Psychological Review
1992, Vol. 99, No. 2, 365-382

0033-295X/92/$3.00

Covariation in Natural Causal Induction

Patricia W. Cheng
University of California, Los Angeles

Laura R. Novick
Vanderbilt University

The covariation component of everyday causal inference has been depicted, in both cognitive and social psychology as well as in philosophy, as heterogeneous and prone to biases. The models and biases discussed in these domains are analyzed with respect to focal sets: contextually determined sets of events over which covariation is computed. Moreover, these models are compared to our probabilistic contrast model, which specifies causes as first and higher order contrasts computed over events in a focal set. Contrary to the previous depiction of covariation computation, the present assessment indicates that a single normative mechanism—the computation of probabilistic contrasts—underlies this essential component of natural causal induction both in everyday and in scientific situations.

We do not perceive the visual world as a two-dimensional mosaic of bits of light patches. Instead, these data from the retina are processed by our central visual system to yield a coherent perception of the world, reflecting its visual and spatial structures. Similarly, we do not perceive our lives or the world beyond as a stream of unconnected elemental events. Here, too, central processes act on the data to yield an organized view, structured in terms of commonsensical and scientific theories. Causal induction is an example of such organizing processes. When a government resorts to violent suppression of its people or yields to peaceful reform, when a couple decides to date or a marriage breaks up, or when an epidemic strikes or a new vaccine controls it, we seek out causes.

How do ordinary people induce the causes of events? Moreover, given that the primary goals of causal induction are the recovery of the causal structure of the world and the prediction of future events, is the mechanism underlying natural causal induction adequate for satisfying these goals? Covariation—the change in the probability of an effect given the presence versus the absence of a potential cause—has generally been regarded as a necessary (although insufficient) criterion of normative causal induction.[1] The computation of covariation has generated a considerable body of research in the cognitive and social literatures, both of which have presented rather messy pictures of the psychological mechanism. These literatures suggest that the covariation component in natural causal induction is nonnormative in many ways. Deviation from normative covariation has also received considerable attention in philosophy. In this article, we evaluate the biases and models discussed in these three domains with respect to focal sets: contextually selected sets of events over which covariation is computed. Moreover, we compare these models to our *probabilistic contrast model* (Cheng & Novick, 1990a, 1990b, 1991).

Deviations From Normative Covariation

Linear-Combination Heuristics

Cognitive psychologists have described a variety of nonnormative heuristics based on linear combinations of the frequencies of the four cells of a 2 × 2 contingency table formed by crossing the presence and absence of a potential cause with the presence and absence of a target effect (e.g., Arkes & Harkness, 1983; Downing, Sternberg, & Ross, 1985; Einhorn & Hogarth, 1986; Jenkins & Ward, 1965; Schustack & Sternberg, 1981; Shaklee, 1983). Arkes and Harkness (1983) reported that their subjects used a variety of heuristics depending on task characteristics. They concluded that "a search for *the* heuristic that people use will be a futile search" (p. 132). Similarly, Shaklee and Tucker (1980, p. 466) concluded that "the variety of rules evident in our results indicates that characterization of group judgment by any single rule would be inappropriate." Other researchers concluded that normal people untrained in statistics typically do not have any concept corresponding to statistical contingency or contrast (Jenkins & Ward, 1965; Smedslund, 1963; Ward & Jenkins, 1965), the putatively normative basis of

The preparation of this article was supported by National Science Foundation Research Grant BNS87-10305 and by the Committee on Research of the Academic Senate of the Los Angeles Division of the University of California on behalf of Patricia W. Cheng. It was also supported by a National Institute of Mental Health Individual Postdoctoral Fellowship and by the dean's office of Peabody College of Vanderbilt University on behalf of Laura R. Novick.

We thank Angela Fratianne, Rochel Gelman, Keith Holyoak, Richard Nisbett, Michael Waldmann, Bernard Weiner, and three anonymous reviewers for their valuable comments on an earlier draft.

Correspondence concerning this article should be addressed to Patricia W. Cheng, Department of Psychology, Franz Hall, University of California, Los Angeles, California 90024-1563.

[1] Even when adaptive learning apparently occurs on the basis of a single trial, covariation of the cause and the effect exists. For example, assuming that rats learn to avoid a new-tasting food after a single ingestion of that food is followed by gastrointestinal illness, the probability of the effect (gastrointestinal illness) in the presence of the potential causal factor (the new-tasting food) is 1, whereas the probability of the effect in its absence (based on prior experience) is presumably substantially less than 1. There is, therefore, a positive covariation between the events.

the concept of covariation (e.g., see Alloy & Abramson, 1979; Jenkins & Ward, 1965; Rescorla, 1968; Salmon, 1984; Skyrms, 1986).

Kelley's Analysis of Variance Model

The causal attribution literature in social psychology has largely measured performance against an apparently different normative standard: namely, variations of Kelley's (1967, 1973) influential proposal that people are "intuitive scientists" who use a mechanism of causal induction analogous to the analysis of variance (ANOVA) (Cheng & Novick, 1990a; Försterling, 1989; Hewstone & Jaspars, 1987; Hilton, 1988, 1990; Jaspars, 1983; Orvis, Cunningham, & Kelley, 1975; Pruitt & Insko, 1980). This literature has, until recently, presented a chaotic picture of causal induction not unlike that in cognitive psychology: Causal induction sometimes conforms to the normative standard but often deviates from it. Such deviations include a bias against using consensus information (which has been regarded as a tendency to ignore base-rate information), a bias toward attributing effects to a person, a tendency for actors and observers to make different causal attributions for the same event, and a tendency to make a variety of unpredicted attributions to conjunctions of factors (for reviews, see Cheng & Novick, 1990a; Jaspars, Hewstone, & Fincham, 1983; Kelley & Michela, 1980; Nisbett & Ross, 1980).

Causes, Enabling Conditions, and Causally Irrelevant Factors

A deviation from normative covariation that has received considerable attention in philosophy and related fields concerns the intuitive distinction people make between causes and enabling conditions (e.g., Einhorn & Hogarth, 1986; Hart & Honoré, 1959/1985; Hesslow, 1983, 1988; Hilton, 1990; Kahneman & Miller, 1986; Mackie, 1965, 1974; Mill, 1843/1973; Taylor, 1983; White, 1965). In response to the question "What caused the airplane to crash?" investigators are unlikely to reply, "The gravitational pull of the earth." Rather, they are likely to reserve the title of "cause" for factors such as the malfunctioning of a critical component of the aircraft, pilot error, or wind shear. Gravity, they might say, was merely a condition that enabled the crash to occur. In contrast to all of these factors, which an investigator would (or could) perceive as causally relevant, the color of the airplane seats or the number of infants on board, for example, would be perceived as causally irrelevant.

It has long been recognized that the distinction between causes and enabling conditions cannot be explained by accounts of inference formulated purely in terms of necessary and sufficient conditions. Mill (1843/1973, p. 329), for example, thought that everyday explanation diverged from scientific explanation in the "capricious manner in which we select from among the conditions which we choose to denominate the cause." Consider a particular airplane crash for which the malfunctioning of the airplane's guidance system and gravity were necessary factors. These two factors hold the same logical relationship to the effect in terms of necessity and sufficiency: The crash would not have occurred either if the component had not malfunctioned or if there had been no gravity; moreover, the malfunctioning of the component and gravity, along with other necessary factors such as the failure of a backup system, were jointly sufficient to have produced that crash.[2]

To compound the puzzlement, the perception of what is a cause or an enabling condition may vary depending on context. For example, Hart and Honoré (1959/1985, p. 35) noted that the presence of oxygen typically would be considered an enabling condition rather than the cause of a fire; the cause might be an event such as the dropping of a lighted cigarette. However, "if a fire breaks out in a laboratory or in a factory, where special precautions are taken to exclude oxygen during part of an experiment or manufacturing process . . . there would be no absurdity at all in *such* a case in saying that the presence of oxygen was the cause of the fire."

To make sense of the distinction between causes and enabling conditions, a number of theorists argued that a causal question invariably implies comparisons among a selected set of events (Einhorn & Hogarth, 1986; Hart & Honoré, 1959/1985; Hastie, 1983; Hesslow, 1983, 1988; Hilton, 1990; Kahneman & Miller, 1986; Mackie, 1965, 1974; McGill, 1989). McGill (1989, p. 189), for example, hypothesized that "individuals structure the to-be-explained event as the difference or deviation between a target episode and a contrasting causal background." On this view, a question such as "What caused the forest to be on fire?" can be understood as, "What made the difference between this occasion in the forest on which there was a fire and other occasions in the forest on which there was no fire?" Note that the selected set of events is often only a subset of the events related to an effect. The expanded question, for example, does not include all events in one's knowledge base that are related to fires: It does not include events in which oxygen is absent, for instance, even though such events (at least in an abstract form) are in a typical educated adult's knowledge base. On this view, shifts in the perception of an event as a cause or an enabling condition are due to the adoption of different contrasting causal backgrounds (i.e., focal sets in our terminology).

Overview

Our probabilistic contrast model may be regarded as a modification of Kelley's (1967) analogy between causal induction and the ANOVA or as an extension of the contingency rule discussed in the cognitive psychology, animal conditioning, and philosophy literatures (e.g., Jenkins & Ward, 1965; Rescorla, 1968; Salmon, 1984). Extending these models by anal-

[2] For types of effects rather than particular instances of an effect, the logical relationship between a factor and the effect is more complicated, because there are often alternative ways of producing a type of effect (e.g., there are multiple ways of producing forest fires). Developing Mill's ideas, the philosopher Mackie (1965, 1974) proposed that an individual condition (e.g., lightning) is an insufficient but necessary part of an unnecessary but sufficient (INUS) conjunctive set of factors (e.g., lightning, the presence of combustible material, and the presence of oxygen) making up a cause of a type of effect (e.g., forest fire). Although more complicated in the case of types rather than instances of effects, the logical relation between an individual condition and the effect remains equivalent for all conditions in the set.

ogy to statistical contrasts, our model proposes that everyday causal inference is based on contrasts (i.e., differences or differences between differences) between the probability of the effect conditional on the presence versus the absence of (single or multiple) potential causal factors. These contrasts are computed for selected factors in a focal set.

The question we address is this: Considering a focal set for selected discrete variables describing the event to be explained, what do ordinary people compute to induce the causes of an event? In this article, we first review our model and its explanation of the distinction among causes, enabling conditions, and causally irrelevant factors (Cheng & Novick, 1991). In this section, we also derive Kelley's (1971) *discounting* principle as a corollary of our model. Second, we review our explanation of the myriad well-documented deviations from Kelley's ANOVA model. To support our explanations, we (a) review two experiments that test our reinterpretations of many of the biases reported in the social psychology literature (Cheng & Novick, 1990a; Novick, Fratianne, & Cheng, 1991) and (b) extend our reinterpretation to reported biases based on different paradigms in that literature. We end the first two parts with a discussion of the theoretical implications of the constraints imposed by a consideration of phenomena observed in philosophy and social psychology for previous theories in these domains. Third, we present a theoretical refutation, in view of such constraints, of all alternative current models in cognitive psychology and philosophy. We discuss (a) the linear combination heuristics proposed by cognitive psychologists, (b) Suppes's model (1970, 1984), and (c) a probabilistic extension of Mill's (1843/1973) method of difference. In addition, we review our analysis (Cheng & Novick, 1991) of the normality criterion, a dominant criterion according to which the distinction between causes and enabling conditions is based on the prevalence of potential causes (e.g., Einhorn & Hogarth, 1986; Hart & Honoré, 1959/1985; Hilton & Slugoski, 1986; Kahneman & Miller, 1986; Mackie, 1965, 1974; Turnbull & Slugoski, 1988). We specify how this criterion and formulations of causality in terms of necessity and sufficiency may be regarded as special cases of our model. Finally, we review and extend the interpretation of an earlier experiment (Cheng & Novick, 1991, Experiment 2) in support of our model against competing models.

The covariation component of everyday causal inference has been depicted as heterogeneous and prone to either systematic or capricious biases. To account for these biases, numerous models and heuristics have been proposed. In contrast with the previous depiction, our assessments converge on a single normative mechanism that underlies this essential component of causal induction.

Scope of the Article

Although covariation is a necessary criterion for causal induction, it is not a sufficient one. There clearly are innate and acquired constraints on the selection of potential causal factors with respect to a given effect. On theoretical grounds, the problem of combinatorial explosion in covariation computation surely requires that there be some innate biases in the inductive process. Empirically, it is clear that animals have such innate biases (Garcia, McGowan, Ervin, & Koelling, 1968; Garcia, McGowan, & Green, 1972). Other biases may be acquired through learning (e.g., Bullock, Gelman, & Baillargeon, 1982; Mendelson & Shultz, 1976).

To reduce the number of variables for which covariation is computed, one plausible criterion is that covariation is evaluated only for factors that are psychologically prior to the target effect. Psychological priority may be established by manipulation, potential manipulation, or perceived temporal priority. Adding this criterion, however, fails to eradicate the following problem: Whereas one normatively defined covariational relation may be designated as causal (e.g., that between touching a red-hot poker and burning one's hand), another may not (e.g., that between a drop in the barometric reading and a subsequent storm). A possible extension of the covariation view to deal with this problem of differentiating between genuine and spurious causes (covariational relations that are causal and noncausal, respectively, following Suppes's, 1970, 1984, terminology) is to adopt a criterion of conditional independence (e.g., Reichenbach, 1956; Salmon, 1980, 1984; Suppes, 1970, 1984). In terms of our model, the adoption of such a criterion involves computing contrasts separately for focal sets that are restricted to events in which a psychologically prior covariational factor (e.g., a drop in atmospheric pressure for the storm example—a factor that is psychologically prior to a drop in the barometric reading) is (a) present and (b) absent. If the contrast for a factor does not noticeably differ from zero in both focal sets, the factor is a spurious cause. Our model adopts the psychological priority criterion and allows the use of the conditional-independence criterion (Cheng & Novick, 1990a, 1991). An alternative potential solution is to assume that some understanding of an underlying causal mechanism is necessary (e.g., Bullock et al., 1982; Salmon, 1984; Shultz, 1982).

A full discussion of the problem of differentiating between genuine and spurious causes would go far beyond the scope of this article. We therefore remove from our discussion normatively defined covariations that are judged to be noncausal. Inference regarding such factors is not accounted for by any of the covariational rules discussed here.

Probabilistic Contrast Model

In our model, causal inferences to explain a target event are determined by contrasts computed over events in a focal set for selected discrete variables describing the target event.

Main-Effect Contrasts

A main-effect contrast, Δp_i, which specifies a cause involving a single factor i, is defined by the contrast (i.e., contingency) rule described earlier:

$$\Delta p_i = p_i - p_{\bar{i}}, \tag{1}$$

where p_i is the proportion of events for which the effect occurs when factor i is present and $p_{\bar{i}}$ is the proportion of events for which the effect occurs when factor i is absent. (A bar above a letter denotes the absence of the represented factor.) If Δp_i is noticeably different from zero (by some empirically deter-

mined criterion), factor i is a cause.[3] Otherwise, i is causally irrelevant. A positive contrast specifies a facilitatory cause; a negative contrast specifies an inhibitory cause (also see Kelley, 1973, on this distinction).

Because a contrast cannot be computed for a factor that is constantly present in a focal set (due to division by zero in the computation of the probability of the effect in the absence of the factor), the causal status of such a factor cannot be determined by events in the focal set; instead, its status is determined by events in other focal sets. Such a factor is (a) an enabling condition if its contrast value is noticeably different from zero (i.e., it covaries with the effect) in another focal set, but (b) causally irrelevant if its contrast value is not noticeably different from zero in other focal sets (Cheng & Novick, 1991).

To illustrate our model with the forest fire example, assume that lightning struck the forest where the fire started immediately before it started. Applying our model to the focal set, we see that the proportion of cases for which fire occurs in the presence of lightning is greater than the proportion of cases for which fire occurs in the absence of lightning. Lightning is therefore a cause. (Notice that our model does *not* require that fire always occur in the presence of lightning to covary with it.) In contrast, the corresponding difference in proportions cannot be computed for oxygen, because oxygen is constantly present in every event in the set. Oxygen is therefore merely an enabling condition. It is not causally irrelevant because people (at least those educated in chemistry) do have a focal set for which oxygen does covary with fire. Finally, the presence of stones in the forest, which does not covary with forest fire in any focal set, is considered causally irrelevant.

Interaction Contrasts

A cause may involve not just a single factor but a conjunction of factors (e.g., the simultaneous presence of positively charged clouds and negatively charged clouds as the cause of thunder; the combination of talent, hard work, and opportunity as the cause of success). In our model, an interaction contrast specifies a cause involving a conjunction of factors. Whereas a main-effect contrast specifies a difference between the proportions of events in which the effect occurs in the presence of a factor and in the absence of it, a two-way interaction contrast specifies a difference between such differences for levels of an orthogonal factor (i.e., a second-order difference; Cheng & Novick, 1990a). A two-way interaction contrast, Δp_{ij}, involving potential causal factors i and j, is defined as follows:

$$\Delta p_{ij} = (p_{ij} - p_{\bar{i}j}) - (p_{i\bar{j}} - p_{\bar{i}\bar{j}}), \quad (2)$$

where p, as before, denotes the proportion of cases in which the effect occurs when a potential contributing factor is either present or absent, as denoted by its subscripts. More generally, interaction contrasts involving n factors are defined as nth-order differences, where n is any positive integer.[4] Like main-effect contrasts, interaction contrasts can be facilitatory or inhibitory, depending on whether they are positive or negative. Our model distinguishes multiple alternative causes (corresponding to multiple main-effect and/or interaction contrasts) from a conjunctive cause (corresponding to a contrast involving multiple factors, i.e., an interaction contrast).[5]

Illustration of Contrasts Computed Over Various Focal Sets

Figure 1 illustrates contrasts specifying causes, enabling conditions, and causally irrelevant factors. It also illustrates the effect of varying focal sets. The figure is assumed to represent the entire set of events that are relevant to a particular effect in a hypothetical person's knowledge base (labeled *universal set* in the figure).

As can be seen in the figure, with respect to the universal set of events, factors q and r are individually necessary (i.e., $p_{\bar{q}} = p_{\bar{r}} = 0$) and jointly sufficient (i.e., $p_q < 1$, $p_r < 1$, but $p_{qr} = 1$) for the occurrence of the effect. According to our model, q and r are the factors in the two-way interaction contrast, Δp_{qr}, which has a value of 1 ($p_{qr} = 1$, $p_{\bar{q}r} = p_{q\bar{r}} = p_{\bar{q}\bar{r}} = 0$). Previous formulations of causality in terms of necessity and sufficiency may be regarded as special extreme cases of our model in which (a) the focal set is the universal set and (b) the proportions are expressed in relation to 0 (i.e., equal to or greater than 0) or 1 (i.e., equal to or less than 1; see preceding example) rather than as proportions.[6]

Now consider Focal Set A. In this set, $p_r = 1$ (i.e., r is sufficient for the occurrence of the effect in that context), and $p_{\bar{r}} = 0$ (i.e., r is necessary for the effect in that context). Because p_r is greater

[3] The proportions are estimates of the corresponding conditional probabilities. We assume that the magnitude of the criterion should reflect the role of sample size in people's interpretations of random sampling fluctuations, but we leave the elucidation of the exact role of sample size in causal induction to future research (see Nisbett, Krantz, Jepson, & Kunda, 1983).

[4] People will no doubt have greater difficulty with interaction contrasts involving greater complexity, and at some maximum level of complexity computation presumably will become impossible. However, because our model is a computational model (in Marr's, 1982, sense of the term) that specifies what is computed, rather than a process model that specifies how the computation is carried out, it leaves the issue of such limitations to a general model of processing limitations. It seems reasonable to expect that a model of processing limitations should apply across many different types of tasks rather than being specific to inference tasks. Although our model does not specify the algorithm whereby contrasts are computed, abundant evidence shows that people and other animals are indeed sensitive to probabilities and changes in probabilities (Estes, 1964; Gallistel, 1990).

[5] Our model applies to dichotomous events but potentially can be generalized to continuous effects (a model in terms of contrasts between means) or continuous causes as well as effects (a model in terms of regressions of the effect on potential causes).

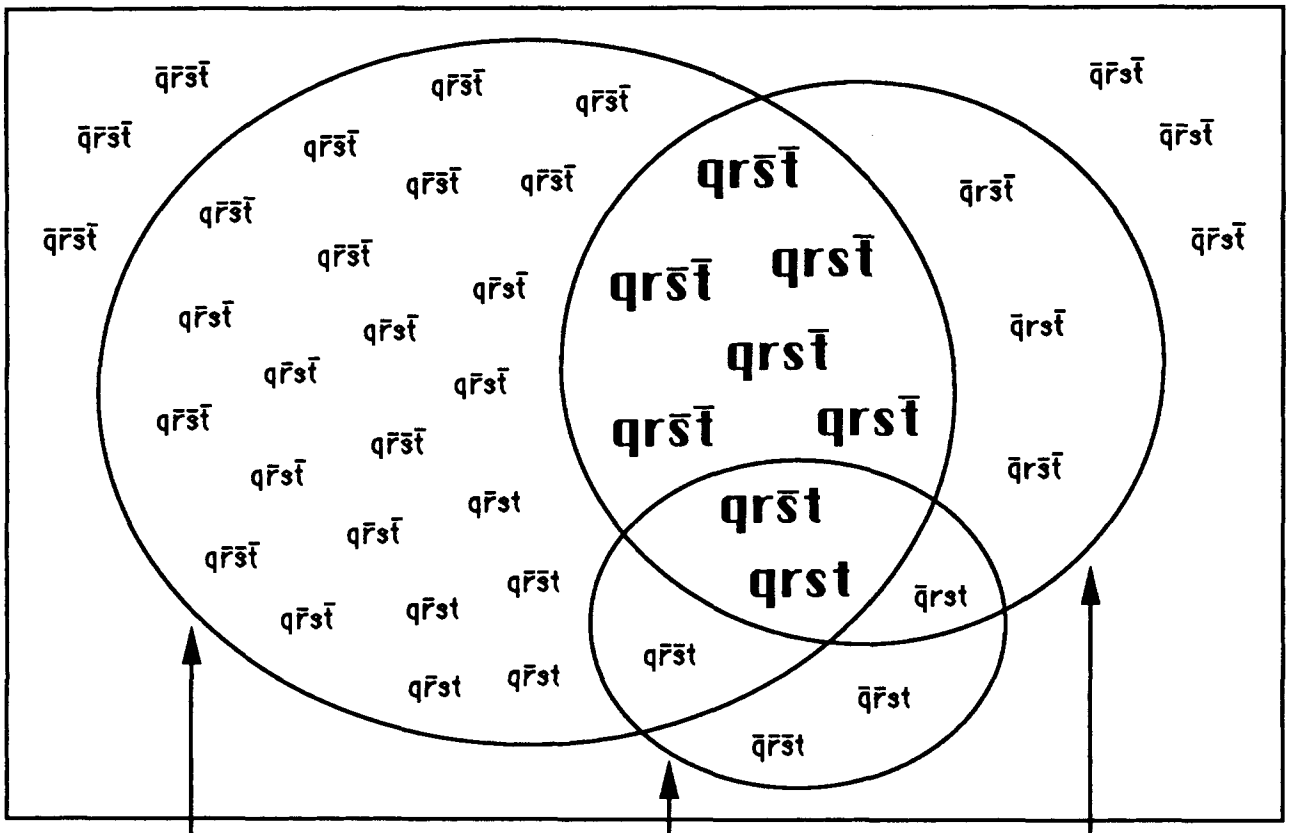
[6] According to our model, a cause consisting of INUS conditions (Mackie, 1965, 1974; see footnote 2) corresponds to an interaction contrast (among other sufficient contrasts, including other INUS conditions), for which the effect occurs (a) with probability of 1 in the universal set when all of its contributing factors are present and (b) with probability of 0 in a focal set in which no other sufficient cause is present when one (or more) of the contributing factors of the INUS conditions is absent. Suppose that a two-way interaction contrast, Δp_{qr}, is such a contrast. It follows that (a) $p_{qr} = 1$ in the universal set (i.e., the conjunction of q and r is sufficient for the effect), (b) $p_{\bar{q}r} > 0$, $p_{q\bar{r}} > 0$, or $p_{\bar{q}\bar{r}} > 0$ in the universal set (i.e., the conjunction is unnecessary for the effect), and (c) $p_{\bar{q}\bar{r}} < 1$ and $p_{\bar{q}r} < 1$ in the focal set in which no other sufficient cause is present (i.e., q and r are insufficient under those circumstances), but (d) $p_{\bar{q}} = 0$ and $p_{\bar{r}} = 0$ in that focal set (i.e., q and r are necessary under those circumstances).

UNIVERSAL SET

$\Delta p_q = p_q - p_{\bar{q}} = 8/32 - 0/12 = .25$ $\quad$ $\Delta p_s = p_s - p_{\bar{s}} = 4/22 - 4/22 = 0$

$\Delta p_r = p_r - p_{\bar{r}} = 8/12 - 0/32 = .67$ $\quad$ $\Delta p_t = p_t - p_{\bar{t}} = 2/11 - 6/33 = 0$

$\Delta p_{qr} = (p_{qr} - p_{\bar{q}r}) - (p_{q\bar{r}} - p_{\bar{q}\bar{r}}) = (8/8 - 0/4) - (0/24 - 0/8) = 1 - 0 = 1$



FOCAL SET A

$\Delta p_q = ?\ (p_{\bar{q}} = ?)$

$\Delta p_r = 8/8 - 0/24 = 1$

$\Delta p_s = 4/16 - 4/16 = 0$

$\Delta p_t = 2/8 - 6/24 = 0$

FOCAL SET B

$\Delta p_q = 2/3 - 0/3 = .67$

$\Delta p_r = 2/3 - 0/3 = .67$

$\Delta p_s = 1/3 - 1/3 = 0$

$\Delta p_t = ?\ (p_{\bar{t}} = ?)$

FOCAL SET C

$\Delta p_q = 8/8 - 0/4 = 1$

$\Delta p_r = ?\ (p_{\bar{r}} = ?)$

$\Delta p_s = 4/6 - 4/6 = 0$

$\Delta p_t = 2/3 - 6/9 = 0$

Figure 1. Computation of covariation within focal sets according to the probabilistic contrast model as an explanation of the distinction among causes, enabling conditions, and causally irrelevant factors. (Each letter [e.g., q] represents a potential causal factor. A bar above a letter [e.g., $\bar{q}$] denotes the absence of that factor. An event is represented by a sequence of letters (e.g., $\bar{q}rst$) denoting the conjunct of those factors in the event. The presence of the effect for an event is represented by larger bold type. The absence of the effect for an event is represented by regular, nonbold letters. Finally, loops and the rectangle enclose events in focal sets.)

than p_r (i.e., $\Delta p_r > 0$), r should be perceived as a cause of the effect for this subset. Factor q, however, is constantly present. A difference in probabilities conditional on the presence versus the absence of q, therefore, cannot be computed for this subset. Thus, q should not be selected as a cause. However, it is an enabling condition, because it does covary with the effect in another focal set: Set C (and to a lesser extent in Set B as well). Notice that q and r, which have the same status in terms of necessity and sufficiency with respect to the universal set, differ in their status with respect to Focal Set A. Factor q, unlike r, is insufficient ($p_q < 1$), and its necessity is undetermined ($p_{\bar{q}}$ is undefined) in that context. Designating r as a cause and q as an enabling condition, therefore, does not conflict with the logical status of these factors within this focal set.

Our model explains the effects of shifting contexts on the distinction between causes and enabling conditions by the selection of different focal sets over which covariation is computed. Consider q and r in Set C. Unlike in Set A, q covaries with the effect, but r is constantly present. Only q, therefore, should be perceived as the cause of the effect for that set. In sum, both q and r covary with the effect in some focal set. These factors should be perceived as (a) causes when the focal set of events selected for a particular context is one in which the factors covary with the effect and (b) enabling conditions when the selected focal set is not one in which the factors covary with the effect. Varying the relevant focal set thus alters which factor should be considered a cause and which should be considered an enabling condition.

Focal Sets A and C illustrate the extreme case of our model in which a cause is necessary and sufficient for the effect. Focal Set B illustrates probabilistic covariations. In this set, both q and r would be considered causes because both yield substantial positive contrasts. The contrast values for these factors are different from those in Sets A and C as a result of the different frequencies with which other necessary factors are present in the two focal sets. In contrast, t is causally irrelevant: It is constantly present in Set B, and it does not covary with the effect in either of the other sets.

Finally, notice that although s is sometimes present and sometimes absent in each of the three focal sets, its presence or absence does not covary with the effect in any focal set. Therefore, this factor, like t, is causally irrelevant to the effect. In sum, computing covariation over different focal sets accounts for the distinctions among causes, enabling conditions, and causally irrelevant factors.

Multiple Independent Causes

We have only considered the situation in which there is a single cause of effect E within a focal set. Now consider the situation in which there are multiple independent causes of E. Let C be a potential cause in the presence of which E occurs with probability $P(E|C)$. When the other causes often produce E, then $P(E|\bar{C})$ will be relatively large. Accordingly, Δp_C will be relatively small. Conversely, when the other causes rarely produce E, Δp_C will be relatively large. That is, Δp_C is an inverse function of how often the other causes produce E.

This prediction may be considered an extension of Kelley's (1971) discounting principle, which is a corollary of Equation 1 if the independence of causes is assumed. According to this principle, "the role of a given cause in producing a given effect is discounted if other plausible causes are also present" (Kelley, 1971, p. 8).[7] Evidence supporting this principle has been reported in numerous experiments (e.g., Jones, Davis, & Gergen, 1961; Lepper, Greene, & Nisbett, 1973; Thibaut & Riecken, 1955). (See the Appendix for a proof of this prediction from Equation 1.)

Deviations From Kelley's Model

Our model may be regarded as a modification of Kelley's (1967, 1973) ANOVA model. Because myriad deviations from his model have been reported in the social psychology literature, the question of how these deviations are to be reconciled with our model no doubt arises. We argue that these deviations, rather than representing irrational biases in the inductive process per se, could be due to discrepancies between the set of events specified by the researcher and the subject's focal set.

Interpretation of Biases Based on Experiments Specifying Configurational Information

Many experiments testing Kelley's (1967, 1973) model specified the stimulus input in terms of the variables of consensus (the amount of agreement between the target person and other people in their responses to the target stimulus on the target occasion), distinctiveness (the amount of disagreement between the target person's response to the target stimulus and his or her responses to other stimuli on the target occasion), and consistency (the amount of agreement between the target person's response to the target stimulus on the target occasion and his or her responses to that stimulus on other occasions). The subject's task is to explain what caused a target person to have a certain reaction to a target stimulus on a target occasion. These three information variables measure covariation along the three dimensions of persons, stimuli, and time, which Kelley (1967, 1973) proposed as independent variables in his ANOVA analogy and which he illustrated in his cube.[8] Following Cheng

[7] The discounting principle concerns the situation in which multiple alternative causal factors are present in the event to be explained. In such situations, the criterion of conditional independence requires computing contrasts for a focal set that is restricted to events in which prior known covariational factors are held constant. If the criterion is applied in these situations, the following refinement of our earlier definition of an enabling condition becomes necessary. Let i be a factor that is constantly present in the current focal set. Factor i is merely an enabling condition for a cause j in that focal set if i covaries with the effect in another focal set, and j no longer covaries with the effect in a focal set in which i is constantly absent. In contrast, i is an alternative to cause j if i covaries with the effect in another focal set, and there exists a focal set in which i is constantly absent, but j continues to covary with the effect in this set. Note that whereas an enabling condition is, by our definition, constant within the current focal set, an alternative cause can be either constant or not.

[8] Materials in these experiments have sometimes been described as providing "prepackaged" covariational information (e.g., Alloy & Tabachnik, 1984; Crocker, 1981). We note that, with respect to our model, these materials—although in summary form—do not give prepackaged covariational information, even for the relatively simple case

and Novick (1990a, 1990b), we use the term *configuration* to denote the pattern of information specified by consensus, distinctiveness, and consistency. For example, one configuration specifies high consensus, low distinctiveness, and low consistency (HLL).

As noted by several investigators (Cheng & Novick, 1990b; Försterling, 1989; Hilton, 1988, 1990; Jaspars et al., 1983; Pruitt & Insko, 1980), information on these variables, often assumed to represent all the data relevant to making causal attributions, actually covers only a subset of the potentially relevant information: specifically, one row, column, and beam of the cube. Previous researchers apparently often made the plausible but erroneous assumption that nonconfigurational information, which includes information on how other people react to other stimuli on other occasions, is *irrelevant* for explaining why a particular person has a certain reaction to a particular stimulus on a particular occasion. (An analogous assumption was made by learning theorists before Rescorla, 1968, demonstrated that the frequency of association between the unconditioned stimulus and the *absence* of the conditioned stimulus is critically important in conditioning.) However, recent evidence in the causal attribution literature indicates that subjects do make use of their assumptions regarding the occurrence of the effect in the nonconfigurational part of the cube. For example, Hilton and Slugoski (1986) insightfully demonstrated that causal attributions were influenced by people's implicit knowledge of norms (i.e., "presuppositions about what a class of persons generally does to a class of stimuli"; Hilton, Smith, & Alicke, 1988, p. 531). Such knowledge homogeneously fills the nonconfigurational (i.e., unspecified) part of Kelley's cube.

We (Cheng & Novick, 1990a, 1990b) hypothesized that because causal attribution is a joint function of the data on which the inference rules operate and the rules themselves, the apparent biases found in previous experiments—rather than being due to the inferential process—may reflect the subjects' assumptions regarding the pattern of information for the unspecified part of the cube. In experiments specifying only configurational information, it is typically not known what assumptions subjects might have spontaneously made regarding the occurrence of the effect in the remainder of the cube. If some subjects do use information in the entire cube as the basis for causal induction, then when this focal set is identified, causal attributions may reveal an unbiased assessment of covariation.

Our hypothesis is supported by several studies in which nonconfigurational information was manipulated or assessed independently of configurational information (Cheng & Novick, 1990a; Hilton & Slugoski, 1986; Novick et al., 1991; Pruitt & Insko, 1980). Here we briefly describe the results of two of our experiments. In one experiment, we explicitly varied the pattern of information in the nonconfigurational part of the cube while keeping configurational information constant (Cheng & Novick, 1990a). Thus, we provided subjects with information that completely filled Kelley's (1967) cube. In a second experiment (Novick et al., 1991), we gave subjects only configurational information and assessed both assumptions concerning the unspecified cells of the cube and causal attributions.

Specifying complete information. With the complete information problems used in the first experiment, we tested our hypothesis in two ways. First, across problems, we counterbalanced presence or absence of the effect over the three dimensions of the cube so that any bias toward a dimension (or an information variable) could not be attributed to asymmetries in the input. Second, for each of four configurations, we constructed a set of problems that shared that configuration but differed in the pattern of information over the nonconfigurational part of the cube. The patterns of nonconfigurational information were chosen such that for some of the problems our model predicts main-effect and interaction attributions that are not predicted by any previous models, including attributions that previously have been reported as biases (e.g., attributions to person, stimulus, or the conjunction of person and stimulus for the high-consensus, low-distinctiveness, high-consistency (HLH) configuration; attributions to the conjunction of person and occasion and to the conjunction of stimulus and occasion for the HLL configuration). Obtaining such attributions would demonstrate that these apparent biases can, in fact, be explained by a normative covariational model. Furthermore, the patterns of nonconfigurational information were constructed such that, over our entire set of problems, our model predicts all possible types of main-effect and interaction attributions that are unpredicted by previous models.

Our results, reported in detail in Cheng and Novick (1990a), showed that the predicted attributions were obtained. For each of the four configurations tested, the various problems sharing a configuration differed reliably in the causal attributions they elicited, as predicted by our model. In particular, for every configuration, the problem for which a particular pattern of responses was predicted by our model showed a reliably higher percentage of such responses than did problems with the same configuration for which those responses were not predicted. Moreover, our counterbalanced set of problems showed no evidence for either a bias toward making a person attribution (or any other attribution) or a tendency to ignore consensus information (or any other type of information) when such information was redefined in terms of probabilistic contrasts to capture covariation over the entire cube rather than over the configuration only.

Besides addressing the issue of bias, our experiment also allowed a comparison between our model and previous models in the social literature. All previous models (Jaspars et al., 1983; Kelley, 1967, 1973; Orvis et al., 1975), with the exception of those of Försterling (1989) and Hilton and Slugoski (1986), made predictions that were based on configurations. One interpretation of all configuration-based models is that they predict the same attributions for all of our problems that shared a configuration. In contrast, our model predicts different attributions for each of these problems. It is possible to separate princi-

in which the focal set consists solely of events in the configuration. Consider computing a main-effect contrast for a target person for this focal set. Computing this contrast involves taking the difference between the proportion of times the effect occurs in the presence and in the absence of that person. In turn, computing the former proportion involves integrating information across the target event, distinctiveness information, and consistency information and computing the latter proportion involves consensus information. This partitioning of the given information is not prepackaged and neither is the integration nor the subtraction.

ples underlying configuration-based models from the literal predictions previously made. An alternative set of predictions for those models may be derived by applying the underlying principles to information over the entire cube. Even under this approach, none of the previous models (configuration based or otherwise) can account for our data. For at least three of the four configurations we tested, all of these models predict either no causal attribution possible or the same attribution for some if not all of the problems sharing a configuration. Our results show that for every configuration tested, every problem sharing the configuration elicited a reliably different pattern of response, as predicted by our model but not by any previous model.

Assessing assumptions about the unspecified part of the cube. Just as we were able to vary the information given for the nonconfigurational part of the cube, subjects in previous experiments also might have varied their assumptions regarding that region from problem to problem or from one person to another for a given problem, thus producing what appeared to be capricious biases. We tested this hypothesis more directly in our second experiment by giving subjects configurational information only and assessing both their assumptions and causal attributions (Novick et al., 1991). For each of two configurations, we constructed scenarios in two content domains (e.g., expertise at dancing vs. music appreciation) that we expected would lead to quite different assumptions concerning the occurrence of the effect in the unspecified cells of the cube because of people's world knowledge about these domains. The resulting patterns of information in the cube (configuration plus subjects' assumptions) were predicted to lead to different causal attributions as determined by probabilistic contrasts computed for the two patterns for each configuration. Both our expectations concerning the dominant assumptions for each scenario and our predictions for the resulting causal attributions were confirmed. Furthermore, our assessment of individual subjects' assumptions allowed us to predict individual differences in subjects' causal attributions. The various causal attributions of a large majority of the subjects were consistent with the probabilistic contrasts computed over the focal sets consisting of the configuration plus subjects' individual patterns of assumptions in the nonconfigurational part of the cube. Thus, what may appear as capricious biases that differ from subject to subject in fact follow from a normative model.

In sum, our results with both complete-information and configurational-information problems indicate that people compute covariation over events in a focal set, which often consists of events in the entire cube. The computed covariation then determines causal inferences.

An Interpretation of Other Results Indicating Bias

Biases have been reported not only in experiments using materials framed in the rather artificial format summarizing configurational information but also in studies that used apparently less artificial formats. In many of these studies (e.g., Chapman & Chapman, 1967, 1969; Jones & Harris, 1967), subjects' implicit assumptions in their focal sets were not manipulated or measured; an interpretation of these findings according to our model (e.g., in terms of subjects' prior assumptions) would therefore be speculative. However, some of these studies did manipulate and, at least partially, measure subjects' assumptions. We present our interpretation of two findings reported in such studies.[9]

Bias against using consensus information. A controversial piece of evidence against the use of consensus information was reported by Nisbett and Borgida (1975). In one of their studies, they asked subjects to read a description of an experiment by Darley and Latané (1968), in which one of the participants was heard, over an intercom, having what sounded like a seizure; the other participants faced the decision of whether to help him. Some subjects (the consensus group) were told the rather surprising results of the study (that most participants helped only after considerable delay or never), whereas other subjects (control) were not. All subjects were then asked to explain the behavior of a (male) participant who never helped. One of the questions asked was "Was the behavior of the participant due to his personality or the situation?" Because most control subjects were found to assume that most people would have helped, Nisbett and Borgida interpreted the covariation principle to predict that the surprising consensus information should generate more situational responses: Whereas the control group should perceive the target participant as an exception among others who would have helped (and thus should attribute the failure to help to the target participant), the consensus group should perceive no variation across the participants with respect to helping (and thus should attribute the behavior to the situation rather than to any particular person). Nisbett and Borgida's results showed that consensus information had no effect on attribution, leading them to conclude that, contrary to common sense and prescriptive norms, people almost totally ignore consensus information.

Notice that none of Nisbett and Borgida's (1975) subjects were told whether the participants behaved similarly in situations other than the seizure situation. Without this information, subjects were presumably free to assume that they did. If subjects made this assumption, in addition to assuming that most people typically would help (as was reported), a main-effect contrast for the target participant would be predicted for the focal set (consisting of subjects' assumptions in addition to the respective experimenter-specified information) for both the consensus group and the control group. In fact, a main-effect contrast would be predicted for the entire group of participants in the consensus condition. That is, the effect (not helping) is more likely for the target person in the control condition and for the entire group of participants (including the target person) in the consensus condition than for the population in general.

Wells and Harvey (1977) found that, when it was emphasized to subjects in a similar experiment that the participants were randomly selected from the general population, subjects in the consensus group did produce more situational attributions, indicating that they did not ignore the consensus information. The effect of emphasizing the representativeness of the sample is to supply additional information concerning how the partici-

[9] Many before us have argued that the selection of information is a source of bias. None, however, have argued and demonstrated that the process of causal induction per se is unbiased.

pants probably behaved in other situations (namely, that they typically would help). With this additional information, a main-effect contrast for the seizure situation would be predicted for the consensus group (one is less likely to help in that situation than in other situations) but not for the control group (one is likely to help both in the seizure situation and in other situations). Thus, the differing results reported by Nisbett and Borgida (1975) and Wells and Harvey (1977) are both consistent with the unbiased use of covariation.

Actor–observer differences. Jones and Nisbett (1972) suggested that, whereas actors tend to attribute their own behavior to characteristics of the situation, observers tend to attribute an actor's behavior to personal characteristics of the actor. The proposed divergent perceptions of the actor and the observer have been documented in a number of studies (see Watson, 1982, for a review). One explanation offered for this divergence was in terms of the ambiguity of causal questions, which allows the adoption of different contrasting backgrounds (Einhorn & Hogarth, 1986; Hilton, 1990; Kahneman & Miller, 1986; McGill, 1989). For example, a question Nisbett, Caputo, Legant, and Maracek (1973) asked their subjects, "Why did you [your best friend] choose this major?", may be interpreted as (a) "Why did you [your best friend] choose this major in particular?" or (b) "Why did you [your best friend] in particular choose this major?" Hilton (1990) and McGill (1989) proposed that actors are likely to presuppose their own presence as a constant background factor and ask themselves what is special about the situation that caused the behavior, thus adopting the first interpretation in this example. In contrast, observers are likely to treat the situation as background and ask what is special about the actors that differentiates them from other people in the same situation, thus adopting the second interpretation in this example. Such a view predicts that if the causal question is disambiguated, the actor–observer differences should disappear.

The results of an experiment by McGill (1989) support this hypothesis, which she tested by comparing ambiguous causal questions to disambiguated versions as just illustrated. Actor–observer differences have been explained in terms of the adoption of differing contrasting backgrounds (Einhorn & Hogarth, 1986; Hilton, 1990; Jones & Nisbett, 1972; Kahneman & Miller, 1986; McGill, 1989). This explanation previously has been interpreted in terms of normality (Einhorn & Hogarth, 1986; Kahneman & Miller, 1986), conversational pragmatics (Hilton, 1990), or Mill's (1843/1973) method of difference (Hilton, 1990; McGill, 1989). We note that computing probabilistic contrasts over events in these differing backgrounds also predicts actor–observer differences. We discuss the alternative explanations later.

Implications of Phenomena Observed in Social Psychology and Philosophy

We have shown that the computation of contrasts over events in a focal set can explain deviations from normative predictions discussed in philosophy and social psychology. We believe that simultaneously considering phenomena in both domains imposes constraints on model construction that are not entirely satisfied by any previous model of causal induction proposed in either literature. On the one hand, the social causal-attribution literature underscores the fact that attributions are often based on probabilistic stimuli (e.g., "Ralph has almost always tripped over Joan's feet while dancing with her"). On the basis of such probabilistic stimuli, subjects are able to make conjunctive attributions as well as simple (i.e., single-factor) attributions. On the other hand, the philosophical literature convincingly shows that the concept of a focal set is central in explaining the distinction between causes and enabling conditions as well as the effects of shifting context on that distinction.

What is missing in previous accounts of causal induction in both social psychology and in philosophy—and what our model provides—is an account of probabilistic causal induction that specifies conjunctive as well as simple causes. Many models proposed in the literature on causes versus enabling conditions are deterministic (Hesslow, 1983, 1988; Hilton, 1990; Mackie, 1965, 1974). It is not obvious how these models can be generalized to account for conjunctive probabilistic causes, even if the probabilistic generalization for simple causes is straightforward. In social psychology, in which the phenomena to be explained are clearly probabilistic, some of the models are nonetheless deterministic (e.g., Försterling, 1989; Hewstone & Jaspars, 1987; Jaspars, 1983; the covariation principle in Kelley, 1967, 1973). Of those that are not, none provides a formal definition of conjunctive causes (e.g., Hilton & Slugoski, 1986; the ANOVA analogy in Kelley, 1967, 1973; McGill, 1989; see Cheng & Novick, 1990a, for a discussion of these models).

Moreover, models in social psychology lack an explicit and generalized concept of the focal set. The focal sets discussed in the literature on causes versus enabling conditions explain the distinction by being subsets of the universal set. Social theorists do implicitly assume that their subjects' focal sets are subsets of the universal set; for example, the condition of the target person being alive is never included as one of the potential causal factors despite the necessity of it for the person's reaction to a stimulus (the event to be explained). These theorists therefore circumvent the puzzle posed by the distinction. However, they have failed to generalize the concept of the focal set. In social causal attribution experiments, the actual focal set (i.e., the one used by subjects) often happens to be a *superset* of the subset assumed by the theorist. The implicitly assumed subsets, therefore, create rather than explain the reported biases in the social literature.

Our model builds on the previous work. It aims at satisfying the constraints imposed by phenomena in both literatures. Using the formal concept of probabilistic contrast, our model is able to account for simple and conjunctive attributions for probabilistic causal relations. Adopting the concept of a focal set, our model is able to explain the deviations in the social literature as well as the distinction among causes, enabling conditions, and causally irrelevant factors.

Other competing models of causal induction have been proposed in cognitive psychology and philosophy. In the following sections, we apply these constraints to our evaluation of these models. None of these models specify a formal account of the induction of conjunctive causes. We argue that these models do not provide an adequate account of the induction of even simple causes.

Linear Heuristics in Everyday Causal Reasoning

All linear-combination heuristics that have been proposed in the cognitive literature may be regarded as special cases of Schustack and Sternberg's (1981) model. Schustack and Sternberg (1981) described causal inference as a linear function of five variables. The variables have weights that are determined empirically by multiple regression. The first four variables involve information about covariation: *a*, the frequency of the joint presence of a potential cause and the effect; *b*, the frequency of the presence of the potential cause coupled with the absence of the effect; *c*, the frequency of the absence of the potential cause coupled with the presence of the effect; and *d*, the frequency of the joint absence of a potential cause and the effect (see Table 1). The fifth variable in their model is a measure of the strength of competing causes. Schustack and Sternberg's regression modeling showed that the weights for *a* and *d* were positive, whereas those for *b* and *c* were negative.

Their model is not normative in two respects. First, the four types of frequency information received different weights. Subjects showed a bias toward giving more weight to *a* and *b* (potential cause present) than to *c* and *d* (potential cause absent). Second, regardless of whether the weights are equal, their model makes anomalous predictions, as we explain.

A variety of specific linear heuristics apparently used by college students to assess covariation have been identified (e.g., Arkes and Harkness, 1983; Jenkins & Ward, 1965; Nisbett & Ross, 1980; Shaklee, 1983; Shaklee & Elek, 1988; Shaklee & Goldston, 1989; Shaklee & Hall, 1983; Shaklee & Mims, 1981, 1982; Shaklee & Tucker, 1980; Smedslund, 1963; Ward & Jenkins, 1965). We state next the four that have been reported to be used by a substantial proportion of subjects. The $a - c$ rule assesses the strength and direction of covariation by comparing *a* and *c*. That is, for cases in which the effect is present, if a potential cause is present more often than it is absent, the covariation is judged to be positive; conversely, if the potential cause is absent more often than it is present, the covariation is judged to be negative. This rule, which was used by 18% of Shaklee and Tucker's (1980) subjects and 36% of Shaklee and Hall's (1983) subjects, has weights of 1 and −1, respectively, for variables *a* and *c* and a weight of 0 for each of the other three variables in Schustack and Sternberg's model described previously. The *a* rule assesses covariation between two factors according to the magnitude of *a* (i.e., the joint presence of the two target factors). Jenkins and Ward (1965) reported that *a* was the best predictor of their subjects' responses. This rule has a weight of 1 for variable *a* and a weight of 0 for each of the remaining variables. The $a + d$ rule assesses covariation on the basis of the frequency of confirming cases (weights of 1 for *a* and *d* and a weight of 0 for each of the remaining variables). Ward and Jenkins (1965) reported the dominant usage of this rule for subjects receiving trial-by-trial information and those receiving trial-by-trial information in addition to summary information. The difference in sums of diagonal cells rule assesses the direction and strength of covariation by computing the difference between the sum of *a* and *d* and the sum of *b* and *c*. This rule, which was used by 35% of Shaklee and Tucker's subjects and 16% of Shaklee and Hall's subjects, has weights of 1 for both variables *a* and *d*, −1 for both variables *b* and *c*, and 0 for the competing-causes variable.

Table 1
Event Frequencies in a Contingency Table Formed by the Presence and Absence of a Causal Factor and of the Effect

	Effect	
Causal factor	Present	Absent
Present	a	b
Absent	c	d

What Do the Linear-Combination Heuristics Predict?

Is causal induction, fundamental as it intuitively seems, based on nothing more than a frail set of heuristics? We challenge this position in view of the intuitive distinction among causes, enabling conditions, and causally irrelevant factors.

Schustack and Sternberg's (1981; also see Downing et al., 1985) model was proposed to describe how people make causal inferences when given incomplete information about complex problems involving multiple factors. Although situations involving causes, enabling conditions, and causally irrelevant factors fall within the purview of their theory, predictions of linear models for such situations have not been considered previously. We derive such predictions here.

Let us examine the predictions of linear heuristics for a focal set in which one of the potential causal variables is constantly present. It is clear that, without the assumption of a focal set, these heuristics cannot account for the effects of changing contexts on causal inference. Even with such an assumption, these heuristics make anomalous predictions. Consider, for example, an answer to the question "What causes it to rain today?" for the focal set in which gravity is constantly present. For this factor in this focal set, because *c* and *d* both equal 0, the result of a linear combination of cell frequencies would be solely determined by *a* and *b*. The relative magnitudes of *a* and *b* will depend on the prevalence of other necessary conditions for rain in the events in one's knowledge base. For a resident of Edinburgh, for whom the other necessary conditions for rain are frequently present, *a* is much larger than *b*. In contrast, for a resident of Los Angeles, where it hardly ever rains, the opposite is true.

The output of a linear combination of cell frequencies is a number that may be positive, zero, or negative, corresponding, respectively, to a facilitatory cause, a causally irrelevant factor, and an inhibitory cause. Therefore, Schustack and Sternberg's (1981) linear-combination rule (for which the weights for *a* and *b* were roughly equal) and the difference in the sums of diagonal cells rule predict that a resident of Edinburgh would reply, "Gravity is a cause of rain," whereas a resident of Los Angeles would reply, "Gravity inhibits rain." For the *a* rule, the $a - c$ rule, and the $a + d$ rule, because *c* and *d* for gravity both equal 0 in this focal set, the causal strength of gravity is equal to *a*. Because *a* is always positive and is higher for a resident of Edinburgh than of Los Angeles, each of these rules predicts that residents of both cities would think gravity is a cause of rain and

that a resident of Edinburgh would believe that it is a stronger cause than would a resident of Los Angeles.

These predictions clearly contradict an ordinary person's intuition that gravity is not a cause of rain but merely a condition that enables rainfall *regardless* of the frequency of rain in one's experience. In sum, our analysis reveals that there is no theoretical construct in the output of a linear-combination rule that could correspond to an enabling condition. According to linear heuristics, an enabling condition has the same status as a cause (either facilitatory or inhibitory depending on the prevalence of the effect).[10]

More detrimental yet to these heuristics than the previously mentioned anomalous predictions, none of these heuristics can distinguish the causal status of a factor such as gravity from causally irrelevant factors that are nearly always present in one's experience, such as houses and automobile exhaust for the inhabitants of Edinburgh and Los Angeles. The virtually constant presence of such factors implies that c and d would be much smaller than a and b and, therefore, that the result of a linear combination of cell frequencies would be largely determined by a and b. Thus, the same predictions would be made for the presence of houses or automobile exhaust as for gravity according to any of these rules! Clearly, no one would consider the presence of either of these factors to be a cause or an inhibitor of rain or the extent to which either factor causes or inhibits rain to be dependent on the frequency of rainfall in one's experience.

In sum, even with the assumption that linear combinations are computed over a focal set of events, the linear heuristics proposed in the cognitive literature erroneously predict that enabling conditions and causally irrelevant factors that are always present (or nearly always present) in one's experience have the same status as either facilitatory or inhibitory causes depending on the prevalence of the effect.

Arguments in Defense of Linear Heuristics

A number of arguments may be made in defense of these heuristics. First, it may be argued that such heuristics should apply only to factors that are attended to. Because factors that are virtually constantly present in one's experience are not salient (although one might argue that the constant presence of automobile exhaust in Los Angeles is quite salient), causal heuristics are not likely ever to be applied to them. In the special case of Schustack and Sternberg's (1981) model, it may in addition be argued that, because of the fifth variable in their model (the strength of competing causes), a normatively covarying competing factor (which is predicted to have greater causal strength than constant factors) would reduce the causal strengths of the constant factors, rendering the predictions for the constant factors less important.

However, these arguments do not explain why, among factors that are virtually constantly present, people differentiate between enabling conditions (e.g., gravity with respect to rain) and causally irrelevant factors (e.g., the presence of automobile exhaust or houses with respect to rain), a differentiation that clearly is made. Moreover, counter to the argument based on salience, even when attention is deliberately brought to the cell frequencies for these factors, people are quite unlikely to be persuaded that these factors are indeed causes and inhibitors.

A second argument is that normatively irrelevant (i.e., noncovariational) factors that are constantly present, such as houses and automobile exhaust in the rain example, are considered to be spurious causes despite the perceived covariation according to linear heuristics. One weakness of this argument is that people probably would not acknowledge any covariational relation at all between rain and causally irrelevant factors such as houses. (In contrast, it seems that people would readily acknowledge a covariational, but noncausal, relation between a drop in the barometric reading and the approach of a storm.) Moreover, this argument cannot be applied to the predictions of linear models regarding constant factors that are normatively causally relevant (gravity is not a spurious cause of rain). Whereas normatively covariational but noncausal relations pose a problem for all purely covariational accounts of causal induction, normatively *noncovariational* relations that are constantly present pose a unique problem for linear models.

A third argument might be that the distinction between a cause and an enabling condition reflects the conversational principle of being *informative* to the inquirer given assumptions about his or her state of knowledge. Thus, whereas a cause is always a condition assumed to be unknown to the hypothetical inquirer (otherwise there would be no reason for asking), an enabling condition is typically a condition assumed to be already known to the inquirer (Hilton, 1990; Mill, 1843/1973; Turnbull, 1986; Turnbull & Slugoski, 1988; cf. Grice, 1975). For example, a competent adult inquiring about an airplane crash presumably does not know about the malfunctioning of the critical component in the airplane but does know that the gravity of the earth exerts a downward force. On this hypothesis, causes and enabling conditions do not reflect differences in underlying beliefs about the true causes of events but rather differences in the informativeness of a covarying factor with respect to an inquirer.

This hypothesis cannot absolve linear heuristics of their transgression in predicting normatively causally irrelevant factors that are constantly present to be either facilitatory or inhibitory causes depending on the prevalence of the effect (e.g., houses inhibit rain in Los Angeles)—the facilitatory or inhibitory status of these factors can hardly be assumed to be already known to the inquirer. For the same reason, neither can this hypothesis prevent such heuristics from predicting a constantly present necessary condition to be an inhibitory cause when the target effect is rare (e.g., gravity inhibits rain in Los Angeles).

This hypothesis can potentially amend linear heuristics only for those cases in which the target effect is prevalent, when a constantly present necessary condition is predicted by these heuristics to be a facilitatory cause (e.g., gravity with respect to rain in Edinburgh). In these cases, it might be argued that the causal status of such a condition is already known to the inquirer. Even for such cases, however, we (Cheng & Novick, 1991) reported evidence showing that the distinction between

[10] For the special case in which the weighted sum happens to be zero, the enabling condition has the same status as a causally irrelevant factor.

causes and enabling conditions cannot be explained by conversational pragmatics.

A fourth argument might be that knowledge such as gravity as an enabling condition for rain or oxygen as an enabling condition for fire is based on academic instruction rather than natural computation. Although it is dubious that the concept of an enabling condition is part of formal instruction, one potential explanation is that academic knowledge is perceived to be less intuitively compelling, albeit more reliable, than naturally computed covariations. Being from a more authoritative source, such knowledge overrides naturally computed covariations; but being less intuitively compelling, it yields the status of merely enabling conditions. Note, however, that because academic instruction remains constant for an individual across contexts at any particular time, this argument cannot account for the effects of context on causal judgments. We review later one of our experiments (Cheng & Novick, 1991, Experiment 2) in which subjects' perceptions of an enabling condition shifted across two contexts because of a manipulation of the focal set, indicating that the status of an enabling condition was not due to academic training.

Explanation of Evidence Supporting Linear Heuristics

If linear heuristics do not describe natural causal induction, why were a variety of them reported to be used by college students to assess covariation? Crocker (1981) and Beyth-Marom (1982) observed that the phrasing of a covariation question seems to influence the particular heuristic used. More specifically, Beyth-Marom noted that the instructions given to subjects regarding the task often emphasized certain aspects of covariation, with the emphasis differing from experiment to experiment. For example, in a task involving the relationship between cloud seeding and rainfall, Ward and Jenkins (1965) told their subjects, "At the end of the experiment. . . you are to judge how much control seeding the clouds had over the occurrence of rainfall. . . . Complete control means that whenever you seed, it rains, and whenever you don't seed, it does not rain" (p. 235). These instructions emphasized the confirming cases (Cells *a* and *d*), exactly those on which their subjects based their judgments. Similarly, emphases on various cell frequencies reflected in the instructions given in the experiments reported by Alloy and Abramson (1979), Jenkins and Ward (1965), Shaklee and Tucker (1980), and Smedslund (1963) were found to closely mirror the biases, or lack of biases, observed in the respective studies. Consistent with Beyth-Marom's analysis, Schustack and Sternberg's (1981) finding regarding higher weights for *a* and *b* (causal factor present) compared with *c* and *d* (causal factor absent) may reflect their instructions to "determine the likelihood that a particular one of the possible causes, in isolation, leads to the outcome" (p. 106). To explain their subjects' emphasis on sufficiency rather than necessity (*a* and *b* rather than *c* and *d*), Schustack and Sternberg (1981) noted, "In our experiments. . . the task was more specific than that of evaluating 'causality'; our subjects were evaluating the probability of the occurrence of the outcome in the *presence* [italics added] of the target" (p. 116). (In terms of our model, the latter assessment concerns p_i, only *one* of the two proportions in our definition of a main-effect contrast.) As Beyth-Marom suggested, subjects in these experiments "appear to do what they are told to do" (p. 513). Because the issue of how causality is related to the dependent variables in these experiments is left unaddressed, the biases reported in this literature are difficult to interpret.

Suppes's Model

According to Suppes (1984), an event C is a cause (a "prima facie cause" in his terminology) of an event E (the effect) if and only if (a) C occurs earlier than E, (b) $P(C) > 0$, and (c) $P(E|C) > P(E)$, where $P(E)$ is the unconditional probability of E occurring. Let ΔP_C represent the difference between $P(E|C)$ and $P(E)$. Because $P(E|C)$ is compared with the baseline defined by $P(E)$, it follows that for a potential cause C with any given $P(E|C)$, ΔP_C will be larger when $P(E)$ is small than when it is large. That is, C will be a stronger or more likely cause when E is rare than when it is prevalent. An exception to this, which we discuss later, is the case in which C is constantly present.

Consider the following two ways in which $P(E)$ may vary for C. Either there is a single cause of E or there are multiple causes. When E has the single cause C, $P(E)$ is a function of $P(C)$. When there are multiple causes of E, $P(E)$ is again a function of $P(C)$. In addition, $P(E)$ may vary depending on how often other causes produce E.

We argue that the predicted variation in ΔP_C as a result of variations in $P(C)$ is anomalous in the case in which C is the only cause (i.e., $P(E \cap \bar{C}) = 0$). We see that in this case

$$\Delta P_C = P(E|C) - P(E) = P(E|C) - P(E \cap C) = P(E|C) \cdot [1 - P(C)].$$

Therefore, ΔP_C is larger when C is rare than when it is prevalent. To take a concrete example, consider the strength of the cause of Down's syndrome for children born of older women and those born of younger women. It is known that the genetic defect that deterministically leads to the syndrome occurs vastly more often among infants of older women than among those of younger women. Thus, Suppes's model predicts that the relevant genetic defect should be perceived as a stronger cause of the syndrome for infants of younger women (for whom the cause is rare) than for those of older women (for whom the cause is more prevalent).

In contrast, the probabilistic contrast for the genetic defect is equal for the two groups of children. For either group, $\Delta p_c = p_c - p_{\bar{c}} = 1.0$. Our model, therefore, correctly predicts that the genetic defect has identical causal strength for the two groups of children.

Let us now consider the case in which there are multiple independent causes of E. In this case, the prevalence of E varies not only as a function of $P(C)$ but also as a function of how often causes other than C produce E. When these other causes often produce E, $P(E)$ will be relatively large. Therefore, for any given C, ΔP_C will be relatively small. Conversely, when other causes rarely produce E, ΔP_C will be relatively large. Like Equation 1, then, Suppes's rule also predicts a reduced contrast as a result of the prevalence of the effect due to other causes (see the Appendix).

In sum, both Suppes's rule and Equation 1 in our model

correctly predict that the prevalence of other causes influences the strength of a potential cause. However, Suppes's rule erroneously predicts that a particular cause should be perceived as stronger when it is rare than when it is prevalent.

Probabilistic Extension of Mill's Method of Difference

Several theorists hypothesized that causal explanation involves the application of Mill's (1843/1973) method of difference to a target episode and a contrasting causal background; that is, a cause is the difference between a target episode in which the to-be-explained effect is present and a contrasting causal background in which that effect is absent (Hilton, 1990; Mackie, 1965, 1974; McGill, 1989). Notice that events are partitioned according to the presence versus the absence of the effect. If one were to recast this hypothesis probabilistically, it would therefore seem most natural and accurate to formulate it in terms of the probability of a potential causal factor conditional on the presence versus the absence of the effect; that is, the contrast rule for potential cause C and effect E is

$$\Delta P_C = P(C|E) - P(C|\bar{E}). \quad (3)$$

In a focal set in which C is constantly present, $P(C|E) = P(C|\bar{E}) = 1$. Therefore, this extension of Mill's method of difference predicts that the factor will have a contrast of 0. Note that for this focal set, regardless of the prevalence of E, $P(E|C) = P(E)$. Therefore, Suppes's model also predicts that C will have a contrast of 0.

One potential interpretation of this result is that C is causally irrelevant in such cases. This interpretation does not permit the prediction of an enabling condition, which seems appropriate when C is causally relevant in some other focal set. These models, however, can be amended by defining an enabling condition as a factor that has a contrast of 0 within the current focal set but a positive contrast in another focal set. This amendment, however, implies that an enabling condition involves conflicting information from two focal sets, one indicating it to be a cause and the other indicating it to be causally irrelevant.

Let C_1 be a factor that is constantly present in the current focal set (e.g., a suspect's being alive at the time a crime was committed in the context of the question "Who caused the disappearance of a wallet from its owner's pocket?") but is causally relevant (i.e., is a cause in another focal set). Now, let C_2 be a cause in some focal set but, within the current focal set, occur with equal nonzero probability when the effect is present as when it is absent. Simple algebra shows that within the current focal set, according to both Suppes's model and Equation 3, C_2 should have a contrast of 0 (likewise according to our model). Note that according to Suppes's model and Equation 3, because C_1 and C_2 each have a contrast of 0 within the current focal set and is a cause in another focal set, the causal statuses of C_1 and of C_2 are indistinguishable. This is true with or without the amendment.

C_1 and C_2 do not seem indistinguishable to us. Consider the previous question about the disappearance of the wallet and the potential causal factor of the suspects "being alive." Let this factor be constantly present in the set of potential suspects (Focal Set 1) but known to be necessary for the effect (guilt in a crime) judging from another focal set (Focal Set 2). Imagine arriving at a world (Focal Set 3) in which being alive at the time a crime was committed is irrelevant for guilt in that crime (e.g., suppose there are ghosts who can steal). The causal status of "being alive" in our world and in this novel world do not seem indistinguishable. Indeed, one might find the change quite disconcerting: "Being alive," something that is a constant condition for theft in our world (Focal Set 1), becomes causally irrelevant in this novel world (Focal Set 3). Whereas juxtaposing information from Focal Sets 1 and 2 to arrive at the integrative status of an enabling condition is undisturbing, juxtaposing information from Focal Sets 3 and 2 produces a clear conflict.

In sum, we judge C_1 (e.g., the factor of "being alive" with respect to theft in Focal Set 1) to be neither causally irrelevant nor indistinguishable from C_2 (e.g., "being alive" in Focal Set 3). These intuitions contradict the predictions made by Suppes's model and by our probabilistic extension of Mill's method of difference.

Normality Criterion

The normality criterion is a prominent explanation of the distinction between causes and enabling conditions that has held sway ever since it was first proposed by the philosophers Hart and Honoré (1959/1985) and Mackie (1965, 1974). A number of psychologists who have considered the distinction concurred with this explanation (Einhorn & Hogarth, 1986; Hilton & Slugoski, 1986; Kahneman & Miller, 1986; Turnbull & Slugoski, 1988; see Cheng & Novick, 1991, for a review). Hart and Honoré maintained that central to the commonsensical concept of cause, and at least as essential as the notions of invariable or constant sequence stressed by Mill and Hume, is the notion of human intervention in a course of events that would normally take place. Postulating the generalization of this notion to cases in which there is no literal human intervention, they suggested that a cause is "a *difference* from the normal course which accounts for the difference in the outcome" (p. 29). On this view, among the set of factors that are individually necessary and jointly sufficient to produce an effect (e.g., an airplane crash, a couch on fire), an abnormal factor (e.g., the malfunctioning of a component in the airplane, a dropped cigarette) will be designated as the cause, whereas normal factors (e.g., the gravitational pull of the earth, the combustibility of the couch) are merely enabling conditions. Here we limit our discussion of this criterion to its interpretation in terms of the statistical sense of prevalence. (See Cheng & Novick, 1991, for tests of two other interpretations of this criterion: namely, Kahneman & Miller's, 1986, default value interpretation and Mackie's, 1974, interpretation of normality in its ethical sense of a correct standard.) Under the statistical interpretation, normality is defined by the prevalence of the causal factor in the context (i.e., focal set) under consideration.

Cheng and Novick (1991) noted that one of the limitations of the normality view is that it does not account for the perception of the causes of prevalent events (e.g., objects staying in place instead of floating weightlessly), which have prevalent factors (e.g., the mass of the object, the gravitational pull of the earth, and so on) that are individually necessary and jointly sufficient to produce the effect. Although people in everyday life typically ask about the causes of only rare events (e.g., Kahneman &

Miller, 1986; Lehnert, 1978; Weiner, 1985), leaving it to scientists to ask questions about prevalent events and discover (or invent) concepts such as gravity, ordinary people do perceive and understand such concepts when they are used in everyday contexts. For example, even though gravity is ubiquitous on the surface of the earth, the statement "The earth's gravity causes objects near its surface to fall" does not sound anomalous (even though a physicist might not put it that way). It clearly carries the usual causal implication that without the cause (e.g., in a special gravity-free chamber) the effect would not occur (i.e., objects would not fall). The statement might be made, for example, in answer to a child's question. It seems to us, then, that a major weakness of the normality position is that it cannot account for ordinary people's perception of causality regarding prevalent events, thereby implying that two distinct mechanisms underlie people's concepts of causality in everyday versus scientific situations.

We propose here that the normality criterion should be regarded as a special case of our model, which can account for causal induction involving either prevalent or rare events with a single mechanism. As illustrated in Figure 1, factors that have a noticeable probabilistic contrast with respect to an effect can be either prevalent or rare. In Focal Set A, the factor that has a large contrast (r) is rare. However, in Set C, the factor that has a large contrast (q) is prevalent. The normality criterion corresponds to the case in which the probabilistic contrast is computed for an effect that is rare in the context in question (as in Set A of Figure 1), with the exception of cases in which the cause of a rare effect (e.g., skin cancer) is prevalent (sunlight). These exceptions involve factors whose contrast values are small but nonetheless noticeable.

Our model overcomes the inability of the normality criterion to account for the perception of causality regarding prevalent events by differentiating between two concepts that are conflated in the normality view—the constant presence of a potential factor and the prevalence of such factors. We predict that people do differentiate between the two.

Manipulating the Prevalence of the Cause and the Effect

Linear heuristics, Suppes's (1984) model, and the normality criterion all predict an impact of the prevalence of a potential causal factor or the prevalence of the effect, or both. In contrast, our model predicts that although the constancy of a potential causal factor will influence causal judgments, the prevalence of neither the factor nor the effect will have any impact in situations in which the effect is not simultaneously produced by multiple alternative causes.

We (Cheng and Novick, 1991, Experiment 2) manipulated (a) the constancy of causal factors and (b) the prevalence of the causal factors and of the effect. More specifically, we manipulated which factor covaried with an effect (plant growth) and which remained constant in two scenarios. One scenario was about the blooming of dandelions, and the other was about the maturation of corn plants. For each scenario, the effect was prevalent in one version and rare in another. In each scenario, one factor covaried with the effect. This factor differed across the scenarios (sunlight in the dandelion scenario versus nutrients in the soil in the corn scenario). The remaining three factors were held constant in each scenario: two were necessary for the effect according to subjects' prior knowledge (water in both scenarios plus nutrients or sunlight) and one was not (the presence of a house next to the plants). Within each scenario, the covarying factor was either prevalent or rare in accord with the prevalence of the effect.

Prevalence was defined for the effect and the positive value of the covarying factor by describing them as occurring in either most of or a few of the cases in the given context. For example, in the prevalent-corn scenario, many corn plants matured (i.e., the effect occurred) in four of the five cornfields tended by a farmer. These four fields had virgin soil (the positive value of the covarying factor), whereas the fifth field had its soil depleted of nutrients by previous farming.

At the end of each scenario was a question on what caused the growth of the relevant plants (e.g., what caused the corn plants to mature in the four recently cleared fields?). Subjects were asked to indicate the causal status (cause, enabling condition, causally irrelevant factor, or inhibitor) of each of the four factors in each scenario. To test the linear heuristics against our model, a second question asked whether each of four items inhibited the growth of the plants. Two of these items were constantly present in the specified focal set—one a necessary factor (e.g., sunlight in the corn scenario), the other unnecessary (the house in both scenarios). The other two items were negative values of two necessary factors—one the covarying factor (e.g., lack of nutrients for the corn scenario), the other a constant factor (e.g., lack of water in the corn scenario).

Before subjects read the scenarios, we introduced our terminology by giving a brief explanation of the distinction between causes and enabling conditions in terms of an example, chosen so as to be neutral with respect to all alternative models that allow the construct of an enabling condition. To measure the focal sets perceived by the subjects, after they made judgments on the causal status of the various factors, they were asked to rate how accurately each of three expanded questions that specified different focal sets reflected their interpretation of the causal question in the scenario. This question served to ensure that our manipulation of focal sets was effective (and it was).

Our model predicts that a potential causal factor that covaries with the effect in the focal set will be considered a cause and will be distinguished from necessary factors that are constantly present in that set. In particular, it predicts that the two scenarios will produce shifts in judgments concerning causes and enabling conditions: Factors that yield a large positive probabilistic contrast in the focal set (sunlight in the dandelion scenario vs. nutrients in the corn scenario) will be perceived as causes; conversely, those that yield a large negative contrast (lack of sunlight vs. lack of nutrients in the respective scenarios) will be perceived as inhibitors. It also predicts that necessary factors that are constant in that set (nutrients and water in the dandelion scenario vs. sunlight and water in the corn scenario) will be perceived as enabling conditions, whereas unnecessary factors that are constant in that set (the house in both scenarios) will be perceived as causally irrelevant. These predictions are independent of the prevalence of the covarying factor (e.g., whether most or few areas have rich soil) and of the effect (e.g., whether most or few corn plants matured) in the focal set.

In contrast, the normality view predicts that, within each

scenario (dandelion or corn), the prevalence of the factors should influence causal judgments. In particular, only in the rare versions should necessary factors be considered causes; in the prevalent versions, necessary factors should be considered enabling conditions despite high probabilistic contrasts for those factors within the focal set. Like the normality view (but for a different reason), Suppes's (1970, 1984) contrast rule predicts that a rare necessary factor will be more likely to be considered a cause than a prevalent one, if one assumes that a factor with a large contrast is more likely to be considered a cause than a factor with a much smaller contrast.

The linear-combination heuristics predict that the prevalence of the effect should influence causal judgments on all factors that remain constant in the focal set regardless of whether they are necessary for the effect. According to heuristics that have a positive weight for a and a negative weight for b, when the effect is prevalent, these constant factors should be considered causes; when the effect is rare, these factors should be considered inhibitors. For example, in the prevalent-corn scenario, the house, sunlight, and water should be identified as causing the corn plants to mature, whereas in the rare version of the scenario, these factors should be seen as inhibiting maturation.

In support of our model, the results indicated that manipulating which factor has a large probabilistic contrast across scenarios had a huge effect on causal judgments. The effect of shifting scenarios also indicates that subjects based their judgments on the focal sets they perceived in the scenarios we constructed rather than merely on their prior knowledge, including book-learned knowledge. Within each scenario, causal judgments were just as predicted by our model but contrary to what were predicted by the normality criterion, the linear-combination heuristics, and Suppes's contrast rule. In particular, varying the prevalence of either the potential causes or the effect had absolutely no impact on judgments of causal status.

Summary and Conclusion

Our probabilistic contrast model integrates normative models of covariation proposed in the cognitive and social psychology literatures and the animal behavior literature, extending them by developing formal definitions of simple and conjunctive causes and by adding an explicit assumption of computation of covariation over focal sets (an idea adapted from the philosophical literature). The present review of our explanations of deviations from normative covariation in the disparate domains of philosophy and social psychology shows that the same concept of computation of probabilistic contrasts over events in a focal set underlies causal induction in both domains. Adding to our previous theoretical analyses (Cheng & Novick, 1990a, 1991), we evaluate alternative theories of causal induction in view of the constraints imposed by a simultaneous consideration of phenomena in the two domains. Our analysis reveals that no alternative model of causal induction in cognitive and social psychology and in philosophy satisfies those constraints. Moreover, we note that (a) Suppes's (1984) model erroneously predicts that the assessment of a potential cause is a function of its prevalence, (b) the class of heuristics based on linear combinations of cell frequencies erroneously predicts that the assessment of a potential cause is a function of the prevalence of the target effect, and (c) the clear difference between a causally irrelevant factor and a constant but relevant condition favors our contrast rule over both an amended version of Suppes's rule and a probabilistic extension of Mill's (1973) method of difference. Furthermore, we derive Kelley's (1971) discounting principle as a corollary of our modification of his ANOVA model. We also specify how the normality criterion and previous explanations of the distinction between causes versus enabling conditions in terms of necessity and sufficiency may be regarded as special cases of our model.

In addition to extending our previous theoretical analyses, we extend our interpretation of previous empirical results. We show that (a) our (Cheng and Novick, 1991, Experiment 2) findings contradict the predictions made by the linear-combination rules and Suppes's (1984) model, (b) the differing results reported by Nisbett and Borgida (1975) and Wells and Harvey (1977) in their controversial debate regarding the use of consensus information are both consistent with our model, and (c) the differing attributions of actors and observers are also consistent with our model.

It seems that causal induction is the proverbial elephant. Many researchers reported on various of the multiple facets of covariation computation, a component of causal induction that has been regarded as essential. These seemingly inconsistent, nonoptimal, shifting facets of this putatively essential component have led some to conclude that a coherent elephant of causality does not exist. We by no means claim that we have a complete view of the beast; as we mentioned, there are important aspects of causality that we skirt. On the basis of our own work and our interpretation of others' reports, however, we believe we have put together enough pieces to suggest that an elephant is indeed there and that it shows signs of being an adaptive animal.

References

Alloy, L. B., & Abramson, L. Y. (1979). Judgment of contingency in depressed and nondepressed students: Sadder but wiser? *Journal of Experimental Psychology: General, 108,* 441–485.

Alloy, L. B., & Tabachnik, N. (1984). Assessment of covariation by humans and animals: The joint influence of prior expectations and current situational information. *Psychological Review, 91,* 112–149.

Arkes, H. R., & Harkness, A. R. (1983). Estimates of contingency between two dichotomous variables. *Journal of Experimental Psychology: General, 112,* 117–135.

Beyth-Marom, R. (1982). Perception of correlation reexamined. *Memory & Cognition, 10,* 511–519.

Bullock, M., Gelman, R., & Baillargeon, R. (1982). The development of causal reasoning. In W. J. Friedman (Ed.), *The developmental psychology of time* (pp. 209–254). San Diego, CA: Academic Press.

Chapman, L. J., & Chapman, J. P. (1967). Genesis of popular but erroneous psychodiagnostic observations. *Journal of Abnormal Psychology, 73,* 193–204.

Chapman, L. J., & Chapman, J. P. (1969). Illusory correlations as an obstacle to the use of valid psychodiagnostic signs. *Journal of Abnormal Psychology, 74,* 271–280.

Cheng, P. W., & Novick, L. R. (1990a). A probabilistic contrast model of causal induction. *Journal of Personality and Social Psychology, 58,* 545–567.

Cheng, P. W., & Novick, L. R. (1990b). Where is the bias in causal

attribution? In K. Gilhooly, M. Keane, R. Logie, & G. Erdos (Eds.), *Lines of thought: Reflections on the psychology of thinking* (pp. 181–197). Chichester, England: Wiley.

Cheng, P. W., & Novick, L. R. (1991). Causes versus enabling conditions. *Cognition, 40,* 83–120.

Crocker, J. (1981). Judgment of covariation by social perceivers. *Psychological Bulletin, 90,* 272–292.

Darley, J. M., & Latané, B. (1968). Bystander intervention in emergencies: Diffusion of responsibility. *Journal of Personality and Social Psychology, 8,* 377–383.

Downing, C. J., Sternberg, R. J., & Ross, B. H. (1985). Multicausal inference: Evaluation of evidence in causally complex situations. *Journal of Experimental Psychology: General, 114,* 239–263.

Einhorn, H. J., & Hogarth, R. M. (1986). Judging probable cause. *Psychological Bulletin, 99,* 3–19.

Estes, W. K. (1964). Probability learning. In A. W. Melton (Ed.), *Categories of human learning* (pp. 89–128). San Diego, CA: Academic Press.

Försterling, F. (1989). Models of covariation and attribution: How do they relate to the analogy of analysis of variance? *Journal of Personality and Social Psychology, 57,* 615–625.

Gallistel, C. R. (1990). *The organization of learning.* Cambridge, MA: MIT Press.

Garcia, J., McGowan, B., Ervin, F., & Koelling, R. (1968). Cues: Their relative effectiveness as reinforcers. *Science, 160,* 794–795.

Garcia, J., McGowan, B., & Green, K. F. (1972). Sensory quality and integration: Constraints on conditioning. In A. H. Black & W. F. Prokasy (Eds.), *Classical conditioning II: Current research and theory.* New York: Appleton-Century-Crofts.

Grice, H. P. (1975). Logic and conversation. In P. Cole & J. L. Morgan (Eds.), *Syntax and semantics* (Vol. 3, pp. 41–58). San Diego, CA: Academic Press.

Hart, H. L., & Honoré, A. M. (1985). *Causation in the law* (2nd ed.). Oxford, England: Oxford University Press. (Original work published 1959)

Hastie, R. L. (1983). Social inference. *Annual Review of Psychology, 34,* 511–542.

Hesslow, G. (1983). Explaining differences and weighting causes. *Theoria, 49,* 87–111.

Hesslow, G. (1988). The problem of causal selection. In D. Hilton (Ed.), *Contemporary science and natural explanation: Commonsense conceptions of causality* (pp. 11–32). Brighton, England: Harvester Press.

Hewstone, M. R. C., & Jaspars, J. M. F. (1987). Covariation and causal attribution: A logical model of the intuitive analysis of variance. *Journal of Personality and Social Psychology, 53,* 663–672.

Hilton, D. J. (1988). Logic and causal attribution. In D. Hilton (Ed.), *Contemporary science and natural explanation: Commonsense conceptions of causality* (pp. 33–65). Brighton, England: Harvester Press.

Hilton, D. J. (1990). Conversational processes and causal explanation. *Psychological Bulletin, 107,* 65–81.

Hilton, D. J., & Slugoski, B. R. (1986). Knowledge-based causal attribution: The abnormal conditions focus model. *Psychological Review, 93,* 75–88.

Hilton, D. J., Smith, R. H., & Alicke, M. D. (1988). Knowledge-based information acquisition: Norms and the functions of consensus information. *Journal of Personality and Social Psychology, 55,* 530–540.

Jaspars, J. M. F. (1983). The process of causal attribution in common sense. In M. R. C. Hewstone (Ed.), *Attribution theory: Social and functional extensions* (pp. 28–44). Oxford, England: Basil Blackwell.

Jaspars, J. M. F., Hewstone, M. R. C., & Fincham, F. D. (1983). Attribution theory and research: The state of the art. In J. M. F. Jaspars, F. D. Fincham, & M. R. C. Hewstone (Eds.), *Attribution theory: Essays and experiments* (pp. 3–36). San Diego, CA: Academic Press.

Jenkins, H., & Ward, W. (1965). Judgment of contingency between responses and outcomes. *Psychological Monographs, 79,* 1–17.

Jones, E. E., Davis, K. E., & Gergen, K. J. (1961). Role playing variations and their informational value for person perception. *Journal of Abnormal and Social Psychology, 63,* 302–310.

Jones, E. E., & Harris, V. A. (1967). The attribution of attitudes. *Journal of Experimental Social Psychology, 3,* 1–24.

Jones, E. E., & Nisbett, R. E. (1972). The actor and the observer: Divergent perceptions of the causes of behavior. In E. E. Jones, D. E. Kanouse, H. H. Kelley, R. E. Nisbett, S. Valins, & B. Weiner (Eds.), *Attribution: Perceiving the causes of behavior* (pp. 79–94). Morristown, NJ: General Learning Press.

Kahneman, D., & Miller, D. T. (1986). Norm theory: Comparing reality to its alternatives. *Psychological Review, 93,* 136–153.

Kelley, H. H. (1967). Attribution theory in social psychology. In D. Levine (Ed.), *Nebraska symposium on motivation* (Vol. 15, pp. 192–238). Lincoln: University of Nebraska Press.

Kelley, H. H. (1971). *Attribution in social interaction.* Morristown, NJ: General Learning Press.

Kelley, H. H. (1973). The processes of causal attribution. *American Psychologist, 28,* 107–128.

Kelley, H. H., & Michela, J. L. (1980). Attribution theory and research. *Annual Review of Psychology, 31,* 457–501.

Lehnert, W. (1978). *The process of question answering.* Hillsdale, NJ: Erlbaum.

Lepper, M. R., Greene, D., & Nisbett, R. E. (1973). Undermining children's intrinsic interest with extrinsic reward: A test of the overjustification hypothesis. *Journal of Personality and Social Psychology, 28,* 129–137.

Mackie, J. L. (1965). Causes and conditions. *American Philosophical Quarterly, ii,* 245–264.

Mackie, J. L. (1974). *The cement of the universe: A study of causation.* Oxford, England: Clarendon Press.

Marr, D. (1982). *Vision.* New York: Freeman.

McGill, A. L. (1989). Context effects in judgments of causation. *Journal of Personality and Social Psychology, 57,* 189–200.

Mendelson, R., & Shultz, T. R. (1976). Covariation and temporal contiguity as principles of causal inference in young children. *Journal of Experimental Child Psychology, 22,* 408–412.

Mill, J. S. (1973). A system of logic ratiocinative and inductive. In J. M. Robson (Ed.), *Collected works of John Stuart Mill* (Vols. 7, 8). Toronto, Canada: University of Toronto Press. (Original work published 1843)

Nisbett, R. E., & Borgida, E. (1975). Attribution and the psychology of prediction. *Journal of Personality and Social Psychology, 32,* 932–943.

Nisbett, R. E., Caputo, C., Legant, P., & Maracek, J. (1973). Behavior as seen by the actor and as seen by the observer. *Journal of Personality and Social Psychology, 27,* 154–164.

Nisbett, R. E., Krantz, D. H., Jepson, C., & Kunda, Z. (1983). The use of statistical heuristics in everyday inductive reasoning. *Psychological Review, 90,* 339–363.

Nisbett, R. E., & Ross, L. (1980). *Human inference: Strategies and shortcomings of social judgment.* Englewood Cliffs, NJ: Prentice-Hall.

Novick, L. R., Fratianne, A., & Cheng, P. W. (1991). *Knowledge-based assumptions in causal induction.* Unpublished manuscript, Vanderbilt University.

Orvis, B. R., Cunningham, J. D., & Kelley, H. H. (1975). A closer examination of causal inference: The roles of consensus, distinctiveness, and consistency information. *Journal of Personality and Social Psychology, 32,* 605–616.

Pruitt, D. J., & Insko, C. A. (1980). Extension of the Kelley attribution model: The role of comparison-object consensus, target-object consensus, distinctiveness, and consistency. *Journal of Personality and Social Psychology, 39,* 39–58.

Reichenbach, H. (1956). *The direction of time.* Berkeley: University of California Press.

Rescorla, R. A. (1968). Probability of shock in the presence and absence of CS in fear conditioning. *Journal of Comparative and Physiological Psychology, 66,* 1–5.

Salmon, W. C. (1980). Probabilistic causality. *Pacific Philosophical Quarterly, 61,* 50–74.

Salmon, W. C. (1984). *Scientific explanation and the causal structure of the world.* Princeton, NJ: Princeton University Press.

Schustack, M. W., & Sternberg, R. J. (1981). Evaluation of evidence in causal inference. *Journal of Experimental Psychology: General, 110,* 101–120.

Shaklee, H. (1983). Human covariation judgment: Accuracy and strategy. *Learning and Motivation, 14,* 433–448.

Shaklee, H., & Elek, S. (1988). Cause and covariate: Development of two related concepts. *Cognitive Development, 3,* 1–13.

Shaklee, H., & Goldston, D. (1989). Development in causal reasoning: Information sampling and judgment rule. *Cognitive Development, 4,* 269–281.

Shaklee, H., & Hall, L. (1983). Methods of assessing strategies for judging covariation between events. *Journal of Educational Psychology, 75,* 583–594.

Shaklee, H., & Mims, M. (1981). Development of rule use in judgment of covariation between events. *Child Development, 52,* 317–325.

Shaklee, H., & Mims, M. (1982). Sources of error in judging event covariations: Effects of memory demands. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 8,* 208–224.

Shaklee, H., & Tucker, D. (1980). A rule analysis of judgments of covariation between events. *Memory & Cognition, 8,* 459–467.

Shultz, T. R. (1982). Rules of causal attribution. *Monographs of the Society for Research in Child Development, 47* (Serial No. 194).

Skyrms, B. (1986). *Choice and chance: An introduction to inductive logic* (3rd ed.). Belmont, CA: Wadsworth.

Smedslund, J. (1963). The concept of correlation in adults. *Scandinavian Journal of Psychology, 4,* 165–173.

Suppes, P. (1970). *A probabilistic theory of causality.* Amsterdam: North-Holland.

Suppes, P. (1984). *Probabilistic metaphysics.* Oxford, England: Basil Blackwell.

Taylor, A. J. P. (1983). *The origins of the second world war* (2nd ed.). New York: Atheneum.

Thibaut, J. W., & Riecken, H. W. (1955). Some determinants and consequences of the perception of social causality. *Journal of Personality, 24,* 113–133.

Turnbull, W. (1986). Everyday explanation: The pragmatics of puzzle resolution. *Journal for the Theory of Social Behaviour, 16,* 141–160.

Turnbull, W., & Slugoski, B. R. (1988). Conversational and linguistic processes in causal attribution. In D. Hilton (Ed.), *Contemporary science and natural explanation: Commonsense conceptions of causality* (pp. 66–93). Brighton, England: Harvester Press.

Ward, W., & Jenkins, H. (1965). The display of information and the judgment of contingency. *Canadian Journal of Psychology, 19,* 231–241.

Watson, D. (1982). The actor and the observer: How are their perceptions of causality different? *Psychological Bulletin, 92,* 682–700.

Weiner, B. (1985). "Spontaneous" causal thinking. *Psychological Bulletin, 97,* 74–84.

Wells, G. L., & Harvey, J. H. (1977). Do people use consensus information in making causal attributions? *Journal of Personality and Social Psychology, 35,* 279–293.

White, M. (1965). *Foundations of historical knowledge.* New York: Harper & Row.

(*Appendix follows on next page*)

Appendix

Role of a Given Cause in the Context of Independent Alternative Causes

We derive the contrast for a potential cause according to our probabilistic contrast model in the cases in which independent alternative causes (a) could be present, (b) are known to be present, and (c) are known to be absent in the event to be explained. We compare these contrasts with the situation in which only a single cause is present in the target event.[11]

Let M denote the event in which potential cause m is present and $\bar{\mathrm{M}}$ denote the event in which m is absent for the situation in which there is a single cause. Let $\boldsymbol{m}$ denote the event in which m is present and $\bar{\boldsymbol{m}}$ denote the event in which m is absent for the situation in which alternative causes, $n_1, \ldots, n_k$, could be present. Assume that $P(\mathrm{M}) = P(\boldsymbol{m})$ and that the effect does not occur when no causes are present. Let e denote the presence of the effect, and $P_{n_i}(e)$ denote the probability of a single cause n_i producing e.

Because e does not occur if none of the causes present produces e,

$$P(e|\boldsymbol{m}) = 1 - P(\bar{e}|\boldsymbol{m})$$

$$= 1 - [1 - P(e|\mathrm{M})] \prod_{i=1}^{k} [1 - P_{n_i}(e)];$$

$$P(e|\bar{\boldsymbol{m}}) = 1 - P(\bar{e}|\bar{\boldsymbol{m}})$$

$$= 1 - \prod_{i=1}^{k} [1 - P_{n_i}(e)].$$

For the case in which the presence of n_i is unknown,

$$P_{n_i}(e) = P(n_i)P(e|n_i).$$

For the case in which n_i is known to be present,[12]

$$P_{n_i}(e) = P(e|n_i). \tag{A1}$$

For the case in which all n_i are known to be absent,

$$P_{n_i}(e) = 0. \tag{A2}$$

Probabilistic Contrast Model

For event M,

$$\Delta p_{\mathrm{M}} = P(e|\mathrm{M}) - P(e|\bar{\mathrm{M}}),$$

where $P(e|\bar{\mathrm{M}}) = 0$.

For event $\boldsymbol{m}$,

$$\Delta p_{\boldsymbol{m}} = P(e|\boldsymbol{m}) - P(e|\bar{\boldsymbol{m}})$$

$$= 1 - [1 - P(e|\mathrm{M})] \prod_{i=1}^{k} [1 - P_{n_i}(e)]$$

$$- \{1 - \prod_{i=1}^{k} [1 - P_{n_i}(e)]\}$$

$$= P(e|\mathrm{M}) \prod_{i=1}^{k} [1 - P_{n_i}(e)] = \Delta p_{\mathrm{M}} \prod_{i=1}^{k} [1 - P_{n_i}(e)].$$

Because $[1 - P_{n_i}(e)] < 1$ if n_i is a cause of e, $\Delta p_{\boldsymbol{m}} < \Delta p_{\mathrm{M}}$. That is, the probabilistic contrast for m is decreased by the addition of n_i, as stated by Kelley's discounting principle. The magnitude of the decrement from Δp_{M} to $\Delta p_{\boldsymbol{m}}$ is (a) proportional to both $p(e|n_i)$ and the prevalence of n_i when the presence of n_i is unknown and (b) proportional to $p(e|n_i)$ when n_i is known to be present. For the special case in which one or more alternative causes always produces e when it is present and it is known to be present whenever m is present (see Equation A1 and Footnote 12), $\prod_{i=1}^{k} [1 - P_{n_i}(e)] = 0$; therefore, $\Delta p_{\boldsymbol{m}} = 0$. For the case in which all n_i are known to be absent, $\Delta p_{\boldsymbol{m}} = \Delta p_{\mathrm{M}}$.

Suppes's Model

Similar predictions follow from Suppes's model. For event M,

$$\Delta \boldsymbol{P}_{\mathrm{M}} = P(e|\mathrm{M}) - P(e),$$

where

$$P(e) = P(e \text{ and } \mathrm{M}) + P(e \text{ and } \bar{\mathrm{M}}).$$

Therefore,

$$\Delta \boldsymbol{P}_{\mathrm{M}} = P(e|\mathrm{M}) - P(e|\mathrm{M})P(\mathrm{M}) - P(e|\bar{\mathrm{M}})P(\bar{\mathrm{M}})$$

$$= [1 - P(\mathrm{M})][P(e|\mathrm{M}) - P(e|\bar{\mathrm{M}})] = [1 - P(\mathrm{M})]\Delta p_{\mathrm{M}}.$$

Similarly, for event $\boldsymbol{m}$,

$$\Delta \boldsymbol{P}_{\boldsymbol{m}} = [1 - P(\boldsymbol{m})]\Delta p_{\boldsymbol{m}}$$

$$= [1 - P(\boldsymbol{m})]\Delta p_{\mathrm{M}} \prod_{i=1}^{k} [1 - P_{n_i}(e)] = \Delta \boldsymbol{P}_{\mathrm{M}} \prod_{i=1}^{k} [1 - P_{n_i}(e)].$$

Because $[1 - P_{n_i}(e)] < 1$ if n_i is a cause of e, $\Delta \boldsymbol{P}_{\boldsymbol{m}} < \Delta \boldsymbol{P}_{\boldsymbol{M}}$ if $P(\mathrm{M}) < 1$. That is, when m is not constantly present, the contrast for m is decreased by the addition of n_i. The magnitudes of the decrements from $\Delta \boldsymbol{P}_{\boldsymbol{M}}$ to $\Delta \boldsymbol{P}_{\boldsymbol{m}}$ in the cases in which (a) the presence of n_i is unknown, (b) n_i is known to be present, and (c) n_i is known to be absent parallel those for the probabilistic contrast model.

[11] We thank Thomas Wickens for his valuable comments on an earlier draft of our derivations. In particular, we thank him for generalizing our derivations to apply to an indefinite number of alternative causes.

[12] Equations A1 and A2, respectively, imply the restriction of the focal set to events in which n_i is present and events in which n_i is absent.

Received July 30, 1990
Revision received July 26, 1991
Accepted August 13, 1991 ■