate cause among entities in which e is produced by causes other than the candidate. Returning to Figure 1 for a different visualization, let us superimpose the influence of the preventive conjunctive cause on the bottom panel, and shrink entities in which e is prevented by this cause. Then, p_{ij} would be the proportion of small entities among the seven entities that currently show e.

Discriminating the Conjunctive Power PC Theory From the Probabilistic Contrast Model: A Deterministic Example

Suppose e always occurs in the presence of i or j alone, never occurs in the absence of both causes, and always occurs in the presence of both causes. An intuitive causal interpretation of this pattern of probabilities is that (a) i and j each always produces e; (b) they do not interact to prevent e; and (c) they may, or may not, interact to produce e—there is no evidence either way. A reasoner therefore definitely should not conclude that i and j interact. Contradicting this intuition, the interaction contrast (Equation 2) from the probabilistic contrast model (Cheng & Novick, 1990) yields the conclusion that i and j interact to prevent e, as that contrast has a value of -1. Our conjunctive power equations, on the other hand, yield conclusions that are in accord with intuition: q_{ij} has an undefined value according to Equations 12 and 14, and $p_{ij} = 0$ according to Equations 16 and 17 (because $\tilde{P}_{++}(e|ij) = 1$ and $\Delta P_{++} = 0$). In the empirical section of this article (see Empirical Tests of the Conjunctive Power PC Theory), we discuss some existing data on reasoners' causal attributions when presented with the type of information in this example.

Both Candidate Causes Are Preventive

Next, we consider situations in which each of the candidate causes, i and j, (potentially) prevents e (e.g., see Figure 2). In these situations, we denote the expected probability of e when both i and j are present but exerted only their simple powers by $\tilde{P}_{--}(e|ij)$, with the subscript indicating that the simple candidate causes are both potentially preventive.

Estimating $\tilde{P}_{--}(e|ij)$

If i and j exerted only their simple powers on e, then when i and j are both present, e occurs if a is present and causes it, and neither i nor j prevents it. In this situation, the expected probability of e is as shown in Figure 5 (see the leftmost equation in the middle panel). This equation is the preventive analogue of the earlier expected probability equation.

As for the earlier situation, to derive the empirical version of the expected probability equation, we need empirical equations for the simple powers of i and j. These are obtained by instantiating Equation 4 for p_i conditional on j being absent and for p_j conditional on i being absent. Assuming that a occurs independently of i and of j, respectively, we obtain

$$p_i = \frac{P(e|\bar{i}\bar{j}) - P(e|i\bar{j})}{P(e|\bar{i}\bar{j})} \tag{18}$$

and

$$p_j = \frac{P(e|\bar{i}\bar{j}) - P(e|\bar{i}j)}{P(e|\bar{i}\bar{j})}. \tag{19}$$

Expanding the theoretical equation for $\tilde{P}_{--}(e|ij)$ according to the relevant simple power equations yields the empirical equation shown in Figure 6 (see the leftmost equation in the middle panel).

Figure 2 illustrates both the theoretical and empirical equations for $\tilde{P}_{--}(e|ij)$. Substituting the causal-power values inferred from that figure (by the mere mortal) into the theoretical equation for $\tilde{P}_{--}(e|ij)$ yields the value of the expected frequency shown in the bottom panel of Figure 2: $\tilde{P}_{--}(e|ij) = 1 \cdot (1 - 2/3) \cdot (1 - 1/3) = 2/9$. Substituting the corresponding relative frequencies from the top three panels of Figure 2 into the corresponding empirical equation yields the same value:

$$\tilde{P}_{--}(e|ij) = \frac{1/3 \cdot 2/3}{1} = 2/9.$$

As for Figure 1, Figure 2 shows that our mathematical criterion of independent influence corresponds to the intuitive one.

Case 3: Evaluating the Conjunctive Power of i and j to Produce e, When a May Have Simple Generative Power and i and j May Each Have Simple Preventive Power

Until now, our results have not depended on whether the simple and conjunctive causes influence *e* sequentially or in parallel. When at least one of the simple causes is preventive and the conjunctive cause is generative, however, as in the present case (and subsequently for Case 5), these two conceptions of causal influence do yield different results. If the conjunctive cause exerts its influence, if any, after the simple causes have completed their influence—what we refer to as the *sequential conception*—a preventive simple cause would not prevent the cases of *e* generated by the conjunctive cause. Under the parallel conception, however, it would. We assume sequential influence here, and we treat parallel influence later (see *The Assumption That the Conjunctive Cause Operates on the Static Outcome of the Simple Causes*).

Under the sequential conception, if *i* and *j* potentially interact to produce *e*, then, as for Case 1, in the presence of *i* and *j*, *e* will occur if (a) it results from the simple causes or (b) it is produced by the conjunction of *i* and *j*. The probability of (a) is simply $\tilde{P}_{--}(e|ij)$. Using the relevant De Morgan's Law to express the observed probability, we obtain Equation 20:

$$P(e|ij) = 1 - [1 - \tilde{P}_{--}(e|ij)] \cdot (1 - q_{ij}). \quad (20)$$

This equation differs from the observed probability equation for Case 1 only in the subscripts on the expected probability term.

It follows algebraically that the theoretical and empirical equations for q_{ij}, the generative power of the conjunction, are as shown in the middle cells of Figures 5 and 6, respectively. The empirical equation for Case 3 is the "inverse" of that for Case 2, in the sense that where there were conditional probabilities of not-*e* there, there are conditional probabilities of *e* here. The equations are similar, however, in that they both express the respective conjunctive powers most directly in terms of the conditional probabilities of the nonevent.

Figure 2 illustrates the theoretical and empirical equations for q_{ij} in Case 3 for the special case in which $q_{ij} = 0$. For this special case, because there is no conjunctive power, the sequential versus parallel distinction is irrelevant.

Case 4: Evaluating the Conjunctive Power of i and j to Prevent e, When a May Have Simple Generative Power and i and j May Each Have Simple Preventive Power

In contrast, if *i* and *j* potentially interact to prevent *e*, then in the presence of *i* and *j*, *e* would occur if (a) it results from the simple causes and (b) it is not prevented by the conjunction of *i* and *j*. Analogous to Case 2, therefore, the observed probability of *e* may be explained by

$$P(e|ij) = \tilde{P}_{--}(e|ij) \cdot (1 - p_{ij}). \quad (21)$$

The only difference in the equations for Cases 2 and 4 is in the subscripts on the expected probability terms. It follows algebraically that the theoretical and empirical equations for p_{ij}, the preventive power of the conjunction, are as shown in Figures 5 and 6, respectively (see the rightmost equation in the middle panel of each figure).

From Figure 6, one can see that the RHS of the empirical equation for this case is identical to that for Case 1, except that the constituent conditional probabilities in the ratio term here,

$$\frac{P(e|ij) \cdot P(e|\bar{i}\bar{j})}{P(e|i\bar{j}) \cdot P(e|\bar{i}j)},$$

are of *e* (the nonevent here), rather than of not-*e* (the nonevent in Case 1). Thus, the expressions for generative and preventive conjunctive power when *i* and *j* are potentially preventive (Cases 3 and 4) bear identical "inverse" relations to the expressions for preventive and generative conjunctive power, respectively, when *i* and *j* are potentially generative (Cases 2 and 1). When *i* and *j* do not interact,

$$\frac{P(e|ij) \cdot P(e|\bar{i}\bar{j})}{P(e|i\bar{j}) \cdot P(e|\bar{i}j)} = 1,$$

and $p_{ij} = 0$. Figure 2 illustrates this special case.

One Candidate Cause Is Preventive and the Other Is Generative

Finally, we consider the case in which one candidate cause, say *i*, is (potentially) preventive, and the other candidate cause, *j* in this case, is (potentially) generative. Our basketball game attendance example (see *Overview and Assumptions*) was of this type. In these situations, we denote the expected probability of *e* when both *i* and *j* are present but exerted only their simple influences by $\tilde{P}_{-+}(e|ij)$.

Estimating $\tilde{P}_{-+}(e|ij)$

If *i* and *j* exerted only their simple powers on *e*, then when *i* and *j* are both present, *e* occurs if (a) *j* causes it or *a* is present and causes it and (b) *i* does not prevent it. The probability of the union of the two events in (a) may be calculated using the relevant De Morgan's Law. Then, taking the probability of the intersection of (a) and (b) yields the equation shown in Figure 5 (see the leftmost equation in the bottom panel).

A comparison of the theoretical equations for $\tilde{P}_{--}(e|ij)$ and $\tilde{P}_{-+}(e|ij)$ (see Figure 5) reveals that they hold identical implications for observable probabilities: For both situations in which *i* is potentially preventive, the expected probabil-

ity of *e* is equal to how often *e* occurs in the presence of causes *j* and *a*, reduced by how often *i* prevents *e* (i.e., the RHS of each equation is the product of the relevant power expansion of $P(e|\bar{i}j)$ and $(1 - p_i)$). Note that, regardless of whether *j* is generative or preventive, $P(e|\bar{i}j)$ is the estimate of how often *j* and *a* together produce *e*. Thus, the empirical equations for $\tilde{P}_{-+}(e|ij)$ and $\tilde{P}_{--}(e|ij)$ have identical RHSs (see Figure 6).

Figure 7 illustrates the concept of the independent influences of a simple preventive cause and a simple generative cause expressed in the theoretical and empirical equations for $\tilde{P}_{-+}(e|ij)$. To compensate for the added conceptual complexity in this figure, we have grouped the entities that exhibit the same response to a cause. So that the powers of both simple causes are defined, the influence of the composite *a* is assumed to be intermediate (i.e., $0 < P(a) \cdot q_a < 1$), as shown in the first panel of Figure 7. The first two panels allow one to infer that *i* prevents *e* one half of the time when *i* (but not *j*) is added to the composite. Analogously, the first and third panels show that *j* produces *e* one third of the time when *j* (but not *i*) is added. As indicated, this one-third probability applies both to the entities that show *e* due to alternative causes and to those that do not. Now, if *i* and *j* independently influence *e*, then if both *i* and *j* had been present, their influences would have superimposed on each other, as shown in the bottom panel.

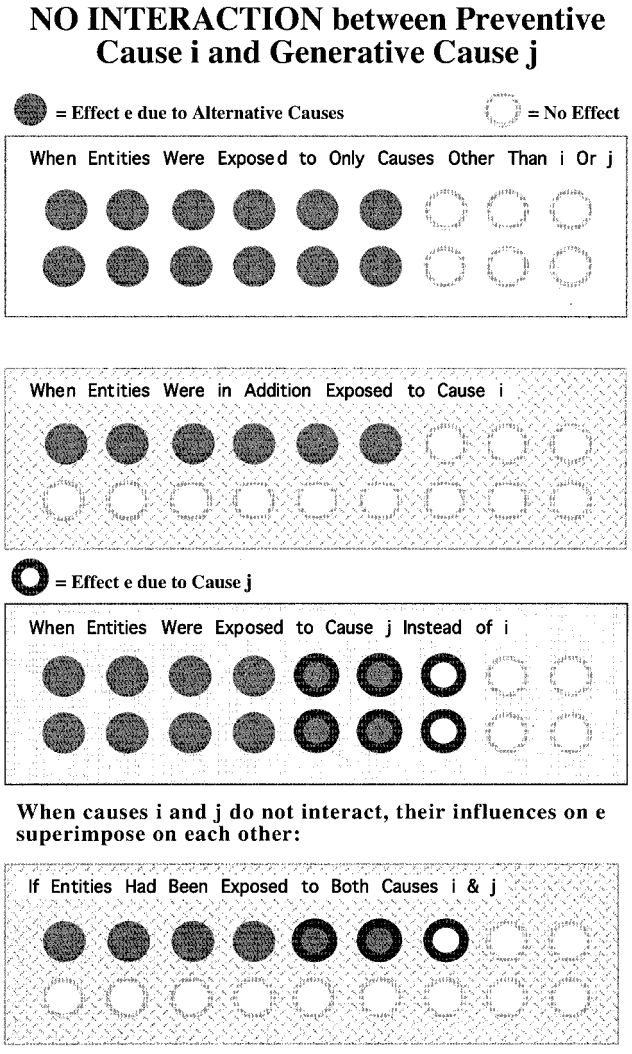

Figure 7. An illustration of the independent influences of a preventive cause and a generative cause on an effect occurring in entities that are presumed to recover completely from the influence of one cause before being exposed to a second one.

To demonstrate that our theory is in accord with the intuition illustrated in Figure 7, we instantiate the empirical equation for $\tilde{P}_{-+}(e|ij)$ with the respective relative frequencies from the top three panels of the figure. This yields the value of the expected frequency shown in the bottom panel of the figure:

$$\tilde{P}_{-+}(e|ij) = \frac{1/3 \cdot 7/9}{2/3} = 7/18.$$

Just as for the other two combinations of simple powers, when one candidate cause is of each type our mathematical criterion of independence corresponds to the intuitive one.

Case 5: Evaluating the Conjunctive Power of i and j to Produce e, When j and a May Each Have Simple Generative Power and i May Have Simple Preventive Power

As for Case 3, we assume that the conjunctive generative cause exerts its influence, if any, on the outcome resulting from the simple causes. Thus, analogous to Cases 1 and 3, the observed $P(e|ij)$ may be expressed as shown in Equation 22:

$$P(e|ij) = 1 - [1 - \tilde{P}_{-+}(e|ij)] \cdot (1 - q_{ij}). \quad (22)$$

The theoretical and empirical equations for q_{ij} for this case are shown in the middle cell in the bottom panel of Figures 5 and 6, respectively.

The empirical equation for this case and that for Case 3, in which both simple causes are potentially preventive, are identical (see Figure 6). They are so because the theoretical equations for q_{ij} for these two cases are identical except that $\tilde{P}_{-+}(e|ij)$ replaces $\tilde{P}_{--}(e|ij)$ (see Figure 5), and the empirical estimates for these two expected probabilities are identical, as we just explained. Figure 7 illustrates the special case in which $q_{ij} = 0$ for both the empirical and theoretical equations for Case 5.

Case 6: Evaluating the Conjunctive Power of i and j to Prevent e, When j and a May Each Have Simple Generative Power and i May Have Simple Preventive Power

In contrast, if *i* and *j* potentially interact to prevent *e*, then in the presence of *i* and *j*, *e* would occur if (a) it results from the simple causes and (b) it is not prevented by the conjunction of *i* and *j*. Analogous to Cases 2 and 4, therefore, the observed probability of *e* may be explained by

$$P(e|ij) = \tilde{P}_{-+}(e|ij) \cdot (1 - p_{ij}). \quad (23)$$

The theoretical and empirical equations for p_{ij} are shown in the rightmost cell in the bottom panel of Figures 5 and 6, respectively.

The empirical equation for p_{ij} in this situation is identical to that for Case 4, in which both simple causes are potentially preventive. Analogous to our discussion of Case 5, this identity follows from the comparability of the respective theoretical equations (see Figure 5) and the equivalence of the empirical estimates for $\tilde{P}_{--}(e|ij)$ and $\tilde{P}_{-+}(e|ij)$. Therefore, as for Case 4, when i and j do not interact,

$$\frac{P(e|ij) \cdot P(e|\bar{i}\bar{j})}{P(e|i\bar{j}) \cdot P(e|\bar{i}j)} = 1,$$

and $p_{ij} = 0$. This special case for the theoretical and empirical equations for Case 6 is illustrated in Figure 7.

Summary of Our Theory of Conjunctive Causal Power

We have presented an analysis of how a reasoner might assess whether two candidate causes interact to influence an effect. Extending Cheng's (1997) theory of simple causes to conjunctive causes, we propose that reasoners consider $\Delta P_{ij} = P(e|ij) - \tilde{P}(e|ij)$, the deviation of the observed probability of an effect e given the presence of both candidate causes i and j from the expected probability of e if the two candidates exerted their simple powers only (Equation 5). To obtain an estimate of the causal power of the conjunction of i and j, reasoners explain each of these two conditional probabilities by the simplest possible conception of the causes of e relevant to that conditional probability (see Table 1). From our set of assumptions, in conjunction with the no-confounding condition, we derived the mathematical results concerning the expected probability of e and generative and preventive conjunctive power summarized in Figures 5 and 6.

As the column labeled $\tilde{P}(e|ij)$ in Figure 6 shows, the empirical equations for the expected probabilities follow two patterns, one for situations in which the simple powers of i and j are both potentially generative, and the other for those in which the simple power of at least one candidate is potentially preventive. These results for $\tilde{P}(e|ij)$, which are illustrated in Figures 1, 2, and 7, imply that different measures of interactive causal influence are applicable depending on the direction of the simple powers.

As the columns labeled q_{ij} and p_{ij} in Figure 6 show, the empirical equations for estimating conjunctive power differ depending not only on the direction of the simple powers (compare the three rows) but also on the direction of ΔP_{ij}.[15] According to our theory, if $\Delta P_{ij} \geq 0$, the reasoner evaluates q_{ij}, the potential generative power of the conjunction of i and j. It is estimated by the potentially positive difference made by the presence of the conjunctive candidate to the frequency with which e occurs among entities in which e is not already produced by causes other than that candidate. Analogously, if $\triangle P_{ij} \leq 0$, the reasoner evaluates p_{ij}, the conjunctive candidate's potential preventive power. It is estimated by the potentially negative difference made by the presence of the conjunction to the frequency with which e occurs among entities in which e is already produced by other causes.

Our results show that conjunctive causal power is analogous to simple causal power. As illustrated in Figure 5, the equations for q_{ij} have a form analogous to Equation 3, the one for simple generative power, and the equations for p_{ij} have a form analogous to Equation 4, the one for simple preventive power. The conjunctive equations differ from their simple power counterparts only with respect to the expected probability of e on their RHSs. As for simple power, conjunctive power allows the reasoner to predict the relative frequency with which the candidate cause influences e in a set of entities in the world, independently of how often other causes in the context might influence e.

Finally, our derivations explain when and why conjunctive causation can be inferred from covariation, thereby explaining some basic principles of experimental design as well as reasoners' corresponding everyday intuitions. They explain why *no confounding* is a requirement for inferring conjunctive causation, just as Cheng's (1997, 2000) derivations do for inferring simple causation. Similarly, the theoretical equations (see Figure 5) for conjunctive power explain two other boundary conditions for inferring interactive causal influence. As we discussed, these equations make predictions regarding situations involving the ceiling effect and its preventive analogue that are in accord with both intuitions and the corresponding principles of experimental design. Thus, these basic principles of design, which are extrinsic to purely covariational measures and either augment or overrule their interpretation, arise from the same assumptions as do the measures under our causal approach, and they are intrinsically incorporated into these measures.

The Relation Between Our Theory and Other Possible Theories

In the sense that our theory applies only to candidate causes and effects of the type specified when the theory's assumptions hold, it is of course merely a specific model. Other assumptions would lead to other models (as we illustrate later with the parallel conception of conjunctive causation; see *The Assumption That the Conjunctive Cause Operates on the Static Outcome of the Simple Causes*). Similarly, causes and effects that are represented by other types of variables (e.g., continuous ones) would require other models. We think, however, that our theory holds a special place in two senses.

Representation of Distal Causal Relations

Our theory explicitly represents distal conjunctive relations rather than proximal ones—that is, those that exist in the world rather than merely in our senses—as the goal of causal discovery, in contrast to all purely covariational models of causation (includ-

[15] When $\Delta P_i = \Delta P_j = 0$, all three expected probabilities are applicable. The values resulting from the various expected probability equations (and likewise those resulting from the applicable conjunctive power measures) agree when the simple generative and preventive powers of i and j all equal 0. For example, suppose e always occurs when i and j are both present but only occurs with a probability of .05 otherwise, so that the simple powers (both generative and preventive) of i and j equal 0. In this case, the expected probabilities ($\tilde{P}_{++}(e|ij)$, $\tilde{P}_{--}(e|ij)$, and $\tilde{P}_{-+}(e|ij)$) would all equal .05, and the q_{ij}s would all equal 1. The only situation in which the various expected probabilities give disagreeing values is when a simple power has an undefined value. For example, instead of letting $P(e|i\bar{j}) = P(e|\bar{i}j) = P(e|\bar{i}\bar{j}) = .05$, let them all be 0 (with $P(e|ij) = 1$ as before). In this case, the simple generative powers of i and j remain at 0, $\tilde{P}_{++}(e|ij)$ is 0, and q_{ij} is 1. But, the simple preventive powers of i and j are undefined, leading $\tilde{P}_{--}(e|ij)$ and $\tilde{P}_{-+}(e|ij)$ and their corresponding q_{ij}s to also have undefined values, as they should.

ing conventional statistics for categorical data).[16] Presumably, distal causal relations remain the goal of causal discovery regardless of the type of variable representing the causes and effects. If so, the same distinction regarding whether distal causal relations are represented explicitly may lead to similar implications for causal inference involving other types of variables under other sets of assumptions. (We review evidence in a later section that untutored reasoners behave as though they are inferring distal causal relations; see Empirical Tests of the Conjunctive Power PC Theory.)

Simplicity of Assumptions

We subscribe to the common view in science that parsimony is a virtue. Compared with other models taking the same distal approach, our assumptions are probably as weak (i.e., general) and as few as they could be to nonetheless allow causal inference regarding conjunctive causes when both simple candidates have causal power. Consider models that infer causal structure without inferring causal strength (Pearl, 2000; Spirtes et al., 2000). Although such structural models make weaker assumptions than ours, they would be able to identify conjunctive causation only when certain patterns of results are observed. In particular, they would do so only for cases in which there is a difference in the state of dependence (i.e., dependent vs. independent) between effect *e* and the conjunction of candidate causes that (a) would be expected assuming that the two candidates influence *e* independently and (b) is observed. For example, if the constituents of the conjunction are both independent of *e*, the conjunction would be expected to be independent of *e*, and any deviation from independence between *e* and the conjunction, which qualitative models can measure, would indicate conjunctive causation. When at least one simple candidate has causal power, however, the same state of dependence would result whether or not there is causal interaction, unless the conjunctive cause coincidentally exactly cancels the influence(s) of the simple cause(s). For the noncoincidental cases, because there would be no difference between the expected and the observed states even when there is causal interaction, qualitative models would be unable to identify conjunctive causation.

Simple models, however, can often be simplistic. We assume that, in the absence of prior causal knowledge, reasoners would consider more complex models when motivated by evidence contradicting a simpler model. For example, they might form hypotheses described by Cheng's (1997) theory of simple power first, but evidence for the violation of the independent-influence assumption in her theory would lead to the consideration of hypotheses described by our theory. In turn, the violation of assumptions in our theory would lead to the consideration of yet more complex hypotheses.

The role of simplicity in guiding theory development motivated our assumption, described in conjunction with Case 3, that reasoners consider the influences of simple and conjunctive causes sequentially. This conception is compatible with the view that reasoners infer a conjunctive cause only after finding that simple causes alone provide an inadequate explanation. In the next section, we consider a different conception of the influences of the simple and conjunctive causes.

Violations of Various Assumptions Underlying the Theory

Our results for $\tilde{P}(e|ij)$ are demonstrably true for situations in which our assumptions hold. Consider a hypothetical case of independent causal influence: Suppose that among the many applicants to a college, 100 are judged to be equally qualified for admission. The college can unfortunately admit only 50 students. There are two admissions officers, each of whom mistakenly assumes that he or she is the one making the final decisions, and each admits 50 on behalf of the college without knowledge of the other's actions. They are the only admissions officers, so that if both of them had been negligent and failed to make any decisions, 0 students would have been admitted. Assuming that their criteria for admission have no related biases (e.g., they each randomly select 50), the expected number of students admitted by the college would be 75. This answer is congruent with our theory but contradicts (a) the interaction contrast from the probabilistic contrast model (Cheng & Novick, 1990, 1992), (b) the summation of associative strengths in the definition of Rescorla and Wagner's (1972) model, and (c) the cross-product ratio, all of which predict that the expected number of students admitted should be 100.

Our assumptions, however, may not hold. In this section, we discuss some implications of violations of two of these assumptions: (a) that alternative causes influence *e* independently of *i, j,* and their conjunction and (b) that the conjunctive cause operates on the static outcome of the simple causes (i.e., the sequential conception of simple and conjunctive causal influence). Even when our particular theory is inapplicable because of a violation of its assumptions, our general approach of representing distal causal relations may nonetheless be applicable for other sets of assumptions.

The Assumption That Alternative Causes Influence e Independently of Candidate Causes i, j, and Their Conjunction

This assumption and its analogue in Cheng's (1997) theory of simple causal power (namely, that the composite *a* influences *e* independently of *i*) are probably nearly always violated. A natural question therefore arises: Why should one bother to read work adopting this assumption? For simple causal power, the answer is that under two conditions, the estimates are nonetheless normative. These conditions are relevant when the assumption of independent influence is violated without the reasoner's knowledge (Cheng, 2000), such as when the interacting factor is unobservable. If the violation involves observable factors only, the reasoner may assess conjunctive power, making the present article rather than Cheng's (2000) article relevant. These conditions are that (a) the unobservable factors that interact with *i* occur with a similar probability across the learning context and the prediction (i.e., transfer) context, and (b) whichever factor or composite of factors in *a* that interacts with *i*—call it *j*—does not do so in such a way that *i* and

[16] Bliss (1939) derived the same expected probabilities of *e* as we did, but we were not acquainted with that work at the time we developed our theory. We are not aware of the distal approach being carried through to the conjunctive cause.

the conjunctive cause composed of *i* and *j* influence *e* in opposite directions (i.e., *i* is generative but the conjunction of *i* and *j* is preventive, or vice versa). Cheng's (2000) analysis explains why in many situations a reasoner is able to make a useful inference regarding simple causal power—for example, that striking a match causes it to light, or that smoking causes lung cancer—despite the obvious violation of the assumption of the independent influence of *i* and *a*. In those cases, the unobservable *enabling conditions* (e.g., oxygen for the lighting of the match) occur with a similar probability across contexts, and they enable rather than disable the generative influence of the candidate (i.e., that of striking a match on lighting).

We have not done a systematic analysis of the implications of the violation of the analogous independent-influence assumption in our conjunctive power theory—an analysis that is much needed.[17] Nevertheless, let us consider an example of this violation and illustrate it by a modification to Figure 1. Suppose (a) that there is some unobservable factor in some of the entities that makes them susceptible to cause *i* and (b) that the same factor prevents them from being susceptible to cause *j*, in such a way that the entities that responded to *i* in the second panel and those that responded to *j* in the third panel become mutually exclusive. In these two panels, because *i* and *j* never both occur, they cannot directly interact with each other. What this pattern of outcomes reveals instead is that there is some interaction involving each of *i* and *j* individually and some common factor in *a*, violating our independent influence assumption. Even though *i* and *j* indirectly interact, it is nonetheless possible that they do not directly interact. For example, suppose that the influences of *i* and *j* shown in the (modified) middle two panels of Figure 1 superimpose in the bottom panel, so that all nine entities show *e* in that panel. It seems to us that the superimposition indubitably indicates that *i* and *j* do not directly interact. But, because our theory only uses information regarding the proportion of entities that show *e* in each panel (in particular, it is oblivious to information on the proportion of entities that show *e* both in the second panel and in the third), it will judge that *i* and *j* interact to influence *e*. What then does an indication of interaction or independence according to our measures mean?

One interpretation is that a conclusion of independence resulting from our measures implies that *i* and *j* do not interact to influence *e* in any way, directly or indirectly (unless there is some coincidental canceling of interactive influences). Independence as indicated by our measures is the cleanest possible type of independence. A conclusion of interaction resulting from our measures, however, does not necessarily imply a direct interaction between *i* and *j*, but could also indicate an indirect one via some common third factor.

To discern the exact manner in which *i* interacts with *j*, more analysis would be needed on additional information. If the factor in *a* that interacts with *i* and with *j* is observable, these respective interactions would be assessable by applying our conjunctive power analysis to them. There is no circularity in that the composite *a* is progressively stripped down. Even if this factor in *a* is not observable, however, these interactions are assessable using our approach, but information of the type in Figure 2 must be available. Specifically, there must be information about the presence or absence of each causal candidate and of the effect from a repeated-measures design in which the entities can be reasonably assumed to recover from exposure to each candidate cause alone. These implications of violating the independent-influence assumption in question are not particular to our approach, but pertain to any assessment of interactive causal influence.

The Assumption That the Conjunctive Cause Operates on the Static Outcome of the Simple Causes

The Sequential (Cure) Conception

In Cases 3 and 5, we assumed that the influence of the conjunctive cause on *e* is not further subjected to the influence of the simple causes. An everyday example of sequential influences would be a medicine curing a disease: The disease (the outcome produced by some cause) first occurs, then the medicine cures it. As we mentioned, the ordering of simple and conjunctive influences we have been assuming is consonant with the view that conjunctive power is assessed only when evidence indicates that the explanation in terms of simple powers alone is inadequate.

We believe that the sequential conception is parsimonious and psychologically plausible; it is probably the default conception. However, it is not the only possible or even reasonable conception to consider. In the following section, we briefly discuss an alternative conception.

The Parallel (Suppressant) Conception[18]

It is possible that the influences of simple and conjunctive causes act in parallel. This conception does not alter the explanations of $\tilde{P}(e|ij)$, as those probabilities do not involve any conjunctive causation. The expected probabilities therefore remain as shown in Figures 5 and 6. The explanations of $P(e|ij)$ do change, however, yielding different estimates of conjunctive power in two of the six cases we considered—when the conjunction may have generative power and at least one of the simple causes has preventive power (Cases 3 and 5).

The critical difference lies in the conception of prevention. In the parallel conception, prevention acts like a general suppressant rather than a cure. An example would be the current sustained drug regimen for HIV-positive patients, which both blocks the progression of AIDS and relieves the symptoms that have already appeared. Because all causes act in parallel, the temporal ordering of the occurrence of the candidate causes does not matter.

In the Appendix, we expand the power explanation of $P(e|ij)$ under the two conceptions for Case 3 to explain the exact difference between them. A consequence of the suppressant assumption is that the upper bound on $P(e|ij)$ is less than 1. Even if the generative causes, including the conjunction, succeed in producing *e* in all entities, only a fraction of these occurrences of *e* will be prevented by neither *i* nor *j*. For Case 3, in which both *i* and *j* may have simple preventive power, the upper bound is $(1 - p_i) \cdot (1 - p_j)$. For Case 5, in which *i* may have simple preventive power and *j* may have simple generative power, the upper bound is $(1 - p_i)$. In contrast, the upper bound on $P(e|ij)$ is always 1 according to the cure conception.

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[17] We thank Clark Glymour for bringing to our attention various such implications.

[18] This is the conception cited in Cheng's (2000) article.

That the upper bound on $P(e|ij)$ is less than 1 means that some possible patterns of data are excluded. Consider the empirical versions of the *suppressant* equations for generative conjunctive power. Our derivations indicate that these equations for Cases 3 and 5 are, respectively,

$$q_{ij} = \frac{P(e|\bar{i}\bar{j}) \cdot [P(e|ij) \cdot P(e|\bar{i}\bar{j}) - P(e|i\bar{j}) \cdot P(e|\bar{i}j)]}{P(e|i\bar{j}) \cdot P(e|\bar{i}j) \cdot [1 - P(e|\bar{i}\bar{j})]} \quad (24)$$

and

$$q_{ij} = \frac{P(e|ij) \cdot P(e|\bar{i}\bar{j}) - P(e|i\bar{j}) \cdot P(e|\bar{i}j)}{P(e|i\bar{j}) \cdot [1 - P(e|\bar{i}j)]}. \quad (25)$$

If these equations are applied to patterns of data that are incompatible with the suppressant assumption, the numerical estimates for conjunctive power would be nonsensical. For example, assume that when both simple causes are absent, *e* occurs with a probability of .8. When one cause is present and the other is absent, *e* occurs with a probability of .4. Finally, when both causes are present, *e* always occurs (i.e., $P(e|ij) = 1$). This last conditional probability is impossible under the suppressant assumption, because *e* is constantly under suppression by the simple preventive causes. Given the conditional probabilities in this example, the relevant suppressant empirical equation (Case 3) will yield $q_{ij} > 1$, which means that the suppressant assumption is violated and the equation does not apply.

In contrast, there is no pattern of data that rules out the cure conception. For example, applying the *cure* empirical equation for Case 3 (see Figure 6) to the above conditional probabilities of *e* sensibly yields $q_{ij} = 1$. Some existing data on reasoners' causal attributions, discussed in the next section, provide evidence favoring the sequential (cure) conception.

Empirical Tests of the Conjunctive Power PC Theory

Overview

In this section, we report, evaluate, and summarize empirical tests of our conjunctive power PC theory. We divide the evidence into two types: tests on (a) judgments of whether there is interactive causal influence and (b) estimated magnitudes of conjunctive power (including magnitudes of zero).

For the first type of judgment, when the simple powers of both candidate causes are nonzero, our theory makes predictions that invariably contradict those of all other theories. Figures 1, 2, and 7 are tests involving this type of situation, and our theory's predictions should be intuitively compelling. To provide further support for this type of situation, we report the results from a new experiment showing that naive subjects respond in accord with readers' intuitions. Regardless of how many candidate causes have nonzero simple power, our theory in addition makes the unique prediction of abstention from causal judgment when a predicted boundary condition for causal inference is violated. In other situations, as can be shown analytically, our theory and the probabilistic contrast model (Cheng & Novick, 1990; see also Rothman & Greenland, 1998) coincide in their predictions on whether there is interactive causal influence. Cheng and Novick (1990) reported experimental results concerning each of these three types of situations. Only some of these results, however, are interpretable for the present purpose.

For the second type of judgment, that of the estimated magnitudes of conjunctive causal power, our new theory makes unique predictions regardless of the simple powers of the candidate causes. White (1998) tested this type of judgment in situations in which the candidate causes each have nonzero simple power. Even in the simple situation in which both candidates have simple powers of zero (but alternative causes may have nonzero simple power), no other account makes the same ordinal predictions as does our theory. We summarize an experiment by Clifford and Cheng (2000) involving this type of situation. Our examples of conjunctive causation in the introduction, concerning the typical causes of flu, lung cancer, and success, are common examples of this type of situation. Mackie's (1974) well-known INUS condition (an insufficient but nonredundant part of an unnecessary but sufficient condition) also concerns this type of situation.

Tests on Whether There Is Interactive Causal Influence When Both Candidate Causes Have Simple Power

Figures 1, 2, and 7. Our no-interaction figures elicit strong intuitive agreement that there is no interaction between the two candidate factors *i* and *j* in their influences on effect *e.* Outcomes that deviate from that shown on the bottom panel of each figure, such as those we discussed, would elicit equally strong intuitive agreement that *i* and *j* interact to influence *e.* As straightforward as these predictions are, no account of interactive causal influence, except ours, predicts them. We have illustrated that our probabilistic contrast model, the best-supported previous psychological model of conjunctive causal discovery, erroneously infers that *i* and *j* interact in two of those figures (Figures 1 and 2). All conventional statistical models for categorical data make the same erroneous inference, as we discuss in The Normative Assessment of Conjunctive Causation.

A feature of our figures makes them straightforward tests of our theory. These figures visually convey the information that causes of *e* other than *i* and *j* do not interact with those candidates (Assumption 2) by displaying entities in the middle two panels that respond to each candidate independently. For example, the probability that entities in Figure 1 both turn dark gray in the second panel and have a black ring in the third panel is the product of the probability of the constituent events—dark gray entities in the second panel and black rings in the third panel. There is no analogue of this visual specification in a between-subjects situation, even though the same assumption may hold.

Superimposition experiment. To test our predictions concerning independent influence, we created four problems that were variants of our no-interaction figures. These problems showed the mere mortal's perspective, with the outcome in the bottom panel omitted. Figure 8 is a black-and-white version of a screen that subjects saw for one of the problems. The problems each involved the exposure of entities (allergy patients) to two simple causes (allergy medicines), both generative for two problems and both preventive for the other two. The effect in question was the occurrence of headaches, a potential side effect of the medicines. Exposure to a medicine was represented graphically by two identically colored capsules in a glass of water (a different color was

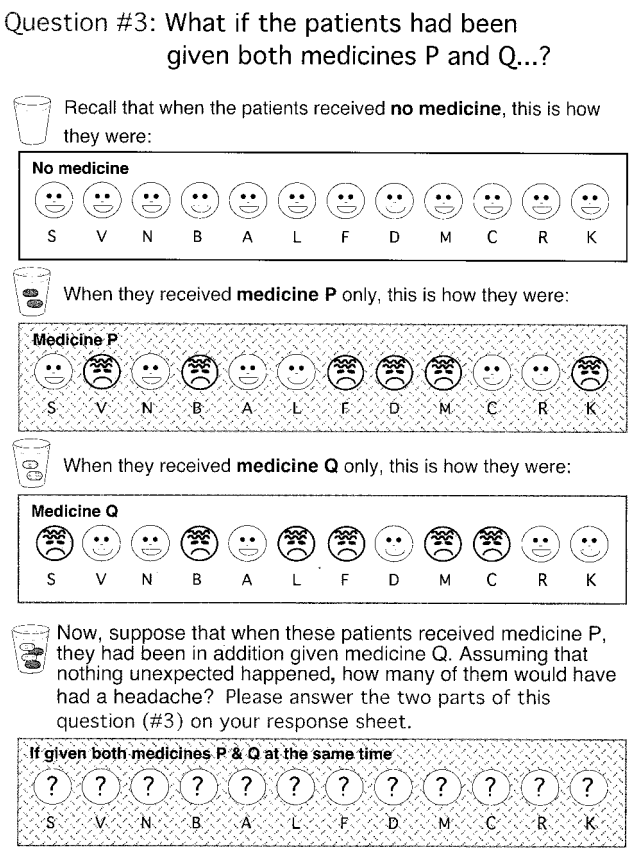

Figure 8. A black-and-white version of the information provided for one problem in which subjects were asked to predict the data pattern that would indicate the independent influence of candidates *i* and *j* when they are jointly present.

used for each medicine). Nonexposure to the medicines was represented by a glass without the capsules.

For both preventive problems, the first panel showed that all allergy patients had headaches when they received neither medicine. One problem, which presented outcomes for 9 patients, was a direct analogue of Figure 2: Every dark gray circle in that figure was replaced by a frowning face, indicating headache, and every light gray ring in that figure was replaced by a happy face, indicating no headache. The drugs therefore respectively prevented headaches for two thirds and one third of the patients. In the other problem, in which there were 12 patients, the second and third panels each showed that 6 patients had headaches, with 3 having headaches in both panels, as would be expected if the patients responded to the two drugs independently. The drugs therefore each prevented headaches for one half of the patients. The two generative problems were exact analogues of the preventive problems, with values of the outcome (headache and no headache) reversed throughout. For example, the first panel showed that none, rather than all, of the patients had headaches when they received neither medicine (see Figure 8). The drugs in these problems therefore caused headaches with the same probabilities as their preventive counterparts prevented them.

Forty undergraduate students enrolled in an introductory psychology class at University of California, Los Angeles, were randomly assigned to receive one of the four problems ($n = 10$ for each problem). The four panels for a problem were presented sequentially and cumulatively on a computer screen until all panels appeared (as in Figure 8). (There was an added screen intervening between the presentation of the middle two panels, which showed a panel depicting the complete recovery of the patients from the first medicine to their original state before the second medicine was given [not shown in Figure 8].) The subjects were asked about the influence of each medicine on headaches as the information was presented.

The fourth panel, which concerned the hypothetical situation in which both medicines had been given, depicted faces covered with question marks (see Figure 8). Subjects were then asked a series of questions that tested the novel predictions of our theory. First, they were asked, "Assuming that nothing unexpected happened, how many [of the patients in this panel] would have had a headache?" To answer this question, subjects first indicated whether more or fewer patients would have had a headache in the bottom panel than in either of the two middle panels, and then they explained their answer. The response options were "more," "fewer," and either "neither" (for the problems with 9 patients and unequal simple powers) or "just as many" (for the problems with 12 entities and equal simple powers). We included this qualitative question to discourage subjects from doing arithmetic mechanically without thinking about causality. All but 2 of the 40 subjects gave answers to this question that were consistent with our theory. (The 2 subjects whose answers were inconsistent with our theory received the preventive problem with 9 entities.)

Then came the critical question—"What is the most likely number of patients who would have had a headache in the bottom panel?"—followed by a blank. The predictions of our theory are as follows: (a) 9 for the generative problem with 12 entities (see Figure 8), (b) 7 for the generative problem with 9 entities (see Figure 1), (c) 3 for the preventive problem with 12 entities, and (d) 2 for the preventive problem with 9 entities (see Figure 2). Collapsed across the four problems, the vast majority of subjects (37 of 40) gave the answer predicted by our theory. Only 1 subject (who received the generative problem with 9 entities) gave the answer predicted by our old probabilistic contrast model (Cheng & Novick, 1990, 1992), the Rescorla–Wagner (1972) model, and the cross-product ratio (i.e., all patients have a headache for the two generative problems and no patients have a headache for the two preventive problems). The other 2 subjects gave other answers with incoherent justifications (these were the same subjects who gave nonconforming answers to the previous question).

For further confirmation of their most preferred response, subjects were next shown two patterns of possible results for the bottom panel (with the order of presentation counterbalanced) and were asked to indicate the pattern with which they agreed. One pattern corresponded to the prediction of our theory (analogous to the bottom panels in Figures 1 and 2); the other corresponded to the prediction of the other models (Cheng & Novick, 1990, 1992; Rescorla & Wagner, 1972; and the cross-product ratio). For this question, 39 of the 40 subjects picked the pattern predicted by our theory to indicate independent influence. The lone exception was the subject whose numeric response to the previous question matched the alternative pattern.

Finally, subjects were asked whether the pattern they had just selected is the best possible answer to the question of what would

have happened if both medicines had been given at the same time. For this question, 37 subjects gave an answer consistent with our theory: 34 indicated that the pattern predicted by our theory was the best possible answer, and 3 indicated that it was not necessarily the best because the drugs might interact (note that these subjects spontaneously explained a deviation in terms of an interaction). Only 1 subject said that the other pattern was the best possible answer (the subject who spontaneously predicted that pattern). Clearly, the results of this experiment strongly favor our theory over other accounts.

Cheng and Novick (1990). In their experiment, Cheng and Novick (1990) tested a set of inference problems (Problems 10, 11, and 12) in which two candidate causes each had a simple generative power of 1: The effect in question never occurred when both candidates were absent but always occurred when either candidate was present without the other. In addition, the effect always occurred in the joint presence of the two causes. Thus, the interaction contrast for these factors from the probabilistic contrast model (Equation 2) has a value of -1, implying preventive interactive influence. In contrast, according to our conjunctive power PC theory, $\Delta P_{++} = 0$, implying that subjects should not indicate that this conjunction is a cause. In fact, most subjects did not. Unfortunately, this result is ambiguous: The conjunctive cause predicted by the probabilistic contrast model is preventive, and our response format did not distinguish between generative and preventive causes. As we elaborate in the next section, subjects were simply asked to explain a target event by choosing from a given list of response categories. Because a preventive cause cannot explain the occurrence of the effect in question in the target event, subjects might have omitted the relevant response category despite their perception of preventive efficacy.

When Only One Candidate Cause Has Simple Power

Overview of Cheng and Novick (1990). Subjects were given short scenarios describing the occurrence of a certain effect over various values of people, stimuli, and occasions (the candidate causal dimensions). To ensure that their responses reflected inferences based on the experimental materials rather than on knowledge retrieved from memory, the problems involved fictional events that were said to occur in an imaginary land where people's customs and preferences often differed from our own. For example, one problem (Number 3) was as follows (Cheng & Novick, 1990, p. 565):

Adam thinks that narcissus (a flower) smells nice on this occasion.

1. In fact, Adam has always thought that narcissus smells nice.
2. But nobody else has ever thought that narcissus smells nice.
3. Everyone has always thought that all other flowers smell nice.

On the basis of such information, subjects were asked to explain what caused the italicized target event by choosing one or more responses from a list of seven given beneath the problem. The first three responses corresponded to the three possible simple causes: the target person, stimulus, and occasion, respectively. For example, the stimulus attribution for the narcissus problem was "there is something special about narcissus in general." The next three responses corresponded to the three possible conjunctive causes involving combinations of two of the three factors. For example, the Person $\times$ Stimulus interaction attribution was presented as "there is something special about the combination of Adam and narcissus (only when they are together)." The final response corresponded to the three-way interaction.

Note that the variables in the stimuli were not binary. Therefore, our new theory applies only if the stimuli were recoded as binary by subjects, for example, to represent that there is something special about the target person that is absent in other people. That such recoding occurred is not implausible given the wording of the response categories and the references to the nontarget values on each dimension in the aggregate (e.g., nobody, everyone, always, all other).

Cheng and Novick's (1990) materials consisted of various sets of three problems in which the problems within each set were logically equivalent; they were merely the three possible rotations of the same logical structure in the Person $\times$ Stimulus $\times$ Occasion space. They were so designed to test a possible bias for perceiving the person dimension as causal more often than other dimensions (i.e., the fundamental attribution error; see Nisbett & Ross, 1980). For our purpose here, the problems in each set were simply replications instantiated with different stimulus materials conducted on different groups of subjects. There were four sets of problems for which the probabilistic contrast model, our conjunctive power PC theory, or both, predict a two-way interaction attribution (e.g., the conjunction of Adam and narcissus in the example). We considered one of these sets in the previous section. Of the remaining three, only two have results that shed light on our new theory. We consider these two sets in detail here.

Results. For the situation in which only one candidate cause has nonzero simple power, our new theory and our old model concur in their predictions regarding whether there is a conjunctive cause when no boundary condition for that inference is violated. Two sets of problems (one consisting of Problems 2, 5, and 8—$N = 66$ for the set; the other consisting of Problems 3, 6, and 9—$N = 72$ for the set) tested in Cheng and Novick's (1990) study concerned this situation. These problems had probabilistic contrasts that yielded predicted causal attributions corresponding to the six possible combinations of (a) a conjunctive cause consisting of two factors (e.g., Person $\times$ Stimulus) and (b) a simple cause consisting of one of the constituents of the conjunction (Person or Stimulus for the example). It is interesting to note that for these problems, a boundary condition for inferring conjunctive causation was violated according to the parallel (suppressant) conception of conjunctive causation but not the sequential (cure) conception, the one that we favor as a default.

The pattern of covariation was as shown in Figure 9. None of the accounts under consideration predicts more than one conjunctive cause; we therefore label the factors composing the predicted conjunctive cause *i* and *j* here for expository purposes. This pattern indicates that the simple preventive power of *i* (narcissus in the example) is 1, and the simple preventive powers of *j* (Adam) and *k* (this occasion) are 0. Collapsing across *k*, the interaction contrast for *i* and *j* from the probabilistic contrast model (Equation 2) has a value of 1, predicting that the conjunction of those factors (Adam & narcissus) is a generative cause. Likewise, according to the sequential (cure) conception of the conjunctive power PC theory, ΔP_{-+} and ΔP_{--} each equals 1, implying that $q_{ij} = 1$. Thus, these

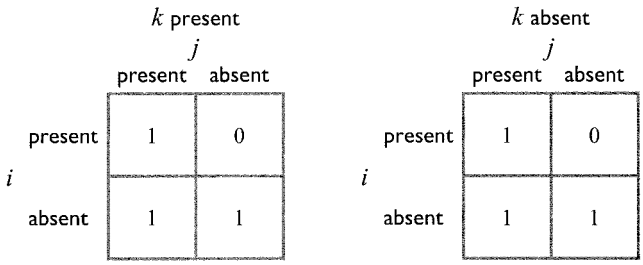

Figure 9. A data pattern used by Cheng and Novick (1990) for six of their causal attribution problems (Nos. 2, 3, 5, 6, 8, and 9).

two views concur in predicting that reasoners should identify that conjunction as a cause. In contrast, according to the parallel conception (Equations 24 and 25), the generative power of this conjunction has an undefined value, leading to the prediction that this conjunction should not be identified as a cause.

The results favor both our old probabilistic contrast model and the sequential conception in our new conjunctive power PC theory over the parallel conception: The predicted conjunctive cause was chosen an average of 70% of the time across the six problems (N = 138 total responses). By comparison, the unpredicted conjunctive causes were chosen an average of only 4% of the time.

Tests on Estimated Magnitudes of Conjunctive Causal Power

White (1998)

In his Experiment 1, White (1998) tested 40 college students on groups of problems for which the two candidate simple causes had nonzero powers ranging from .25 to .75. Subjects were asked to judge the "likelihood that the cause of the effect" was Candidate A, Candidate B, and "some kind of interaction between A and B" (White, 1998, p. 141). Each judgment was made on a 100-point scale in which 0 meant *definitely not the cause of the effect* and 100 meant *definitely the cause of the effect.* This experiment was not designed to test our conjunctive power theory. For example, the type of question White used does not separate causal strength from reliability (see Tenenbaum & Griffiths, 2001). Moreover, like Cheng and Novick's (1990) dependent measure, this one does not allow subjects to distinguish between generative and preventive influence (for 2 of the 11 problems, A and B were both preventive; for a 3rd problem, the conjunction was preventive). Judgments concerning preventive causes are therefore difficult to interpret. Nevertheless, it is possible to isolate two sets of comparisons, involving 6 of the 11 problems, that minimize these concerns. These comparisons reveal that White's results concerning estimated magnitudes of conjunctive causal efficacy in situations in which the simple candidate causes have nonzero power strongly favor our conjunctive power theory over alternative accounts.

We first consider three problems (White, 1998, Data Sets 1.1, 1.2, and 1.7) for which our probabilistic contrast model (Cheng & Novick, 1990) and the cross-product ratio both predict no interaction between A and B. In contrast, according to our conjunctive power PC theory, the power of the interaction in these problems is 1. Consistent with our conjunctive power theory, the mean judged likelihoods for the interaction were quite high (overall mean judgment = 71.52). Even the lowest of these mean judgments is reliably greater than 0. Moreover, these three conjunctive candidates, along with three of the four other conjunctive candidates for which our new theory predicts that the power is 1, received a higher mean judgment than any candidate in the experiment, simple or conjunctive, for which our new theory predicts a causal power less than 1.

Next, we consider the mean likelihood judgments for the interaction in three problems (White, 1998, Data Sets 1.3, 1.4, and 1.5) for which the probabilistic contrast model predicts equal conjunctive causal strength, whereas our new theory predicts decreasing conjunctive power (see Table 2; unlike the other data sets, Set 1.4 had simple powers of 0). The cross-product ratio predicts increasing likelihoods from Data Set 1.3 to Data Sets 1.4 or 1.5 (note the increasing values above 1 on this criterion in the table) and no difference between the latter two. In support of our conjunctive power theory, the mean judged likelihoods for the interaction decreased across problems (see Table 2), with a reliable difference between each pair of judgments. Confounding by reliability should not be an issue for these problems: The expected probability of the outcome decreased across problems, leading to increasing reliability for generative conjunctive power (see Buehner & Cheng's,

Table 2
Event Frequencies, Corresponding Indicators of Conjunctive Causation, and Observed Means in Three Conditions of White's (1998) Experiment 1

	Relative frequency of the effect				Predictions of alternative models		(Conjunctive) power PC predictions			Mean judgments
Data set	A & B present	A only present	B only present	A & B absent	Interaction contrast	Cross-product ratio	Simple powers of A and B[a]	Expected probability of effect[b]	Conjunctive power[a]	Likelihood of A × B interaction
1.3	16/16	4/16	4/16	0/16	0.50	7.72	0.25	0.4375	1.00	86.18
1.4	12/16	4/16	4/16	4/16	0.50	9.00	0.00	0.2500	0.67	65.51
1.5	8/16	4/16	4/16	8/16	0.50	9.00	−0.50	0.1250	0.43	39.41

[a] For all three data sets, the simple powers of Candidates A and B were identical. Simple powers were calculated according to Cheng's (1997) power PC theory, with a minus sign added to mark preventive power. Conjunctive powers were calculated according to our new conjunctive power PC theory. [b] The expected probability of the effect when A and B are both present, assuming that they exerted only their simple influences. The smaller the expected probability, the larger the effective sample size for estimating conjunctive causal influence.

1997, explanation of variations in effective sample size, even when the actual sample sizes do not vary).[19]

White's (1998) results allow another type of evaluation of our old and new accounts of conjunctive causal influence. Recall that our conjunctive power theory, unlike our probabilistic contrast model, allows comparison between estimates of causal strength for simple and conjunctive causes. Seven of White's problems (Data Sets 1.1, 1.2, 1.3, 1.6, 1.7, 1.10, and 1.11) had a conjunctive generative power of 1.0 but simple powers ranging from .25 to .75. In support of our causal-power approach, for each of these problems, the observed judged likelihood of the interaction was higher than the judgments for the two simple candidate causes (the overall mean judged likelihood for the conjunction was 74.35, compared with 39.49 for the 14 simple causes).

Clifford and Cheng (2000)

Overview. We now turn to the results of an experiment measuring estimated magnitudes of conjunctive power. Clifford and Cheng (2000) tested some such predictions of our conjunctive causal-power theory against those made by the most viable purely covariational accounts.[20] Their experiment concerned a simple situation involving conjunctive causation: Two candidate causes of an effect *e*, labeled *i* and *j* for the purpose of exposition here, have no simple causal power but may (or may not) in combination influence (i.e., generate or prevent) *e*. The *base rate* of *e* (i.e., the probability of *e* in the absence of the conjunctive candidate) varied across problems; because the component factors had no simple power, there was a single base rate of *e* within a problem (whenever *i* and *j* were not both present, *e* occurred with the same probability, determined by how often alternative causes produced *e*). The candidates having no simple power implies that they had no simple preventive power, a condition for distinguishing the sequential and parallel conceptions of conjunctive causation; the results of this study therefore do not distinguish between these conceptions, but instead test what is common to them against alternative accounts.

In this situation, Cheng and Novick's (1990, 1992) interaction contrast model reduces to the classical ΔP model (Jenkins & Ward, 1965; see Equation 1), with the modification that the conjunction of *i* and *j* is treated as a single elemental unit—whenever *i* and *j* are not both present, the candidate (i.e., the conjunction of *i* and *j*) is considered absent. Rescorla and Wagner's (1972) model may be modified analogously to treat the conjunction as a single elemental unit (see Clifford & Cheng, 2000). The predictions of that model regarding elemental units would therefore apply to the conjunction.

To test these accounts, Clifford and Cheng (2000) compared subjects' estimates of conjunctive causal strength across meaningful subsets of problems. One set of comparisons involved conjunctive candidates that had identical positive, negative, or zero ΔP_{ij} (or simple ΔP for the conjunction; the two contrasts are equivalent here), but varied with respect to the base rate of *e*. Our conjunctive causal-power theory, like Cheng's (1997) theory of simple causal power, predicts that with an increase in the base rate of *e*, estimates of the causal strength of such conjunctive candidates with the same ΔP_{ij} will increase when ΔP_{ij} is positive, decrease when ΔP_{ij} is negative, and remain at zero when ΔP_{ij} is zero (see equations in Figure 5). A second set of comparisons involved candidates with the same conjunctive power but varying values of ΔP_{ij}. Our theory predicts that causal estimates for such candidates will not differ. No purely covariational theory predicts either pattern of results. These divergent predictions reflect the key difference between the two approaches in what is assumed to be invariant across contexts: the (unobservable) distal causal relation in the derivation of our conjunctive power measures versus the (observable) proximal association in all purely covariational models.

Design. There were 10 within-subject conditions, corresponding to 10 conjunctive candidates that varied in their power and ΔP_{ij} (see Table 3, first four columns). Each condition (i.e., each causal inference problem) involved a different combination of two candidate factors (medicines). The conditions that involved generative conjunctive candidates and those that involved preventive ones were mirror images of each other. Some pairs of conditions varied the base rate of *e* for conjunctive candidates with the same level of ΔP_{ij}, yielding different conjunctive powers across conditions when ΔP_{ij} was nonzero (GH vs. CD and MN vs. IJ) but no such change in conjunctive power when ΔP_{ij} was zero (OP vs. QR). Other pairs varied ΔP_{ij} for conjunctive candidates with the same causal power (CD vs. EF and IJ vs. KL). Finally, there were two pairs of candidates for which ΔP_{ij} and conjunctive power predict differences in opposite directions (EF vs. AB and KL vs. ST).

The differential predictions made by our theory and the ΔP model (Jenkins & Ward, 1965) or interaction contrasts (Cheng & Novick, 1990) should be clear from Table 3. Rescorla and Wagner's (1972) model makes the same predictions as does the ΔP model if its learning parameter, β, is restricted to remain constant in the presence of *e* (the unconditioned stimulus in its terminology) and in its absence (Chapman & Robbins, 1990; Cheng, 1997). Without this restriction, as long as the ordering of the values of β is consistent (e.g., $\beta_{US} > \beta_{\overline{US}}$ for all data points in a condition), this model predicts that the base rate of *e* should influence causal strength in the same direction for candidates with an equal positive ΔP_{ij} and for those with an equal negative ΔP_{ij} (e.g., see Danks, 2003; Wasserman, Elek, Chatlosh, & Baker, 1993). Specifically, the (absolute) magnitudes of the judged causal strengths will be smaller as the base rate of *e* increases for any fixed positive or negative ΔP_{ij} if $\beta_{US} > \beta_{\overline{US}}$ but larger if $\beta_{US} < \beta_{\overline{US}}$.

The cross-product ratio predicts the same ordinal pattern of causal judgments as does ΔP or interaction contrast except for conditions CD and IJ (see the fifth column in Table 3). A value of 1 on this criterion indicates no interaction. Increasingly large values above 1 indicate increasingly strong generative causes, whereas increasingly small values below 1 indicate increasingly strong preventive causes. CD is a critical medicine combination for which ΔP predicts the same low value as for GH, whereas con-

[19] There were some groups of problems for which the conjunctive power was equally 1, but the judged likelihoods were observed to reliably differ. All such differences, however, concerned cases in which there was a corresponding difference in reliability, due to differences in the effective sample size for estimating the expected probability of the outcome or in the actual sample size for estimating the observed probability of the outcome. The ambiguity of the response scale is likely to have exacerbated the confounding by variation in reliability.

[20] We thank Deborah Clifford for her helpful comments on our account of this experiment.

Table 3
Measures of Conjunctive Causation With Corresponding Event Frequencies, Theoretical Predictions, and Observed Medians and Means in the 10 Conditions of Clifford and Cheng's (2000) Experiment on the Estimation of Conjunctive Causal Influence (N = 60)

Medicine and condition labels	Measures of conjunctive causation				Observed frequencies of headaches, the effect		Causal power predictions	Results	
	Generative conjunctive power	Preventive conjunctive power	ΔP or interaction contrast	Cross-product ratio	Frequency of e given c, the conjunctive candidate	Frequency of e given not-c[a] (i.e., "base rate of e")	No. of people[b]	Median observed no. of people[c]	Mean observed no. of people ± SD[d]
GH	0.50		0.50	73	18/36	0/36	50	50.0	49.6 ± 3.2
CD	0.75		0.50	10	30/36	12/36	75	75.0	69.3 ± 23.1
EF	0.75		0.75	211	27/36	0/36	75	75.0	71.0 ± 16.2
AB	1		0.50	73	36/36	18/36	100	100.0	88.3 ± 26.6
OP	0	0	0	1	12/36	12/36	0	0.0	−0.6 ± 4.3
QR	0	0	0	1	24/36	24/36	0	0.0	0.1 ± 11.0
MN		0.50	−0.50	0.01	18/36	36/36	−50	−50.0	−45.7 ± 23.1
IJ		0.75	−0.50	0.10	6/36	24/36	−75	−75.0	−70.0 ± 18.9
KL		0.75	−0.75	0.00	9/36	36/36	−75	−75.0	−72.3 ± 13.0
ST		1	−0.50	0.01	0/36	18/36	−100	−100.0	−91.0 ± 26.6

[a] not-c implies the three situations, individually and collectively, in which the patients did not receive both medicines. [b] The estimated changes in the number of people out of 100, according to causal power, who would have headaches if given the indicated combinations of medicines. [c] The medians of the observed estimated changes in the number of people out of 100 who would have headaches. [d] The means (± one standard deviation) of the observed estimated changes in the number of people out of 100 who would have headaches.

junctive power predicts the same high value as for EF. The cross-product ratio predicts an even lower ordinal value than does ΔP. IJ is the preventive analogue of CD for which analogous differences in predictions apply.

Materials and task. As the goal of Clifford and Cheng's (2000) experiment was to study people's natural capability to assess conjunctive causation, with as little interference as possible from other mental processes, their materials and task were designed to minimize demands on comprehension, attention, and memory. For the same reason, they were designed to isolate the judgment of causation, differentiating it from variables that are not specific to causal judgments, such as the reliability of the judgment.

The following general cover story preceded the 10 problems in Clifford and Cheng's (2000) study:

> You are an employee for a company that distributes new medicines for the treatment of allergies and arthritis. Your job is to review information regarding a possible side-effect of the new medicines under consideration for distribution. Although they have been found to be clearly effective in treating their respective illnesses, these medicines may, either individually or in combination, cause headaches, prevent headaches, or have no influence at all on headaches.
>
> You will see the results of experiments that were conducted to study the influence of these medicines on headaches. For each experiment, participants were *randomly assigned* to one of four groups: one that received no medicines at all (the control group), one that received one of the medicines, one that received a different medicine, and one that received both medicines together. All participants in these experiments had both arthritis and allergies. Based on the data presented, judge whether each individual medicine and combination of medicines has a side-effect on headaches, and if so, whether it causes or prevents them. Your success in the company is highly dependent on your accurate assessment of these side-effects.

Notice that the cover story states that the patients in the studies were randomly assigned to the four groups. The purpose of this information was to encourage subjects to assume that alternative causes were constant across situations. This assumption of independent occurrence is a precondition for testing our theory's predictions on the values of conjunctive power.

Each candidate cause (i.e., each allergy or arthritis medicine) was denoted by a single letter label, *A–T*. The outcome, *e*, was headache in the patients. As in our superimposition experiment, a blue frowning face represented a patient with a headache, and a white smiling face represented a patient without a headache. Thus, in each study, a trial consisted of a single patient to whom a particular medicine or combination of medicines had been administered, or not, and who either frowned or smiled.

Figure 10 shows a black-and-white version of the computer screen that illustrated the results of a study testing the conjunctive cause consisting of the combination of Medicine C and Medicine D. As shown in the figure, each problem presented information about the results for 144 patients (i.e., trials) in a given study: 36 in each of the four possible treatment conditions. The resulting set of four relative frequencies of headaches allowed the estimation of $P(e|ij)$ and $\tilde{P}(e|ij)$ in our theory as well as ΔP in covariational theories and the cross-product ratio.

For each problem, subjects estimated the strength with which each candidate cause influences *e*. They first assessed the causal

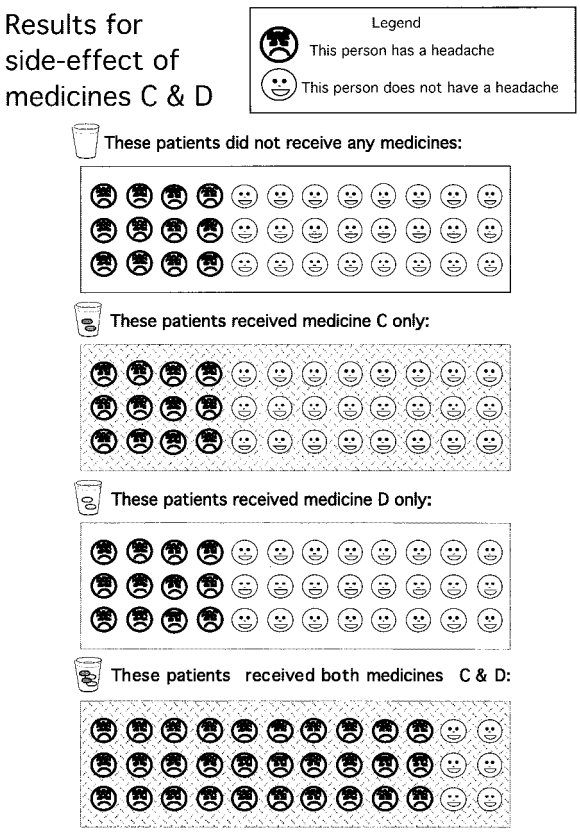

Figure 10. A black-and-white version of the stimulus materials from a condition in Clifford and Cheng's (2000) study. The figure depicts the results of a study testing the influences of Medicines C and D on headaches.

strengths of the individual medicines ("What effect does Medicine [x] alone have on headaches?"), then that of their conjunction ("What effect do Medicines [x and y] in combination have on headaches?"). Causal strength was measured in two steps: (a) a qualitative assessment of whether a candidate cause (each medicine individually or the two in combination) has any influence on *e* and (b) if the candidate was judged to have an influence, an assessment of the direction of the influence and an estimate of its extent, as indicated by the frequency of the outcome under a counterfactual intervention on entities that did not show that outcome. In particular, subjects were asked how many patients out of 100, all of whom did not show an outcome, would now have the outcome if the candidate cause were introduced. For example, when subjects indicated that a candidate caused headaches, they were asked to estimate the number of patients who would get a headache if the candidate were given to 100 patients without headaches.

The counterfactual questions removed alternative influences of *e* of like kind as the candidate (generative or preventive), thereby allowing the influence of the candidate to manifest itself without contamination. They also required subjects to generalize from the learning to the test context, thereby allowing the measurement of what subjects assumed to be invariant across contexts—either the proximal relation or the distal one.

Using frequency as the dependent measure increases the concreteness of the reasoning task and therefore would likely improve accuracy (Tversky & Kahneman, 1983). In particular, it may be less natural to conflate reliability and causal strength when the latter is indicated by a specific number of discrete entities among a set of such entities in the world, rather than by a subjective rating on a scale, as has been typical in previous psychological studies of causal induction.

To encourage a uniform interpretation of the values on the dependent measure, Clifford and Cheng (2000) exposed all subjects at the outset to three conjunctive candidate causes that should yield a wide range of answers on the dependent measure (Parducci, 1965): The first three problems illustrated a generative cause, a preventive cause, and a noncausal candidate. For one group of subjects, the first three problems were GH, ST, and OP; for a second group, they were MN, AB, and QR. The other seven problems for each group were presented in a different random order for each subject.

Results. If subjects ($N = 60$) answered that a medicine "has no effect" on headaches, their judged causal strength was scored as zero. If they answered that a medicine "causes" headaches, their numerical estimate in response to the counterfactual question was scored verbatim as a positive number; and, if they answered that a medicine "prevents" headaches, their numerical estimate was scored as a negative number. As expected, all the individual medicines were judged to be noncausal by virtually all subjects.

The results for the conjunctive candidates are summarized in Table 3. The table lists medians in addition to means, because the distributions of estimates for most of the conditions were clearly skewed. The results strongly support the predictions of our conjunctive power theory over those of the probabilistic contrast model (Cheng & Novick, 1990) and the cross-product ratio.

We consider first the comparison of the predictions of the conjunctive power PC theory and the probabilistic contrast model. With an increasing base rate of *e*, the absolute values of the average causal judgments for problems with the same level of ΔP_{ij} increased when ΔP_{ij} was positive (compare condition GH with CD and condition CD with AB) and decreased when ΔP_{ij} was negative (compare condition ST with IJ and condition IJ with MN). Moreover, when causal power and ΔP_{ij} predict differences in opposite directions (conditions AB and EF and conditions ST and KL), the average causal estimates differed in the direction predicted by causal power. These observed differences, which were all highly reliable, indicate that subjects' estimates of causal strength reflected conjunctive causal power.

In contrast, causal judgments remained at or near zero when ΔP_{ij} was zero and did not differ reliably across those two conditions (OP and QR), despite a variation in the base rate of *e*. Likewise, when candidates shared the same causal power but varied in ΔP_{ij} (generative conditions CD and EF and preventive conditions IJ and KL), the average causal estimates were nearly identical, indicating that estimates of causal strength were not a function of ΔP_{ij}.

Next, we consider how the predictions of the cross-product ratio fared. As we noted earlier, this criterion and the ΔP model differ in their predictions for only 2 of the 10 conditions. For these conditions, the observed judgments deviated even more from the predictions of the cross-product ratio than from those of the probabilistic contrast model. For example, CD was observed to

have a causal strength about as high as that for EF, as predicted by our theory, rather than equal to that for GH, as predicted by ΔP, or lower than that for GH, as predicted by the cross-product ratio. Therefore, all comparisons favoring our theory over ΔP also favor our theory over the cross-product ratio.

The complex patterns of results found by Clifford and Cheng (2000) follow from our parameter-free conjunctive power theory but contradict the ΔP model, interaction contrasts, the cross-product ratio, and all consistent parameter settings of Rescorla and Wagner's (1972) model. As discussed by Clifford and Cheng, their results also refute the weighted ΔP model (e.g., see Anderson & Sheu, 1995) and linear combination models (Schustack & Sternberg, 1981), two additional purely covariational accounts. In conclusion, these results indicate that the distal causal relation in our conjunctive power measures, rather than the proximal association in all purely covariational models, is what the reasoner assumes to be invariant across contexts.

Summary and Directions for Future Empirical Research

Tests of our conjunctive power PC theory come in a variety of forms. Our theory makes predictions about whether patterns of information presented to reasoners will lead them to perceive interactive causal influence. If such influence is perceived, our theory makes further predictions as to the direction (generative or preventive) and strength (i.e., power) of the conjunctive cause. In certain circumstances, our theory predicts that reasoners should withhold judgment as to the causal status of a conjunctive candidate—the available information is insufficient for deciding whether the candidate has causal efficacy. For some patterns of information, our theory makes the same predictions as do previous theories of interactive causal influence, and empirical evidence supports both our theory and the other theories. For other patterns of information, our theory makes unique predictions. Table 4 summarizes five types of tests of our new theory (the list is not exhaustive) and indicates the status of each of five accounts of causal discovery with respect to these tests. The results we have reported here only begin to test our theory. They sometimes come from studies that were not designed to test it and are consequently suboptimal. Moreover, alternative explanations have not been adequately tested. Nonetheless, where discriminating data exist, the results so far clearly favor our new conjunctive power PC theory over other existing accounts.

The strongest type of test involves judgments of the magnitude of conjunctive power in situations in which one or both candidate causes have nonzero simple power. Good measures of conjunctive power for such situations are challenging to construct. There is no everyday term that corresponds to a conjunctive cause. Neither is there a counterfactual expression of the concept in terms of ob-

Table 4
Hierarchy of Tests for Discriminating Among Accounts of Interactive Causal Inference, and the Status of Several Accounts With Respect to These Tests

	Account of interactive causal inference				
Type of test	Conjunctive power PC	Power PC[a]	Interaction contrast	Cross-product ratio	Rescorla–Wagner
Identify independent influence of two causes with simple (generative or preventive) power (Figures 1, 2, & 7; superimposition experiment)	SUPPORTED	SUPPORTED	contradicted	contradicted	contradicted
Identify the presence of interactive influence when one constituent of the conjunction has simple power (Cheng & Novick, 1990)	SUPPORTED (default sequential conception)	—	SUPPORTED	SUPPORTED	SUPPORTED
when both constituents of the conjunction have simple power (superimposition experiment)	SUPPORTED	—	contradicted	contradicted	contradicted
Estimate the strength of a conjunctive cause when alternative causes may have generative power but the constituents of the conjunction have no simple power (Clifford & Cheng, 2000)	SUPPORTED	SUPPORTED	contradicted	contradicted	contradicted
when alternative causes may have generative power and the constituents of the conjunction may have simple (generative or preventive) power (White, 1998, Experiment 1)	SUPPORTED	—	contradicted	contradicted	—

Note. A dash indicates that the theory is not applicable to the type of test indicated. The relevant empirical evidence for the cell entries in each row is indicated in parentheses after each type of test.
[a] This is a model of simple, rather than interactive, causal influence. It is included to show what the conjunctive power PC theory does that the simple cause theory cannot.

servable events only, as there is for simple causal power. Assuming the absence of all causes other than the conjunctive candidate will not do—the candidate does not exist except in the presence of the constituent simple causes. Better tests of our theory for such situations await further methodological insights.

The Normative Assessment of Conjunctive Causation

Psychological models of causal discovery are not the only ones that take the purely covariational approach. As we mentioned earlier, all conventional statistical measures of independence for categorical data are purely covariational in the same sense, namely that they do not explicitly incorporate the possible existence of distal causal relations into their inference procedures. In this section, we consider some inferences produced by the criterion of independence used in conventional statistics, as well as the implications of our theory for that criterion (we do not address the issues of reliability and effect size, concepts that make use of but go beyond the criterion of independence). We also consider the interpretability of the output of various measures of independence when applied to test interactive causal influence and draw some conclusions concerning the normative assessment of conjunctive causation.

Standard Statistical Measures of Independence

Being purely covariational, conventional statistics do not inherently distinguish between covariation and causation. To cope with this distinction, conventional statistics justify causal inferences only with the aid of experimental design. In particular, scientists assume that conventional statistics are overridden by the principles of experimental design: When a relevant design principle (e.g., that of control) is violated for a set of data, statistics based on that set are simply assumed to have no causal interpretation.

These principles, however, cannot salvage conventional statistics. Even when no design principle is violated, conventional statistical measures can err, as we illustrate by applying such measures to the data patterns depicted in Figures 1 and 2 (assuming that the patterns recur in sufficiently many other entities to yield reasonable sample sizes). The first problem is that the same statistical measure would be applied to both figures, because such measures, like other purely covariational ones, treat generative and preventive causal influences symmetrically, as mirror reflections of each other with respect to the effect. In particular, in conventional scientific practice, one does not apply different statistical measures of interaction depending on the direction of the main effects. Recall, however, that the product definition of independence applies to only the proportion of not-*e* entities in Figure 1, but it applies to only the proportion of *e* entities in Figure 2. It follows that the same measure, whatever it might be, cannot possibly yield independent influence for both figures.

Worse yet, conventional statistics cannot yield an intuitively correct answer for either figure. Standard statistical measures are always applied to all levels of all variables, dependent as well as independent, about which one cares to infer, as shown in all textbook examples illustrating any of these measures (e.g., Fienberg, 1980; Freedman et al., 1998; Hays, 1997). One obviously has to iterate the relevant computation over both levels of binary independent variables (otherwise there would be no manipulation). Because dependent and independent variables are not treated differently in measures for discrete variables, the same therefore goes for binary dependent variables. Thus, to obtain the expected frequency in each cell of a design, all conventional statistical measures of independence for categorical data apply the product definition of independence to both levels of binary dependent variables (e.g., both *e* and not-*e*). But, by De Morgan's Laws, the two levels cannot both correspond to conjunctions of events, because *e* and not-*e* are complementary. Negating the conjunction (intersection) "*e* fails to be produced by *i* and *e* fails to be produced by *j*," for example, would result in the disjunction (union), "*e* produced by *i* or *e* produced by *j*" (see the bottom panel of Figure 1). When applied to Figures 1 and 2, therefore, conventional statistics violates De Morgan's Law.

Not only is the practice of iterating over more than one level of a dependent variable natural for conventional categorical statistics, it is unavoidable when sample size may vary across conditions. Because such statistics operate on raw rather than relative frequencies, if the assessment of independence were based on a single level of a dependent variable, the resulting expected frequencies might reflect nothing more than differing sample sizes in the various conditions. (In fact, even when all levels of dependent variables are included, maintaining equal sample sizes across conditions is necessary to avoid confounding for purely covariational measures. In contrast, systematic bias will not result from unequal sample sizes using our theory, because our theory operates on relative rather than raw frequencies.)

The anomaly of these results, made evident by our figures, is the rule rather than the exception. It is not, for example, specific to data with an observed value of zero in a cell (e.g., for the frequency of *e* in the condition depicted in the top panel of Figure 1). Consider a simple generalization of the data pattern in Figure 1 that is created by duplicating the row of entities in each panel, exactly as it is, 10 times, and adding the influence of an independent cause that produces *e* in one of the 10 resulting rows (say, the first) in each panel: The outcome is a pattern that has 90 entities in each panel, and *e* occurs, respectively from the top to the bottom panel, in 9, 63, 36, and 72 entities. As before, the bottom panel illustrates the superimposition of the second and third panels and, therefore, the independent influences of *i* and *j* on *e*. Our conjunctive power PC theory specifies no interaction for this pattern of data, as it does for Figure 1. (Cheng's, 1997, theory of simple causes yields estimates of two thirds and one third, respectively, for q_i and q_j, as before, and an estimate of one tenth for $P(a) \cdot q_a$.)

In contrast, let us consider the cross-product ratio (Equation 26), the criterion of independence (Fienberg, 1980) adopted by all conventional statistical measures—the measures for assessing reliability as well as those for assessing effect size, including the X^2 and G^2 tests, log linear models, and logistic regression—to test for, and measure the degree of, association between binary variables (e.g., see Fienberg, 1980; Freedman et al., 1998; Hays, 1994; Wickens, 1989):

$$CPR = \frac{n_{121} \cdot n_{211} \cdot n_{112} \cdot n_{222}}{n_{111} \cdot n_{221} \cdot n_{122} \cdot n_{212}}. \tag{26}$$

Each n_{ije} in this equation is a cell frequency for the $2 \times 2 \times 2$ table formed by varying the presence versus absence of causal factors *i* and *j* and the effect, *e*, repectively. Applied to the data pattern

under consideration, the cross-product ratio, with a value of 3.5, would conclude that *i* and *j* interact (recall that a value of 1 indicates independence). In other words, the value of the measure of association for *i* and *e* at one level of *j* is 3.5 times that at the other level of *j*: The odds ratio of *e* for *i* when *j* is absent is

$$\frac{n_{121} \cdot n_{222}}{n_{221} \cdot n_{122}} = 21;$$

whereas when *j* is present, the odds ratio is

$$\frac{n_{111} \cdot n_{212}}{n_{211} \cdot n_{112}} = 6.$$

It is possible that conventional statistics simply adopt a stricter criterion of independence and that whenever our theory and compelling intuitions infer that there is interactive causal influence, so do conventional statistics. But that is not the case. Consider two examples of the converse situation, in which our theory and compelling intuitions both infer interaction, but conventional statistics conclude otherwise. Suppose that all panels in Figure 1 remain the same except the bottom one, which instead shows that if the entities had been exposed to both *i* and *j*, *e* would have occurred in all nine entities. Our theory would conclude that *i* and *j* interact in this situation (and would estimate their conjunctive power to be 1). In contrast, all conventional statistical measures (with an adjustment for an observed value of 0 in a cell; see Gart & Zweifel, 1967; Haldane, 1955), as well as, coincidentally, the interaction contrast from the probabilistic contrast model (Cheng & Novick, 1990; Rothman & Greenland, 1998), would concur that *i* and *j* now do not interact (e.g., the value of the cross-product ratio would be 1). Recall that in the superimposition experiment reported earlier, in which 40 college students were asked to choose whether the data pattern predicted by our conjunctive causal-power theory (e.g., a mere mortal's view of the bottom panel of Figure 1) or that predicted by the cross-product ratio (e.g., the revised bottom panel described here) corresponded to their conception of independent influence, only 1 student chose the pattern specified by conventional statistics.

Likewise, if the four panels each contain 100 entities, with *e* occurring, respectively, in 1, 10, 10, and 55 of them, as shown in Figure 11, the cross-product ratio (with a value of 1) would indicate no interaction. Readers may form their own opinions about whether these data indicate independent influence or interaction.

What are the causal assumptions that would render these conclusions of conventional statistics sensible? The fact that these assumptions are left implicit does not imply that they are unnecessary for interpreting data patterns concerning the presence or absence of causal candidates and of the effect. Until such assumptions are made explicit, it is difficult to evaluate whether they (a) hold in a situation, (b) are coherent, (c) are simple, or (d) are plausible.

In contrast, our theory specifies that *i* and *j* interact in Figure 11 (with $q_{ij} = .45$). Given the frequencies of *e* shown in the first three bars in the figure, our theory would predict no interaction if the frequency in the fourth bar were 18 rather than 55 (as indicated by the dashed line in that bar). More generally, it can be shown that when applied to test hypotheses involving the independent influence of two binary causes on a binary effect, unless at least one of the candidate causes by itself does not influence *e*, standard statistical measures of independence and our theory differ in their

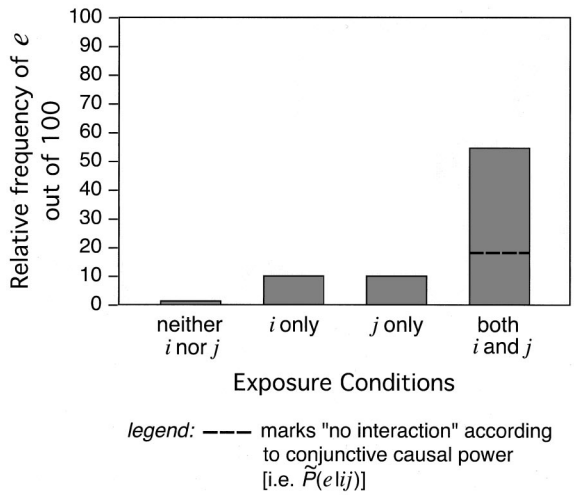

Figure 11. Example data pattern illustrating no causal interaction between candidate causes *i* and *j* on effect *e* according to the cross-product ratio. The dashed line indicates that there is no interaction according to conjunctive causal power (i.e., $\tilde{P}(e|ij)$).

predictions regarding the absence of interactive causal influence (i.e., whenever standard measures indicate that there is no interactive causal influence, our theory indicates that there is, and vice versa). If at least one of the candidate causes by itself does not influence *e*, because at most one simple candidate is causal, the superimposition of the influences of the causes becomes trivial, and the measures agree on whether there is interactive causal influence. Even when standard statistical measures of independence and our theory coincide in their predictions regarding the presence of interactive causal influence, they differ on estimates of the strength of the influence.

On the Interpretability of the Output of Various Measures of Interactive Causal Influence

We have considered some of the consequences of explicitly incorporating the possible existence of distal causal relations into the inference procedure for the discovery of such relations. The difference this approach makes can also be seen more directly by considering the interpretability of the output of purely covariational models versus that of our theory. Conjunctive causal power, like simple causal power, is a probability (on a ratio scale) that has a well-defined meaning in terms of frequencies of events in the world: Recall our illustration of the mapping of conjunctive causal power onto estimated relative frequencies of events in the world as the proportion of radiating rings (in our illustration) among the light gray ones in the bottom panel of Figure 1. The output of purely covariational models, in contrast, does not have any analogously concrete interpretation; rather, it is merely a vague ordinal rating of associative strength in a particular direction within an arbitrary range. For example, the cross-product ratio ranges from 0 to ∞, with 1 indicating independence. Consider again our earlier example in which the panels in Figure 1 each have 90 entities, of which, respectively, 9, 63, 36, and 72 show the effect. As we noted earlier, the cross-product ratio concludes that *i* and *j* interact, as the value of the ratio is 3.5. It is not clear how this

value would be mapped onto the events conveyed by that pattern of data. To what, exactly, does 3.5 refer?

In the case of probabilistic contrasts (Cheng & Novick, 1990, 1992; Rothman & Greenland, 1998), whereas ΔP_{ij} can be as large as 2 and as small as -2, simple contrasts range only from 1 to -1.[21] For example, consider a situation in which *e* occurs with certainty when either *i* or *j* is present without the other but never occurs when neither or both of these factors is present. In this case, Equation 2 yields an interaction contrast of -2. With the complementary probabilities in the four conditions, the contrast equals 2. As with the output of the cross-product ratio, it is not clear how these contrast values map onto entities in the world. It might be argued that the range of output values from these models can be adjusted by a scale transformation (e.g., taking the log of the cross-product ratio to give a range from $-\infty$ to ∞, with 0 indicating no interaction). Scaling, however, does not impart additional meaning, and the values would remain ordinal.

Conclusions

Our results imply that a normative statistical procedure for assessing interactive causal influence would vary across cases (see Figures 5 and 6). In particular, a different measure would apply depending on the direction of (a) each of the two simple causes and (b) the deviation of the observed frequency of *e* given the joint presence of the two candidates from that expected assuming that the candidates exerted only their simple powers (i.e., the sign of the conjunctive probabilistic contrast). Such a procedure has never previously been advocated and may seem complicated. The alternative, however, is to apply a single measure, as in standard statistics, that only sometimes yields interpretable values and that does not have a concrete mapping onto events in the world.

It seems to us that, with respect to the evaluation of conjunctive causal hypotheses, untutored reasoning is not less but more normative than standard statistical measures. Our results therefore stand in contrast to the genre of psychological research that looks to mainstream formal analyses for a definition of rationality and measures how untutored reasoning falls short of this standard (Kahneman, Slovic, & Tversky, 1982; Meehl, 1954).

General Conclusions

We have discussed some differences in the assessment of interactive causal influence between our causal-power approach and the purely covariational approach. We believe that the source of these differences lies in the different goals of the two approaches. Under the causal-power approach, the reasoner's goal is to infer causal power, which he or she believes to be a distal relation—one that exists in the world, despite the fact that it is not observable and may in various ways fail to be manifested in the covariations observed. In contrast, under the purely covariational approach, the reasoner's sole goal is to compute covariation—a proximal relation that exists in the reasoner's senses. That is, he or she foregoes inferring the distal relation that produces the proximal relation, ignoring its existence altogether by having no representation of it.

Different goals yield different results. The purely covariational approach yields inferences that do not license predictions about the consequences of actions. For example, despite the covariation between flattened snakes and the conjunction of morning school bells and blooming daffodils, flattened snakes will not appear on the road if one intervenes with that conjunction—say by ringing school bells on a winter morning in a green house full of blooming daffodils. To override the unwarranted recommendations for action that purely covariational statistics would have implied, 20th century science relied on the principles of experimental design. Our analysis indicates, however, that such principles are incapable of overriding a class of such recommendations: As illustrated in the previous section, even when such principles hold, anomalous conclusions can result from conventional statistics. In contrast, the causal-power approach yields inferences that (a) allow the reasoner to predict the consequences of actions, for example, that striking a dry match (a conjunctive cause) would light it and (b) are in accord with some compelling principles of experimental design, without the need to invoke them as overriding assumptions. Empirical evidence suggests that untutored reasoners adopt the causal-power approach and make causal judgments that are normative in these senses.

[21] Peter White called our attention to this problem (personal communication, August 23, 1994; see also White, 1998).

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Appendix

Conjunctive Power for Cases 3 and 5 Under the Assumption That the Simple Causes and the Conjunctive Cause Operate in Parallel

To explain the exact difference between the sequential and parallel conceptions, we expand the power explanation of $P(e|ij)$ under the two conceptions for Case 3. We first fill in the derivation for the sequential (cure) conception reported earlier. Recall that according to this conception, *e* will occur if (a) it is produced by *a* and not prevented by either *i* or *j* or (b) it is produced by the conjunction of *i* and *j*. Substituting the expression for $\tilde{P}_{--}(e|ij)$ given in its theoretical equation (see Figure 5) into the theoretical equation for $P(e|ij)$ for this case (Equation 20), and making use of the no-confounding condition, we obtain this expansion of $P(e|ij)$ according to the cure conception:

$$P(e|ij) = P(a) \cdot q_a \cdot (1 - p_i) \cdot (1 - p_j) + q_{ij} - P(a) \cdot q_a \cdot q_{ij} \cdot (1 - p_i) \cdot (1 - p_j). \quad \text{(A1)}$$

Now, consider the parallel (suppressant) conception. According to this conception, *e* will occur if (a) it is produced by *a* or by the conjunction of *i* and *j* and (b) it is not prevented by either *i* or *j*. Thus, to generate the power explanation of $P(e|ij)$, one computes the probability of the union of the events listed in Part a and then computes the probability of the intersection of the events listed in Parts a and b, which yields (again making use of the no-confounding condition):

$$\begin{aligned} P(e|ij) &= [P(a) \cdot q_a + q_{ij} - P(a) \cdot q_a \cdot q_{ij}] \cdot (1 - p_i) \cdot (1 - p_j) \\ &= P(a) \cdot q_a \cdot (1 - p_i) \cdot (1 - p_j) + q_{ij} \cdot (1 - p_i) \cdot (1 - p_j) \\ &\quad - P(a) \cdot q_a \cdot q_{ij} \cdot (1 - p_i) \cdot (1 - p_j). \end{aligned} \quad \text{(A2)}$$

The righthand sides (RHSs) of Equations A1 and A2 differ only in that there is a q_{ij} term in Equation A1 that stands alone, whereas in Equation A2 the corresponding term is multiplied by $(1 - p_i) \cdot (1 - p_j)$.

This difference leads to an alternative estimate of generative conjunctive power under the suppressant conception, namely,

$$q_{ij} = \frac{\Delta P_{--}}{(1 - p_i) \cdot (1 - p_j) - \tilde{P}_{--}(e|ij)}. \quad \text{(A3)}$$

Equation A3 differs from the corresponding theoretical equation for the cure conception (middle cell of Figure 5) in that $(1 - p_i) \cdot (1 - p_j)$ replaces 1 in the denominator of the RHS. As expected according to the suppressant conception, $(1 - p_i) \cdot (1 - p_j)$ becomes the upper bound on $P(e|ij)$, because even if the generative causes succeed in producing *e* in all entities, only a fraction of them—namely, $(1 - p_i) \cdot (1 - p_j)$—will be prevented by neither *i* nor *j*. In contrast, the upper bound on $P(e|ij)$ is 1 according to the cure conception.

An analogous derivation for Case 5, in which only one simple cause, *i*, is preventive, yields the following estimate for generative conjunctive power under the suppressant conception:

$$q_{ij} = \frac{\Delta P_{-+}}{(1 - p_i) - \tilde{P}_{-+}(e|ij)}. \quad \text{(A4)}$$

Here, $(1 - p_i)$ is the upper bound on $P(e|ij)$, rather than 1 as in the cure conception (middle equation in the bottom panel of Figure 5).

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