BUCKLE: A MODEL OF CAUSAL LEARNING

by

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To my parents.

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CHAPTER I

INTRODUCTION

This paper presents a new model of causal induction called BUCKLE (Bidirectional

Unobserved Cause LEarning). Existing models of causal induction (e.g. Anderson &

Sheu, 1995; Busemeyer, 1991; Cheng, 1997; Cheng & Novick, 1990, 1992; Dickinson,

Shanks, & Evenden, 1984; Jenkins & Ward, 1965; Schustack & Sternberg, 1981; White,

2002) either ignore or make simplistic assumptions about unobserved causes. In contrast,

BUCKLE makes relatively sophisticated inferences about the occurrence of unobserved

causes in a given situation, which allow unobserved causes to be learned just like

observed causes. As a result, BUCKLE explains learning of not only unobserved but also

observed causes better than existing models of causal induction. Before presenting

BUCKLE, we illustrate why the role of unobserved alternative causes is critical to the

understanding of human causal learning.

The Importance and Difficulty of Understanding Alternative Causes

Current models of causal induction have traditionally assumed that the input

available to reasoners comes in the form of covariation; how the causes vary with their

effects. In the case of a single cause and effect, covariation can be summarized in a table

like the one in Figure 1. Thus, a learner observes whether presence or absence of a

causal candidate is followed by presence or absence of an effect, and translates these

observations into beliefs about causal relations. Much work has been dedicated to

Effect

	Present	Absent
Present	A	B
Absent	C	D

Cause

Figure 1 - A contingency table summarizing the covariation between two binary events. Each cell of the table represents one of the possible observations.

exploring how this translation is made (see Shanks, Holyoak, & Medin, 1996 for an extensive review).

Later work, however, has suggested that inferences about one cause may critically depend on how learners deal with other, alternative causes (e.g., Cheng, Park, Yarlas, & Holyoak, 1996). For example, Spellman (1996) had participants learn about two liquids (one red and one blue) and their influence on flowers blooming. When participants were asked about causal efficacy of the red liquid, their judgments were not simply based on how the red liquid and blooming covaried. Instead, participants systematically used observations in which the alternative cause (blue liquid) was held constant (a strategy referred to as conditionalizing), just as scientists control for potential confounding variables in experimental design (see also Goodie, Williams, & Crooks, 2003; Waldmann & Hagmeyer, 2001). Clearly, conditionalizing is advantageous because it prevents wrongly attributing causal efficacy to a candidate. For instance, upon observing that more men than women are scientists, one should conditionalize on differences in

socialization before concluding genetic differences as the cause (see also Simpson's paradox; Simpson, 1951).

Although conditionalizing allows learners to avoid mistaking illusory covariation as causation, it is often not feasible because it requires alternative to be observed. Alternative causes can be unobserved because they require special instruments or methods to be observed (e.g., genetic influences on cancer). More frequently, learners lack observations about alternative causes simply because they could not possibly consider all alternative causes of a particular event. Thus, lacking observations about alternative causes seems to be the rule rather than the exception.

Are Details about Unobserved Alternative Causes Necessary?

One elegant solution to this problem has been proposed in Power PC theory (Cheng, 1997; see also Pearl, 2000 for a similar approach). The power PC theory tempers traditional covariation (i.e., ΔP) by performing something analogous to conditionalizing over a composite of all alternative causes, a. The strength of a is unknown. However, Cheng (1997) shows that if we assume that a occurs independently of the target cause, c, that is, $P(c|a) = P(c|\sim a)$ (henceforth, no-confounding assumption), it is possible to equate the probability of the effect in the absence of a cause, $P(e|\sim c)$, with probability that a is present and causes the effect (see Cheng, 1997 for the proof). Thus, using $P(e|\sim c)$, which is computable from observable events, instead of the probability or strength of a, which are unobserved, these accounts avoid the need to observe alternative causes.

However, recent work has showed that people do not believe that the no-confounding assumption is required for causal inference. These studies demonstrated that people are willing to make causal judgments despite acknowledging violations of the no-confounding assumption (see Hagmeyer & Waldmann, 2004; Luhmann, 2005; and Experiment 6 in this paper). Then, how do people infer causation from covariation under confounded situations?

In contrast to the strategy taken by the Power PC theory, several models of causal induction assume that people learn the causal strength of unobserved causes just like observed causes (Rescorla & Wagner, 1972; Thagard, 2000). For example, the model proposed by Rescorla and Wagner (1972) assumes that there is an unobserved cause that is present on every observation and that this cause accrues associative strength just as observed causes do. The model that we introduce in this paper, BUCKLE, also assumes that causal learning involves sophisticated inferences about the probability and strength of unobserved causes.

In some sense, however, it seems counterintuitive that people would learn without direct observations. Thus, the first order of business is to determine whether people are willing to provide judgments of unobserved causes. After establishing people's willingness to make judgments about unobserved causes, we will present several accounts of such judgments, including BUCKLE.

CHAPTER II

EVALUATING UNOBSERVED CAUSES

In Experiment 1, participants observed the contingency between one target cause and one target effect only. They were then asked to judge the causal strength of the target cause as well as one alternative, unobserved cause. Participants were told that if they were unable to evaluate a cause, they should provide a response of "N/A" (i.e., not applicable). Experiment 1 also varied the number of unobserved causes to examine whether an increased number of unobserved, alternative causes would influence willingness to make causal judgments.

Experiment 1

Method

Participants

Twenty Vanderbilt University undergraduates participated for partial fulfillment of course credit.

Stimuli consisted of three electrical systems each consisting of a number of colored buttons and a light (see Figure 2 for an example). Each of the buttons used in the experiment was a different color to aid in their memory and to ensure that participants did not confuse the different systems. The three systems differed in the number of buttons (2, 3, or 4). In each system, the state (pressed or not pressed) of exactly one button was observable. The states of the remaining button(s) were hidden from view. This omission was denoted by a large question mark superimposed over the button(s) as shown in Figure 2. The state of the light (lit or not) was always observable.

The entire experiment was conducted on Apple iMacs using SuperLab. Participants first received overall instructions about the experiment. They were told that

White button.

Gray button was pressed.

Light turned on.
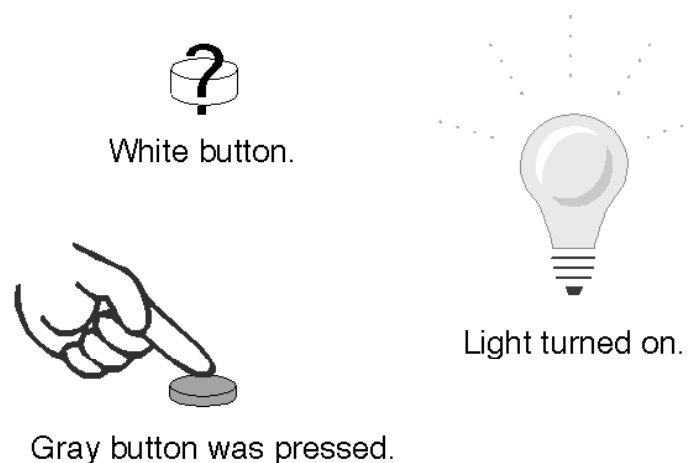
Figure 2 - A sample trial. The states of the grey button and the light are observed on every trial. The white button is unobserved; information about its state is unavailable on every trial.

they would be examining a series of electrical systems previously constructed by the experimenters. Participants were told that it was their job to discover how each system worked and that, to do so, they would view a series of tests that had been run on the systems. Each of these tests contained information about whether the observed button was pressed or not and whether or not the light had turned on. Each participant saw all three systems in a counterbalanced order.

When participants encountered each system they were first told about its constituent parts (e.g., "One red button, one blue button, and a light."). Participants were not told whether this was an exhaustive list of components. They were then told that the experimenters had run a set of tests on the system to discover how it worked. They were told that the data pertaining to some of the buttons had been lost so that information about only a single button and the light would be available on each trial. Participants were also told that they would be asked to evaluate the extent to which each of two buttons caused the light to turn on. Which buttons they were to evaluate was indicated in the instructions. One of the evaluated buttons was always the observed button and the other was one of the unobserved buttons.

After receiving these instructions, participants proceeded to view the set of tests (i.e. trials), presented in a randomized order. Trials were presented one at a time and each remained on the screen until the participant pressed the spacebar to continue. Participants were then asked to rate the causal strength of two buttons. Each button was evaluated separately and the observed cause was always evaluated first[1]. Participants

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[1] The fixed order of questions provides a strong test of people's willingness to judge unobserved causes because judging the observed cause should highlight the lack of information about the unobserved cause.

were asked to judge, "the extent to which pressing the [color] button caused the light to turn on." Participants responded on a scale from –100 ([color] button prevented the light from turning on) to 100 ([color] button caused the light to turn on), with zero labeled as, "[color] button had no influence on the light."

To estimate participants' willingness to respond to these questions, participants were instructed to respond with "N/A" when they felt they could not make a judgment. Below the rating scale was a reminder that, "If you cannot make a judgment, please write 'N/A'." The contingency (i.e., ΔP = P(effect|cause) – P(effect|no cause), Jenkins & Ward, 1965) between the observed cause and the effect was identical across all systems using the cell frequencies shown in Figure 3. The cell frequencies used resulted in a contingency of ΔP = 0.5.

Predictions of ΔP-based models

The contingency (i.e., ΔP) between the unobserved cause and effect cannot be compute and is not even tightly constrained even in the system with only a single unobserved button. Assuming that the observed and the unobserved buttons are the only two possible causes of the symptoms and that events always have causes (an additional assumption not conveyed in our instructions; Bullock, Gelman, &, Baillargeon, 1982), $\overline{O}E$ observations imply that the unobserved cause was present. On the remaining trials (i.e., $OE, O\overline{E}$, and $\overline{O}\overline{E}$), however, it is unclear whether the unobserved cause was present or absent, making it impossible to compute ΔP for the unobserved cause. More specifically, if the presence of *u* were to correlate perfectly with presence of *e*, then *u* would be present in all 7 cases of *OE* , as well as all 7 cases of $\overline{O}E$, but absent in all 7

Effect
                              Present Absent

                    Present   7   0
        Observed
          Cause
                    Absent   7   7

Figure 3 - A summary of the observations presented to
participants in Experiment 1. The contingency table summarizes
the covariation of the observed cause and the effect. No such
table can be fully constructed for an unobserved cause.

cases of $\overline{O}\overline{E}$. Thus, $\Delta P = P(e|u) - P(e|\sim u) = 1.0 - 0.0 = 1.0$ for u. If presence of u were

to negatively correlate with e, then u would be absent in all 7 cases of OE, but present in

all 7 cases of $\overline{O}E$ (again, assuming that something has to cause E), and present in all 7

cases of $\overline{O}\overline{E}$. Thus, $\Delta P = 0.5 - 1.0 = -.0.5$. Given that ΔP for the unobserved cause could

be anywhere between -0.5 and 1.0, if participants were simply computing ΔP, they should

have responded with "NA" for the strength estimate of the unobserved cause in all cases.

Results and Discussion

        The critical question in Experiment 1 was whether participants were willing to

evaluate causes they had not observed. First, despite the fact that no covariation

information was presented for the unobserved cause, all participants were willing to make

a causal strength judgment of both the observed and unobserved causes in the system

with only one unobserved cause (e.g., Figure 2). Thus, people are willing to evaluate a

cause with which they have no direct experience under at least some conditions. Participants apparently felt they had enough information to make a reasonable judgment.

It could be argued, however, that this result obtained because participants were generally unwilling to use the "N/A" response. This possibility motivates a second observation: participants were willing to use the "N/A" response when evaluating systems with a greater number of unobserved causes. Participants were significantly less likely to respond when there were 2 and 3 unobserved causes (55% and 35 % responding respectively) than when there was only one unobserved cause (100%), $\chi^2(1, N=20)=9$, p<.005 and $\chi^2 (1, N=20)=13$, p<.001, respectively using McNemar's test (McNemar, 1947). Thus, it was not until the stimuli included multiple unobserved causes that participants were unwilling to make causal judgments[2]. The fact that willingness was lower for situations with multiple unobserved causes suggests that participants may typically choose to only learn about a single unobserved cause. Such a representation is common in the modeling of causal learning (e.g., Rescorla & Wagner, 1972; Cheng, 1997; Griffiths & Tenenbaum, 2005) where the single unobserved cause represents the aggregate of all unobserved causes.

Having demonstrated that participants learn specific causal strength estimates of unobserved causes, the next question is how they do so. As illustrated earlier, even models that acknowledge the existence of unobserved causes (e.g., Cheng, 1997; Cheng & Novick, 1990, 1992) avoid making specific estimates of the causal strength of those unobserved causes. Similarly, models that do not acknowledge unobserved causes

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[2] Causal ratings, although difficult to be interpreted due to the large number of N/A responses in the 3 and 4 button conditions, also reflect increasing uncertainty as a function of increased number of unobserved causes (M = 70.25, 32.72 and 16.43, SD = 28.82, 32.89, 33.75 for the 2, 3 and 4 button conditions, respectively).

(White, 2002; Schustack & Sternberg, 1981; Jenkins & Ward, 1965) cannot make causal strength judgments about unobserved causes because covariation is unavailable. In what follows, we describe causal learning models that can handle unobserved cause learning. In Part 2, we present BUCKLE's account. In Part 3, we present alternative, existing accounts (Rescorla & Wagner, 1972; Thagard, 2000)[3].

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[3] Griffiths and Tenenbaum's (2005) causal support model will not be discussed in this paper because it concerns the learning of causal structure and not causal strength, which is the main question of this paper.

CHAPTER III

BUCKLE

To learn about causal relationships, BUCKLE uses two steps, each of which is performed during each observation. The first step is to replace the missing information about unobserved causes. BUCKLE computes the probability that the unobserved cause is present based on the available information and the known roles of causes and effects. Once this step is completed, the unobserved cause is treated just like an observed cause; it is present with some probability. The second step is to learn the strengths of each cause-effect relationship. This learning is accomplished via an error-correction algorithm.

The bi-directionality of these two steps (illustrated in Figure 4) is the essence of
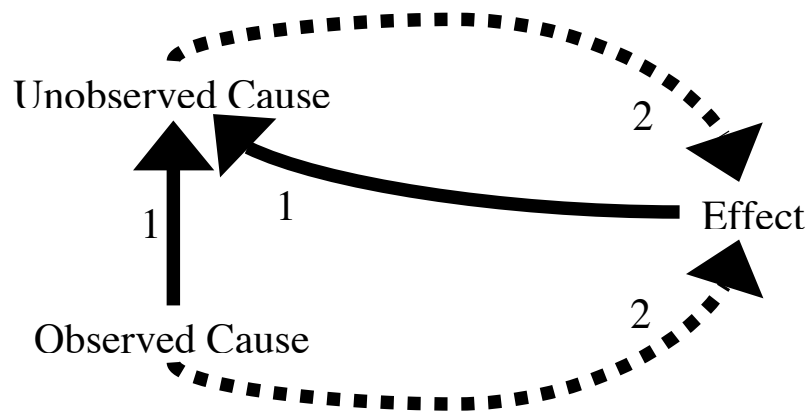
Figure 4 - A diagram illustrating the operation of BUCKLE's two steps. The solid arrows labeled with a "1" represent BUCKLE's first step: available information about the state of the observed cause and effect is used to predict the likelihood of the unobserved cause. The dashed arrows labeled with a "2" represent BUCKLE's second step: information about the two causes is used to predict the effect.

BUCKLE. Available information is first used to predict the presence of the unobserved cause (see arrows labeled "1" in Figure 4). This newly computed information, along with the information available about other causes, is then used to predict the presence of the effect (see arrows labeled "2" in Figure 4).

<div align="center">Formal Description of BUCKLE</div>

Because subsequent experiments use situations with a single observed cause, a single unobserved cause, and a single effect, the formal details of BUCKLE will be given for this simplified case[4]. The observed and the unobserved causes will be represented as o and u, respectively. The effect is represented by e. Conventionally, 1 represents presence and 0 represents absence. Thus, an observation in which the observed cause is present and the effect is absent is represented by $o = 1$ and $e = 0$ (for the sake of brevity, we will often use the conventional abbreviation in the text, e.g., $O\overline{E}$). The strengths of o and u will be represented by q_o and q_u respectively.

<div align="center">*Step 1: Inference of Unobserved Cause*</div>

The first step taken by BUCKLE is to infer how likely it is that the unobserved cause is present in a given trial. To do this, the values of o and e are first set according to the state of the observed cause and effect in the current observation (e.g., o=1, e=0).

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[4] Typical real world situations include multiple observed causes. People may acknowledge this fact, in which case BUCKLE could be modified to accommodate additional unobserved causes. On the other hand, people may lump all the unobserved alternative causes into a single composite causes (e.g., Cheng, 1997) in which case it would be more appropriate to use the version described here. Regardless, different causal situations are simply generalizations of what is described here.

Given the current strength of observed and unobserved causes and this input, the

probability that the unobserved cause is present may be computed using Bayes theorem:

$$P(u = 1 | o = O, e = E) = \frac{P(e = E | o = O, u = 1) \bullet P(u = 1 | o = O)}{\sum_{U=[0,1]} P(e = E | o = O, u = U) \bullet P(u = U | o = O)} \quad (1)$$

The denominator of Equation 1 is simply $P(o=O, e=E)$ computed by summing the

probabilities of each of the different ways in which $o=O$ and $e=E$. Exactly how

$P(e=E|o=O,u=U)$ is computed depends on the values of o, e, q_o, and q_u (see Appendix

A)[5]. For example, imagine that on a given trial, q_u and q_o are positive (i.e., o and u

produce rather than prevent their effects), the observed cause occurs (i.e., $o=1$), and the

effect does not (i.e., $e=0$). The denominator of Equation 1 is the probability that o was

present but failed to generate e, and either u was absent or u was present but failed to

generate e[6]. This probability is given by the expression:

$$P(o = 1, e = 0) = \{P(o) \bullet (1 - q_o) \bullet [1 - P(u | o = 1)]\} + \\ [P(o) \bullet (1 - q_o) \bullet P(u | o = 1) \bullet (1 - q_u)] \quad (2)$$

The quantities q_u and q_o are the causal strengths of o and u, which are stored and

updated after each observation as explained in the next section. Therefore, the causal

strengths used in Equation 2 would be those resulting from the previous trial. The prior

probability of o being present, $P(o)$, does not influence result of Equation 2 because the

value of o is observed on each trial (accordingly, each equation in Appendix A can be

simplified to eliminate $P(o)$, but they are presented as is for conceptual clarity). For

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[5] Computing this expression also depends on the assumed parameterization. See below
for more discussion of parameterization.
[6] These expressions depend critically on the assumed parameterization. See the section
on BUCKLE's second step and the General Discussion for more discussion of this issue.

$P(u|o)$, we will use a uniform distribution ($P(u|o=1) = P(u|o=0) = .5$) and that remains static.

When the observed cause occurs, and the effect does not, the numerator of Equation 1 is the probability that o was present but failed to generate e, and u was present and failed to generate e. This probability can be expressed as:

$$P(u=1, o=1, e=0) = P(o) \bullet (1-q_o) \bullet P(u|o=1) \bullet (1-q_u) \qquad (3)$$

Combining, Equations 2 and 3, P(u=1 | o=1, e=0) can be computed as:

$$P(u|o=1, e=0) = \frac{P(o) \bullet (1-q_o) \bullet P(u|o=1) \bullet (1-q_u)}{\{P(o) \bullet (1-q_o) \bullet [1-P(u|o=1)]\} + [P(o) \bullet (1-q_o) \bullet P(u|o=1) \bullet (1-q_u)]}$$
$$(4)$$

Recall this expression assumes that o and u produce rather than prevent their effects (i.e., q_u and q_o are positive). If one of the two causes were assumed to be preventative, BUCKLE would use different expression to compute P($u|o,e$). For example, consider the observation used in the above example (i.e., o=1, e=0), but this time, suppose that q_u is negative (i.e., u is preventative). Given these quantities, the observation of (o=1, e=0) could have occurred in three ways; (1) o was present but failed to produce e, and u was absent; (2) o was present but failed to produce e, and u was present; or (3) o was present and produced e, but u was present and prevented e. Thus, despite the fact that the observation (i.e., o and e) is the same as above, Equation 1 will be computed differently. Appendix A provides the computations for all 16 possible cases. Which of these 16 expressions is used on a given trial is completely determined by the current values of o and e (available from the input) and q_u and q_o (modified during learning).

The second step of BUCKLE is to use the available (observed and inferred) information to learn about the strength of each causal relationship. The algorithm BUCKLE uses to learn is adapted from a suggestion by Danks, et al. (2003; see the section titled Similarity between BUCKLE and Other Models of Learning in the General Discussion)[7]. This learning algorithm relies on error-correction to learn causal relationships. Information about the presence (i.e., o and e) is first used to predict how likely the effect is to be present given the current set of beliefs (i.e., q_o and q_o). This prediction is then compared with whether or not the effect actually occurred. The difference between the predicted and actual states of the effect (the error) forms the basis of learning.

For the remainder of this article, we will assume that causes combine their influence in the manner of a noisy-OR gate when causes are generative and in the manner of a noisy-AND-NOT gate when causes are preventative; assumptions that have received recent support (see Cheng, 1997; Novick & Cheng, 2004; Griffiths & Tenenbaum, 2005; Danks, Griffiths, & Tenenbaum, 2003; Steyvers, Tenenbaum, Wagenmakers, & Blum, 2003). For example, when both o and u are generative, the effect would be present when either cause produced the effect. Thus, the effect is predicted according to the following expression:

$$e_{predicted} = P(e) = (o \bullet q_o) + (u \bullet q_u) - \left[(o \bullet q_o) \bullet (u \bullet q_u)\right] \quad (5)$$

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[7] The algorithm we use is not the Bayesian strength estimator emphasized by Danks, et al. (2003). Danks, et al. acknowledge that this estimator is computationally intensive and its psychological plausibility is currently uncertain. Instead, the inspiration for BUCKLE's learning comes from what Danks, et al. (2003) refer to as the causal power analogue of the augmented Rescorla-Wagner model. BUCKLE can also be applied to situations in which all causes are observed; in such cases, BUCKLE reduces to the Danks model.

In this expression, o is equal to one when the observed cause is present and zero when absent, and u is the probability that the unobserved cause is present on this trial (i.e., $P(u|o,e)$). When u is preventative and o is generative (i.e. $q_u < 0$, $q_o > 0$), e is predicted according to the following:

$$e_{predicted} = P(e) = o \bullet q_o \bullet \left\{ \left[u \bullet \left(1 - (-q_u)\right) \right] + \left[1 - u \right] \right\} \quad (6)$$

When u is generative and o is preventative (i.e. $q_u > 0$, $q_o < 0$), e is predicted according to the following:

$$e_{predicted} = P(e) = u \bullet q_u \bullet \left\{ \left[o \bullet \left(1 - (-q_o)\right) \right] + \left[1 - o \right] \right\} \quad (7)$$

When neither cause is generative, $P(e)=0$. These are the equations that BUCKLE uses to make its predictions. The resulting quantity, $e_{predicted}$, is used as the predicted value of e. The difference between $e_{predicted}$ and the actual value of e is used to adjust causal strengths according to the following expressions:

$$q_{o(n)} = q_{o(n-1)} + \alpha_o \beta (e - e_{predicted}) \quad (8)$$

$$q_{u(n)} = q_{u(n-1)} + \alpha_u \beta (e - e_{predicted}) \quad (9)$$

The quantities $q_{o(n-1)}$ and $q_{o(n-1)}$ are the causal strengths resulting from the preceding trial. The strength of each cause is updated separately. The quantities α and β represent learning rates associated with causes and effects, respectively. A value of 0.5 is used for β. When the observed cause is present, $\alpha_o = \alpha_{o\text{-present}}$ where $\alpha_{o\text{-present}}$ will be treated as a free parameter and allowed to vary between zero and one. When the observed cause is absent, $\alpha_o = \alpha_{absent} = 0.0$. For the unobserved cause, Equation 10 is used to compute a value of α to take into account the fact that the unobserved cause is only present with some probability.

$$\alpha_u = \left[P(u \mid o,e) \bullet (\alpha_{u-present} - \alpha_{absent}) \right] + \alpha_{absent} \quad (10)$$

This equation results in α_u=0 when P(u|,o,e)=0, and α_u=$\alpha_{u\text{-}present}$ when P(u|,o,e)=1, just as

for the observed cause. For values of P(u|,o,e) between 0 and 1, α increases linearly and

in proportion to the value of P(u|,o,e). The variable $\alpha_{u\text{-}present}$ will be treated as a second

free parameter and allowed to vary between zero and one.

To review, BUCKLE completes two steps for each observation. BUCKLE first

infers how likely the unobserved cause is to be present and then learns the causal

strengths of all causes. Beyond these two steps, the particular algorithms behind each

step of BUCKLE's operation are interchangeable (see the section entitled The

Interchangeable Nature of BUCKLE in the General Discussion for more discussion on

this point).

Simulation of BUCKLE

In this section, we illustrate BUCKLE's behavior using a series of simulations.

The first order of business is to ensure that BUCKLE can replicate people's judgments of

observed causes in a traditional causal learning paradigm. After doing so, we begin

evaluating BUCKLE's learning of unobserved causes.

Observed Cause Learning

To illustrate BUCKLE's ability to account learning in a traditional paradigm, we

simulate the results from Experiment 3 of Buehner, Clifford, & Cheng (2003). This

experiment was chosen for three reasons. First, BUCKLE computes the sufficiency of a

cause (i.e., the probability that an effect would occur given that a cause is present), and

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Buehner et al. (2003) is one of the few existing studies that judiciously asked participants to judge the sufficiency of a cause (see Buehner, Clifford, & Cheng, 2003 for a discussion about causal questions). Second, BUCKLE, in its current form, can only learn from trial-by-trial presentation (as opposed to a summary format where the contingency information is conveyed all at once) because it updates its beliefs as each trial is presented. Buehner et al.'s (2003) Experiment 3 utilized this presentation format. Third, this dataset provides a range of findings and allows us to evaluate BUCKLE's generality. In an attempt to provide a thorough test of the Power PC theory (Cheng, 1997), Buehner, et al. designed ten different conditions, each of which contained a different set of covariation information. These conditions implied causal strengths (according to the Power PC theory) ranging from –1 to 1 and included a range of generative, preventative, and non-contingent conditions.

In Buehner et al.'s Experiment 3, participants received each of the 10 conditions separately with the observations (24 of them per condition) presented in a random order. On each trial, information about whether or not a new patient had taken medication was presented on the computer screen. After one second, information about whether or not the patient had a headache was presented alongside the cause information. After all the trials for a condition were presented, participants were asked to judge the strength of the medication-headache relationship.

To determine whether BUCKLE is able to capture the basic features of people's learning, we ran BUCKLE in each of the ten conditions from Experiment 3 of Buehner, et al. (2003). Since BUCKLE is sensitive to trial order (see Experiment 7) it is important to simulate the experiment using the identical presentation order. However, Buehner, et

al. (2003) used randomized orders for each participant and did not report these orders. For this reason, we created 1000 simulated participants (i.e., 1000 new presentation orders). For each simulated participant, the order of the observations in each condition was randomized (just as in Buehner, et al., 2003). Using the directed search algorithm described by Hooke and Jeeves (1960), BUCKLE's $\alpha_{o\text{-}present}$ and $\alpha_{u\text{-}present}$ were fit to the mean causal judgments reported by Buehner, et al. This was done for each simulated participant. All other parameters were set as described in Table A1. The final values of the strength parameter q_o (multiplied by 100 to match the scale used by participants) were taken as the judged strength of the cause. (In all subsequent simulations of BUCKLE, the methods described here will be used unless noted otherwise.)

To assess BUCKLE's fit, we computed both R^2 and the root-mean-squared deviation (RMSD = SQRT(SSE/(N)), where N is the number of observations modeled, or conditions in this case, 10; Shunn & Wallach, 2002). BUCKLE accounted for 98% of the variance in participants' judgments and resulted in an RMSD of 14.45. This fit appears to be as good as the Power PC theory itself (R^2=.97 and RMSD=24.00) and better than ΔP (R^2=.87 and RMSD=18.62). Of course, because these models differ in the number of free parameters (Power PC and ΔP are parameter-free), it is difficult to assess relative goodness of fit. (Experiment 6 also addresses the Power PC theory's predictions about observed cause learning.) The point of the current simulation, although not diagnostic in distinguishing between these models, is that BUCKLE is able to account for a significant portion of people's behavior in traditional causal learning situations (i.e., those that do not involve obvious unobserved causes).

Unobserved Cause Learning

In this section, we illustrate BUCKLE's unobserved cause learning using a series of simple simulations. In particular, we illustrate that BUCKLE predicts the strength of an unobserved cause depends on (1) the probability that u is present on a given trial (i.e., P(u|,o,e)), (2) the information observed on the given trial (i.e., values of o and e), and (3) the current estimates of the unobserved and the observed cause's strengths (i.e., q_u and q_o). In all of the simulations reported in this section, $\alpha_{u\text{-}present}$, and $\alpha_{o\text{-}present}$ are fixed to be 0.5 because there are no empirical data to fit.

Effect of P(u|o,e) on q_u

BUCKLE predicts that the probability of u being present on a given trial should influence how q_u changes on that trial. For instance, suppose a learner observes *OE*. If u were likely to be present on this trial, then the presence of the effect would be more likely to be attributed to u than if u were likely to be absent on that trial. BUCKLE makes this prediction because α_u is modulated in proportion to the magnitude of P(u|o,e) (see Equation 10).

To illustrate, we presented BUCKLE with a single observation to see how it would affect q_u. Prior to the observation, q_o and q_u were set to 0.5. Figure 5 illustrates the values of q_u that result from the exposure to the single observation of each of four types of events (i.e., *OE*, etc). As can be easily seen in Figure 5, the learning that occurs with respect to q_u is highly dependent on the probability of the unobserved cause occurring (i.e., P(u|o,e)). More specifically, when P(u|o,e) = 0, α_u is zero as explained earlier and thus, q_u does not change (see Equation 9). When P(u|o,e) = 1, q_u changes and does so differently depending on the type of observation. For instance, consider

observing $O\overline{E}$. Because both u and o are moderately generative causes in this

simulation, BUCKLE predicts that e is likely to be present (see Equation 5). However,

because e was absent, q_u decreases (see Equation 9).

Effects of values of o and e on q_u

The above simulation suggests that different kinds of experience have different

kinds of influence on q_u. In this section, we examine this phenomenon more closely by

presenting multiple observations of the same type and examining how the values of q_o, q_u,

and P($u|o,e$) change during learning. The results of four simulations are presented in

Figure 6. In each of these simulations, the model is presented with a single type of

observation (e.g., OE) 15 times. Figure 6 illustrates BUCKLE's predictions about q_o, q_u,

as well as P($u|o,e$) after each trial in each learning situation.

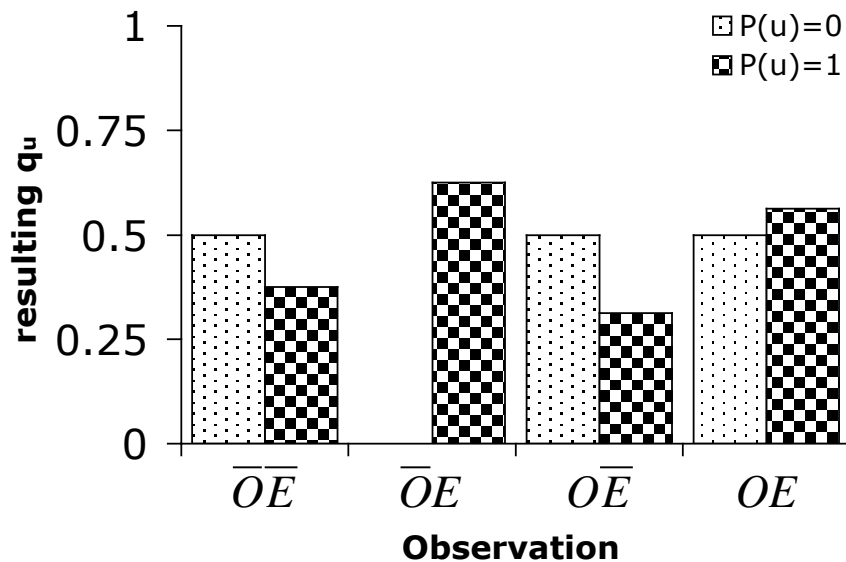Figure 5 - A diagram illustrating the operation of BUCKLE's two steps. The
solid arrows labeled with a "1" represent BUCKLE's first step: available
information about the state of the observed cause and effect is used to predict the
likelihood of the unobserved cause. The dashed arrows labeled with a "2"
represent BUCKLE's second step: information about the two causes is used to
predict the effect.

First consider the case in which 15 observations of *OE* were encountered (first panel in Figure 6). Because *o* and *e* are co-occurring, q_o increases over the course of learning. BUCKLE also increases q_u as well, though much less than q_o. This suggests that reasoners would not rule out the possibility that an alternative cause might be causing the observed effect. In addition, P($u|o,e$) remains relatively unchanged throughout learning. These changes appear psychologically plausible. According to BUCKLE, the changes in q_u take place for the following reason. At the beginning of learning, $\alpha_u < \alpha_o$ because P($u|o,e$) = .5 and o = 1 (see Equation 10). Because of this, q_o increases more rapidly than q_u (see Equations 8 and 9). Because q_u increases to moderate levels, P($u|o,e$) remains moderate. Note that, though the perceived strength of the two causes increase at different rates, BUCKLE does not predict extreme competition between the two causes (such as in the constraint-satisfaction model; see PART 3). Once the strength of the observed cause reaches near-maximal levels, the strength of the unobserved cause does not decrease, the rate of increase simply slows.

Second, consider the case in which 15 observations of $\overline{O}\overline{E}$ were made (second panel in Figure 6). As can be seen, this type of observation is relatively uninformative. All three quantities (i.e., q_u, q_o, and P($u|o,e$)) remain unchanged from their initial values. This prediction mirrors the finding that such observations generally have little influence on causal strength judgments (Schustack & Sternberg, 1981). BUCKLE predicts this for two reasons. First, because the observed cause is constantly absent, α_o is zero, and thus q_o will not change. Second, because both q_o and q_u are zero at the beginning of learning, the effect is always predicted to be absent. This prediction is always met, and thus q_u will not change (see Equation 9).
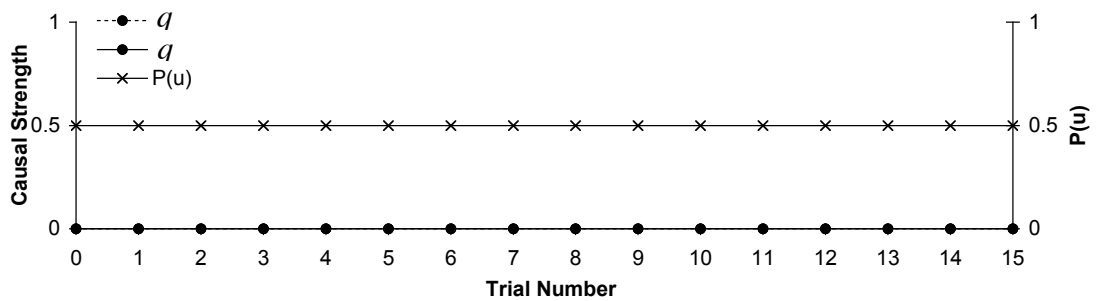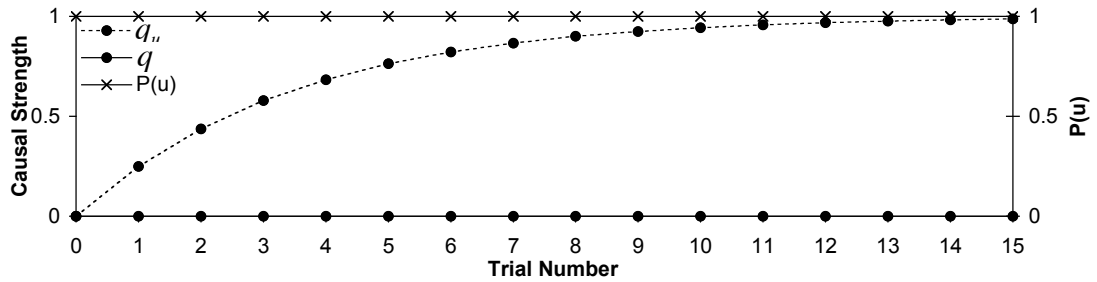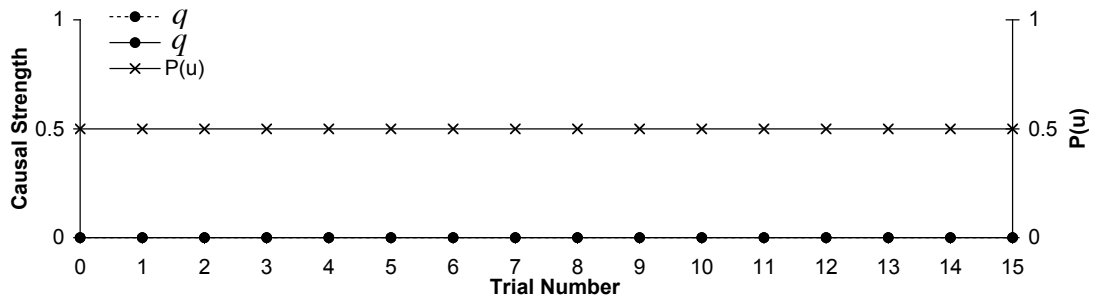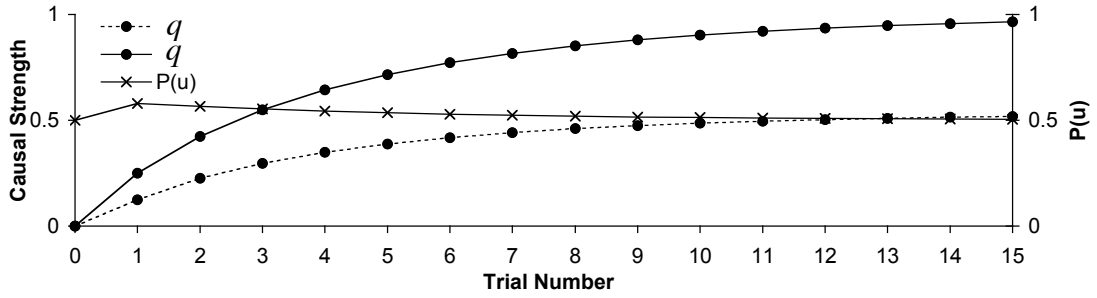
Figure 6 - BUCKLE's predictions about how q_u, q_o, and P(u|,o,e) will change over time in four situations. In each case, a single type of observation (e.g., $\overline{OE}$) was presented for 15 trials.

Third, consider the case in which 15 observations of $\overline{O}E$ were made (third panel in Figure 6). In this case, q_o remains 0, but q_u increases. This is because $\overline{O}E$ necessarily implicates the operation of an unobserved cause (Luhmann & Ahn, 2003). Because the observed cause is absent, it could not have generated the effect. The only way in which an $\overline{O}E$ observation could have occurred is if an unobserved cause was present and generated the effect. This is reflected in the probability computation found in Appendix A. That is, assuming that $q_u >= 0$ (i.e., assuming u is not preventative), when $o=0$ and $e=1$, the probability of u is always 1. The certainty with which the unobserved cause is present allows α_u to reach maximal levels (see Equation 10) and thus leads to large changes in q_u (see Equation 9); the perceived strength of the unobserved cause increases significantly. Because of the special status of $\overline{O}E$ observations, we will often refer to them by the more meaningful label: unexplained effects (Luhmann & Ahn, 2003).

The significant influence of unexplained effects predicted by BUCKLE is in line with several previous empirical demonstrations. Luhmann and Ahn (2003) and Hagmayer and Waldmann (2004) have already demonstrated the influence of unexplained effects on inferences about unobserved causes (Luhmann and Ahn's (2003) experiments are reported in detail later in this paper). For instance, Hagmayer and Waldmann (2004) varied $P(E \mid \overline{O})$ from zero to .67, and asked participants to estimate the causal strength of both the observed and unobserved causes. They found that when $P(E \mid \overline{O})$ was high, the unobserved cause was perceived as strong, and when $P(E \mid \overline{O})$ was low, the unobserved cause was perceived as weak.

Finally, consider the case in which 15 observations of $O\overline{E}$ are made (bottom panel in Figure 6). One might expect that $O\overline{E}$ observations would have a similar effect

on the perceived causal strength of unobserved causes as $\overline{OE}$; these observations could suggest that an unobserved cause prevented the effect from occurring. If this were the case, then the unobserved cause should be perceived as preventative (i.e., $q_u<0$). However, as can be seen in Figure 6, this is not the case. Instead, BUCKLE predicts that $O\overline{E}$ observations have relatively little influence; all three quantities (i.e., q_u, q_o, and P(u|,o,e)) again remain unchanged over the course of learning. This is because $O\overline{E}$ observations may occur either when the unobserved cause prevents the effect or when the observed cause is insufficient to bring about the cause (see Equation A11 in Appendix A). The former interpretation suggests that the unobserved cause is preventative whereas the latter interpretation allows the unobserved cause to be generative. In the current simulation, because q_o is zero, the former interpretation is more likely. According to BUCKLE, if the relative probability of the two interpretations were reversed, then $O\overline{E}$ observations would lead the unobserved cause to be perceived as preventative.

BUCKLE's prediction about the influence of $O\overline{E}$ can be contrasted with the work of Schulz, Sommerville, and Gopnik (2005). In characterizing the causal learning of preschoolers, these authors suggest that $O\overline{E}$ observations necessarily suggest the influence of (preventative) unobserved causes. Indeed, when $O\overline{E}$ observations were encountered in a situation containing an unobserved cause (their Experiment 1), children who were asked to prevent the effect preferred to utilize the unobserved cause. Schulz et al. (2005) suggest that the influence of $O\overline{E}$ results from preschoolers' strict belief that effects necessarily follow their causes. Holding such a belief would be analogous to modifying BUCKLE's probability computations such that the observed cause could not have failed to bring about its effect. Once modified, the ambiguity of $O\overline{E}$ observations is

eliminated and $O\overline{E}$ observations would lead learners to perceive the unobserved cause as preventative. It is an empirical question whether adults would also hold such strict beliefs about causal relations (see current Experiments 2-5 and 7).

Effects of q_u and q_o on P(u|o,e)

The final set of simulations illustrates how varying beliefs about the causal strengths in BUCKLE (i.e., q_u and q_o) can influence the probability of u (though to a smaller degree than the values of o and e). For example, when encountering an *OE* observation and q_u is believed to be positive, BUCKLE predicts that u is more likely to be present when q_u is high than when q_u is low (see the left panel of Figure 7). In other words, BUCKLE makes an intuitive prediction that when an effect occurs, a strong unobserved generative cause is more likely to have been present than a weak unobserved
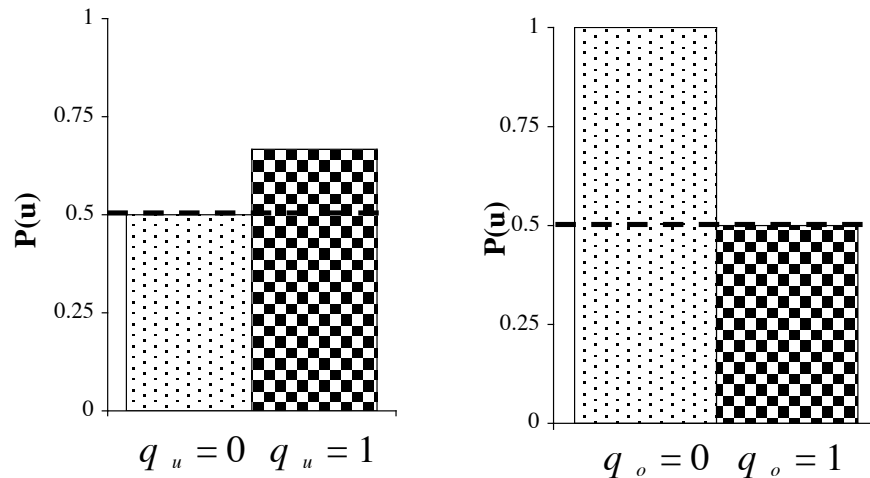
Figure 7 - Illustration of how BUCKLE predicts that beliefs about causal strength influence inferences about P(*u*|*o*,*e*) during an *OE* observation. The graph on the left illustrates how q_u influences P(*u*|*o*,*e*) (assuming q_o = .5) The graph on the right illustrates how q_o influences P(*u*|*o*,*e*) (assuming q_u = .5).

cause is.

Conversely, BUCKLE predicts that when encountering an *OE* observation, u is less likely to be present when an observed, generative cause's strength (q_o) is high than when it is low (see the right panel of Figure 7). In other words, if one believes that an observed cause is a strong causal candidate, one is less likely to postulate presence of an unobserved cause than if the observed cause is a weak causal candidate. Both of these predictions can be derived from Equation A4 in Appendix A.

Summary

BUCKLE learns using two steps. BUCKLE first replaces the missing data using an assortment of available information. In the second step, BUCKLE learns the causal relations assuming the unobserved cause is present with some probability. Using a variety of simple situations, we have illustrated that these steps provide a set of intuitive predictions.

CHAPTER IV

ALTERNATIVE MODELS

We consider two other models that include learning about unobserved causes. First, we consider constraint-satisfaction models (e.g., Thagard, 2000). These models suggest that the strength of unobserved causes is inversely related to the strength of an observed cause, akin to the well-known discounting principle (Kelley, 1967; Morris & Larrick, 1995). Second, we consider the associative models proposed by Rescorla and Wagner (1972). This model assumes that unobserved cause is constantly present and learns about it just as it learns about observed causes. In what follows, we describe each of these models in more detail and present experiments to compare them.

Constraint-satisfaction Networks

Constraint-satisfaction refers to the process of finding a set of states that satisfies a set of constraints or criteria and has been studied extensively in the field of artificial intelligence. In psychology, the states often refer to beliefs and there is evidence that people engage in constraint-satisfaction both to solve problems (e.g., McClelland & Rumelhart, 1981) and to remain internally consistent, or coherent (e.g., Holyoak & Simon, 1999).

In our discussion, we will use one particular variant of constrain-satisfaction networks: the Interactive Activation and Competition (IAC) model (McClelland & Rumelhart, 1981). This model has been used to explain a wide variety of psychological

findings (Thagard, 2000 for an overview) including aspects of causal learning and inference (e.g., Read & Marcus-Newhall, 1993; Hagmeyer & Waldmann, 2002). As we will see, these models also provide an intuitive prediction about how people learn about unobserved causes.

IAC models are networks in which each node corresponds to either an observation or explanation. Each node has an activation level that represents the degree to which that observation or explanation is believed. Positive activation represents belief and negative activation represents disbelief. Nodes are connected with bi-directional links so that directly connected nodes have mutual influence on each other. Observation nodes are connected to explanation nodes with links that either have a positive (consistent) or negative (inconsistent) weight. Explanations nodes are connected to each other with negatively weighted links so that explanations are inhibited by active alternative explanations (Morris & Larrick, 1995; Thagard, 2000; Baker, Mercier, Vallée-Tourangeau, Frank, & Pan, 1993; Price & Yates, 1993). In addition, observation nodes are connected to a special node whose activation is always maximal (i.e., 1). This allows observations (more so than explanations which must be inferred) to be accepted relatively easily (though it is possible for them to be rejected).

To initiate learning, each node is assigned a small, random amount of activation. The input to each node, i, is based on the activation of all directly linked nodes, a_j, and the weight of the intervening links, w_{ij}.

$$input_i = \sum_j w_{ij} * a_j \quad (11)$$

This input is then fed into an activation function that determines how the inputs affect each node's activation[8].

$$a_i(t+1) = a_i(t) \bullet (1-d) + \begin{array}{l} input_i \bullet \left[1 - a_i(t)\right] \quad \text{if } input_i > 0 \\ input_i \bullet \left[a_i(t) - (-1)\right] \quad \text{otherwise} \end{array} \qquad (12)$$

The activation of all nodes is updated iteratively and in parallel until the activation settles. At that point, the activation of explanation nodes is taken as a measure of the degree to which the propositions should be accepted or rejected.

The IAC model used for the remainder of the current study includes two explanation nodes. One of these nodes represents the proposition that the observed cause is causally responsible for the effect. The other node represents the proposition that the unobserved cause is causally responsible for the effect. There are also four observation nodes (one for each possible combination of observed cause presence/absence and effect presence/absence). Observations in which a cause and the effect are in the same state (i.e., both present or both absent) act as evidence in support of an intervening causal relationship. Thus, nodes representing such observations are connected to the observed cause explanation node with positively weighted links. On the other hand, observations in which the observed cause and the effect are in different states (i.e., one present and one absent) should act as evidence against an intervening causal relationship. Thus, the nodes representing such observations are connected to the observed cause explanation node with negatively weighted links.

Learning about unobserved causes occurs because hypothesis nodes receive input even when they represent hypotheses about unobserved causes. That is, though the

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[8] The parameter d represents decay and, following previous applications of IAC (e.g., Thagard, 2000), will be set to .05 for all subsequent simulations.

unobserved cause explanation receives no input from the observations (because the information about unobserved cause is unavailable in the input), it does receive input from the observed cause explanation via the negatively weighted link between them. The unobserved cause node is thus active to the extent that the competing explanation is inactive. In turn, a strong unobserved cause will tend to decrease the perceived strength of the observed cause (similar to the discounting principle). In sum, the constraint-satisfaction model generally suggests that the perceived strength of an unobserved cause will be related inversely to the perceived strength of the observed cause.

The Rescorla-Wagner Model

The learning model described by Rescorla and Wagner (1972; RW hereafter) assigns each cause and effect a node in a simple network. The inputs to the network represent events that are encountered first (often causes) and the outputs of the network represent those events that follow (often effects). Each input node is then connected to each output node. The strength of each cause is represented by the weight of the connection between its node and the effect node. Causal learning in this model amounts to adjustments of the connection weights as follows.

$$\Delta V_n = \alpha\beta(\lambda - \Sigma V_{n-1}) \quad (13)$$

In this equation, λ is an indicator of whether the effect is present or absent. When the outcome is absent, λ is 0. When the effect is present, λ is positive and its specific magnitude depends on the strength of the effect (a value of 1 is typical and will be used in all subsequent simulations). The parenthetical quantity is the amount of error on the nth trial; the difference between the summed strength of the present causes (ΣV_{n-1}; the

predicted value of the effect) and the observed effect (λ). The saliency of the cause is represented by α and the saliency of the effect is represented by β.

Thus, according to RW, learning is accomplished via an error-correction algorithm. The summed causal strength of the present causes acts as a prediction about the presence of the effect. This prediction is then compared to the actual observation of the effect. The difference between the prediction and the actual observation is then used to modify the network weights (i.e. causal strengths). Over time, this algorithm will tend to minimize the error between the prediction and the observation.

Of particular interest is the fact that RW always learns about an unobserved cause. Like BUCKLE, RW adds an extra cause node into its network. Unlike BUCKLE, this cause is assumed to be present on all trials. This unobserved cause is often interpreted as representing the experimental context or background, but could also be thought of as a composite of all unobserved causes (e.g., Cheng, 1997). For example, Shanks (1989) states that, "occurrences of the [effect] in the absence of the target cause … must be attributed to the background" (p. 27)." Like BUCKLE, RW learns about this unobserved cause just as it learns about observed causes. Thus, the critical difference between BUCKLE and RW is that, whereas RW makes a simple assumption about the probability of the unobserved cause, BUCKLE attempts to make somewhat more sophisticated, dynamic inferences about the probability of the unobserved cause.

CHAPTER V

EMPIRICAL TESTS OF THE MODELS

In what follows, we test these models in 7 experiments. In Experiment 2 the

constraint-satisfaction model is compared with BUCKLE. Recall that the constraint-

satisfaction model predicts an antagonistic relationship between competing explanations

(e.g., causes) whereas BUCKLE does not. Experiment 3 replicates the qualitative

predictions of BUCKLE using more ecologically valid methods. Experiments 4-6

compare RW and BUCKLE. As explained earlier, a critical difference between the two

models is that RW learns about a constantly present unobserved cause, whereas

BUCKLE inferences about the probability of the unobserved cause being present change

over time. In addition, the Power PC theory will be discussed in the context of

Experiment 5. Finally, Experiments 7 and 8 test additional predictions derived from

BUCKLE and attempt to gain a richer understanding of people's causal learning.

Experiment 2

To test the constraint-satisfaction model, we conducted a causal learning

experiment that varied the statistical relationship between the observed cause and the

effect. The details of simulation results of the two models for this experiment will be

provided later. In this section we simply provide conceptual explanations of the models'

predictions in order to motivate the design of the experiment. The experiment consisted

of four conditions illustrated in Table 1. All four conditions include OE and $\overline{OE}$

Table 1 - The design used in Experiment 2. Each condition contains OE and $\overline{O}\overline{E}$ observations. Only the presentation of $\overline{O}E$ and $O\overline{E}$ observations differs as shown in bold.

Condition	Unncessary	Zero	Perfect	Insufficient
Contigency Structure	7 0 **7** 7	7 **7** **7** 7	7 0 0 7	7 **7** 0 7
BUCKLE's Predictions				
Observed Cause	Moderately positive	Low (but positive)	High	Moderately positive
Unobserved Cause	High	High	Low (but positive)	Low (but positive)
Constraint-satisfaction Predictions				
Observed Cause	High	Zero	High	High
Unobserved Cause	Low (negative)	Zero	Low (negative)	Low (negative)
MLE				
Observed Cause	100	0	100	50
Unobserved Cause	100	100	0	0

observations. What varies across these conditions is whether or not $\overline{O}E$ and $O\overline{E}$

observations are included.

The Zero condition contains both $\overline{O}E$ and $O\overline{E}$ observations and results in

correlation of zero between the observed cause and the effect ($\Delta P=0$). The Perfect

condition contains neither $\overline{O}E$ nor $O\overline{E}$ observations and results in a perfect correlation

between the observed cause and the effect ($\Delta P=1$). The remaining two conditions each

constitute moderately strong relationships ($\Delta P=0.5$). The Unnecessary condition includes

$\overline{O}E$ observations (i.e., unexplained effects), which render the observed cause partially

unnecessary though completely sufficient (the effect is never absent when the observed

cause is present). The Insufficient condition includes $O\overline{E}$ observations, which render the

observed cause partially insufficient though completely necessary (the effect never occurs in the absence of the observed cause).

Table 1 also contains the predictions of the various models. According to the constraint-satisfaction model, the unobserved cause should be strongest when the observed cause is weakest (the Perfect condition). In all other cases, the observed cause is positively correlated with the effect and thus the unobserved cause should be perceived as negative. BUCKLE predicts that unexplained effects (i.e., $\overline{O}E$ observations) will most significantly influence unobserved cause judgments. This influence leads BUCKLE to predict that the unobserved cause should be perceived as stronger in the two conditions that include unexplained effects (Unnecessary and Zero) than in the two conditions that do not (Perfect and Insufficient). Thus, the Unnecessary condition provides the most critical test comparing BUCKLE and the constraint-satisfaction model with respect the predictions on the unobserved cause.

Those looking for the "correct" causal strengths in these four conditions can look to the Maximum Likelihood Estimates (MLE) listed in Table 1. These estimates are the most likely values for q_o and q_u given an (arguably) reasonable set of assumptions (see Appendix B for details on how we compute the MLE).

We used two different dependent variables across Experiments 2A and 2B because different models measure different quantities. BUCKLE estimates causal sufficiency: the degree to which a cause is sufficient to bring about its effect. The constraint-satisfaction model computes less specific quantities[9]. Thus, to test BUCKLE,

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[9] The constraint-satisfaction model, like RW, measures association (i.e., the degree to which the cause and effect co-occur). Association, like a regression weight, does not

Experiment 2A uses a query that specifically taps the notion of causal sufficiency (see Buehner, et al., 2003). Experiment 2B uses the traditional, but ambiguous method of eliciting causal judgments (e.g. To what extent does X cause Y?) typically used when evaluating constraint-satisfaction models (e.g. Hagmeyer & Waldmann, 2002).

Additionally, participants in Experiments 2A and 2B were told that nothing, other than the two buttons, could influence the light. This was done to equate participants' assumptions about the situation with the assumptions used in the modeling reported later. Experiment 3 will assess people's more natural assumptions about unobserved causes by not telling participants about the existence of an unobserved cause until after learning was completed.

Method

Participants. Fifty-four Vanderbilt University undergraduates (24 in Experiment 2A, 30 in Experiment 2B) participated for partial fulfillment of course credit.

Materials and design. Stimuli consisted of four electrical systems similar to those used in Experiment 1 (see Figure 2 for an example). Each of the buttons used in the experiment was a different color to aid in their memory and to ensure that subjects did not confuse different systems. Each system contained exactly one button whose state (pressed or not) was observable, one button whose state was unobservable and a single light. The unavailable state of the unobserved button was denoted via a large question mark superimposed over the button. The state of the light (on or off) was always observable.

---

distinguish between sufficiency and necessity. Association is simply a holistic measure of the strength of a relationship.

Both Experiments 2A and 2B used a 2 X 2 factorial design by crossing the inclusion of $\overline{O}E$ observations with the inclusion of $O\overline{E}$ observations. Table 1 summarizes the actual cell frequencies for each condition and the contingency between the observed cause and the observed effect.

Procedure. The procedure was same as in Experiment 1 except for the following changes. Each participant saw all four systems in a counterbalanced order. The trials within each system were presented in a quasi-randomized order. The set of trials was divided into blocks such that two of each trial type was presented within each block. The order of trials within these blocks was randomized. This was done to ensure that the different types of trials were evenly distributed throughout each participant's experience, given the previous studies showing the effect of presentation order in causal induction (e.g., Lopez, Shanks, Almaraz, & Fernandez, 1996; Dennis & Ahn, 2001).

After viewing the entire set of trials, participants were asked to rate the causal strength of the observed and unobserved button separately. In Experiment 2A, participants were told to, "Imagine running 100 new tests in which the [color] button was pressed and the [color] button was not. On how many of these tests do you expect the light to turn on?" Participants responded with a number between 0 and 100. In Experiment 2B, participants were asked to, "judge the extent to which pressing the [color] button caused the light to turn on." Responses could range from –100 ("[color] button prevented the light from turning on") to 100 ("[color] button caused the light to turn on") with zero label as, "[color] button had no influence on the light turning on."

Unlike Experiment 1, participants were not allowed to respond with "N/A." This was done for two reasons. First, the willingness to estimate unobserved causes was not

the main concern of Experiment 2. Second, although no participant in Experiment 1 gave "N/A" responses for systems involving an unobserved variable (such as those used in the current experiment), we wished to maximize the number of numerical responses because of our primary interest in comparing causal strength estimates across the four conditions.

In addition to the causal strength ratings of the two buttons, participants were also asked to rate how confident they were in each of their causal judgments. This task was added in an attempt to disentangle participants' causal beliefs from confidence in those beliefs. Doing so also allows us to examine whether participants, although willing to estimate causal strengths of an unobserved cause, feel as confident about these judgments as with an observed cause. These confidence ratings were made on a 7-point scale ranging from 1 ("Not at all confident") to 7 ("Very confident"). A visual representation of the scale, indicating the endpoints and their labels, was present for participants' reference while making their judgments.

Results and Discussion

Experiment 2A

Participants' causal judgments can be seen in Figure 8. As predicted by BUCKLE, the presence of unexplained effects (i.e., $\overline{OE}$) is particularly influential in driving judgments of the unobserved cause. A 2 ($\overline{OE}$ present / absent) X 2 ($O\overline{E}$ present / absent) repeated measures ANOVA on causal judgments of unobserved causes revealed a significant main effect of unexplained effects ($\overline{OE}$ observations), $F(1, 23) = 43.19$, $p <$ .0001, because participants gave much higher ratings on conditions with unexplained effects (M = 73.35, SD = 30.27) than on conditions without unexplained effects (M =
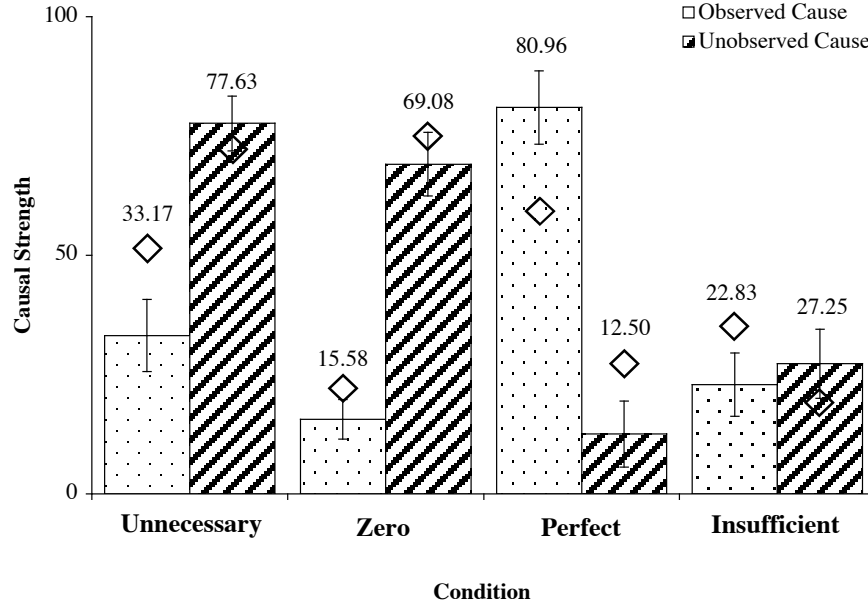
Figure 8 - Causal strength judgments from Experiment 2A. Error bars indicate standard error. The diamonds represent BUCKLE's strength estimates.

19.88, SD = 35.11). No other significant effects were observed. Note that participants'

judgments generally conformed to the MLE (R^2 = .67) except when judging the observed

cause in the Unnecessary condition where judgments were substantially lower.

Because participants were not allowed to decline judgment, it is possible that,

though participants held the causal beliefs reflected in their judgments, they did so with

little confidence. Thus, we turn to participants' confidence ratings (Figure 9). First, note

that confidence ratings for unobserved causes were significantly greater than the

midpoint of the scale (all p's < .05). Second, we compared participants' confidence

ratings for observed causes (M = 4.88, SD = 1.59) with their confidence ratings for

unobserved causes (M = 4.83, SD = 1.51) separately for each condition. There were no

significant differences between these ratings in any of the conditions (all p's > .3). Thus,

not only were participants' able to make judgments of the unobserved cause, but

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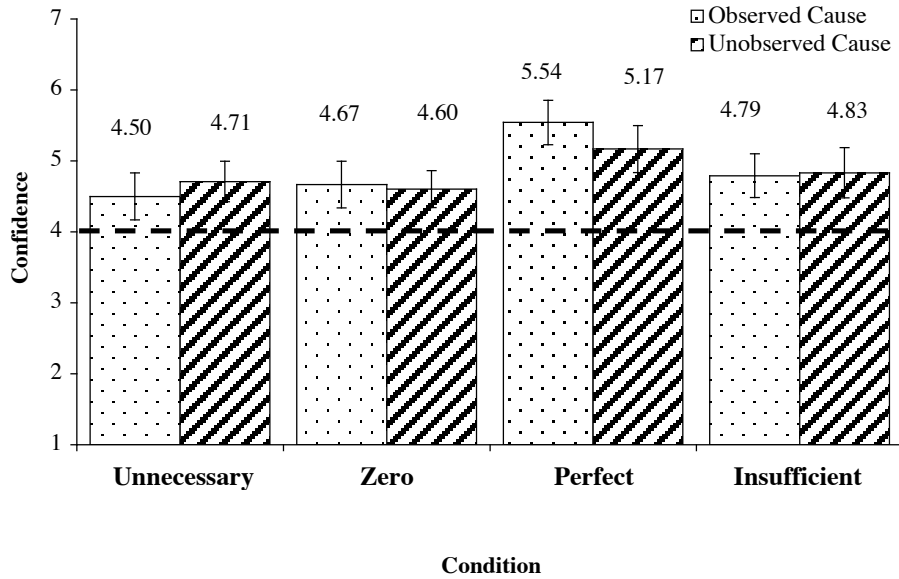
Figure 9 - Confidence judgments from Experiment 2A. Error bars indicate standard error.

participants were just as confident in these judgments as they were in their judgments about observed causes.

Simulating Experiment 2A with BUCKLE. We used BUCKLE to simulate each of the conditions used in Experiment 2A by presenting the model with the exact same set of observations in the exact same order that participants received them. The results of these simulations can be seen in Figure 8. As expected, the perceived strength of the unobserved cause (q_u) was high in the two conditions that included $\overline{OE}$ observations and low in the two conditions that did not. These results closely mirror the empirical findings. With regards to the judgments (including judgments of both unobserved and observed causes) from Experiment 2A (which used the methodology appropriate to test BUCKLE), BUCKLE's estimates accounted for 79% of the variance in and resulted in an RMSD of 12.90. Note that BUCKLE provides a better fit than the MLE and accounts for participants' lower judgments of the observed cause in the Unnecessary condition.

Experiment 2B

Participants' causal ratings from Experiment 2B can be seen in Figure 10. A 2 ($\overline{OE}$ present / absent) X 2 ($\overline{OE}$ present/ absent) repeated measures ANOVA was again performed on causal judgments of unobserved causes. This analysis revealed a significant main effect of unexplained effects, $F(1, 29) = 40.80$, $p < .0001$, because participants gave much higher ratings on conditions with unexplained effects ($M = 70.73$, $SD = 37.48$) than on conditions without unexplained effects ($M = 1.29$, $SD = 46.41$). No other significant effects were observed. These findings are comparable to those from Experiment 2A and support the idea that the presence of unexplained effects significantly influences judgments of the unobserved cause.

Participants' confidence ratings in Experiment 2B (Figure 11) were more varied than in Experiment 2A. In the Unnecessary and the Zero conditions, the confidence ratings for the unobserved cause ($M = 4.70$ and 4.19, $SD = 1.41$, 1.38 respectively) did not differ from confidence ratings of observed causes ($M = 4.89$ and 4.42, $SD = 1.20$, 1.36 respectively; p's > 0.26). In contrast, confidence judgments for the observed cause were greater than those for the unobserved cause in both the Perfect, ($t(29) = 3.65$, $p <$ .01), and the Insufficient conditions ($t(29) = 2.5$, $p < .05$). The reason for these differences is unclear, especially given that no such differences were found in Experiment 2A. Yet, it is interesting to note that participants were only more confident in their ratings of the observed cause when they did not observe $\overline{OE}$ (i.e., a particularly influential observation for learning about the unobserved cause according to BUCKLE).
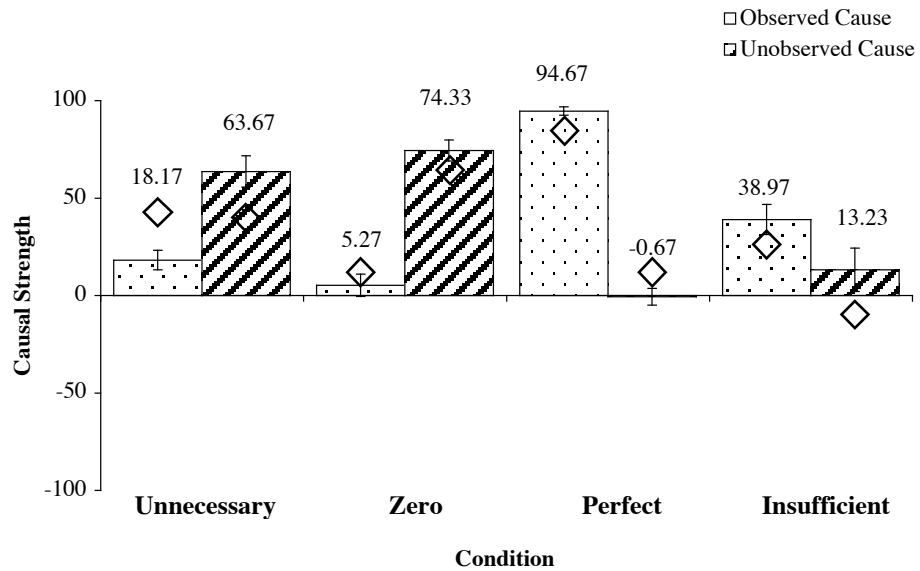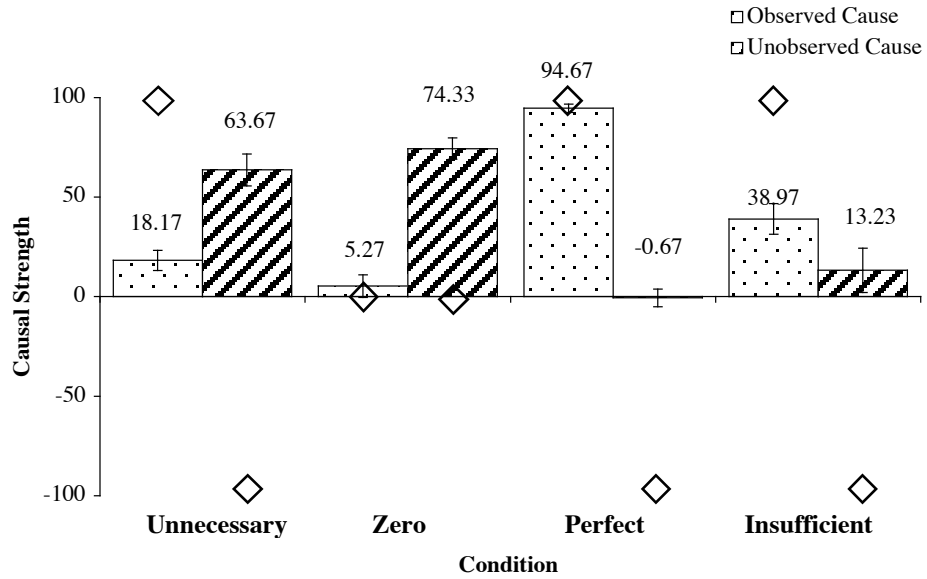
Figure 10 - Causal strength judgments from Experiment 2B. Error bars indicate standard error. The empirical data in the two plots is identical. In the top graph, the diamonds represent the constraint-satisfaction model's strength estimates. In the bottom graph, the diamonds represent the RW's strength estimates.

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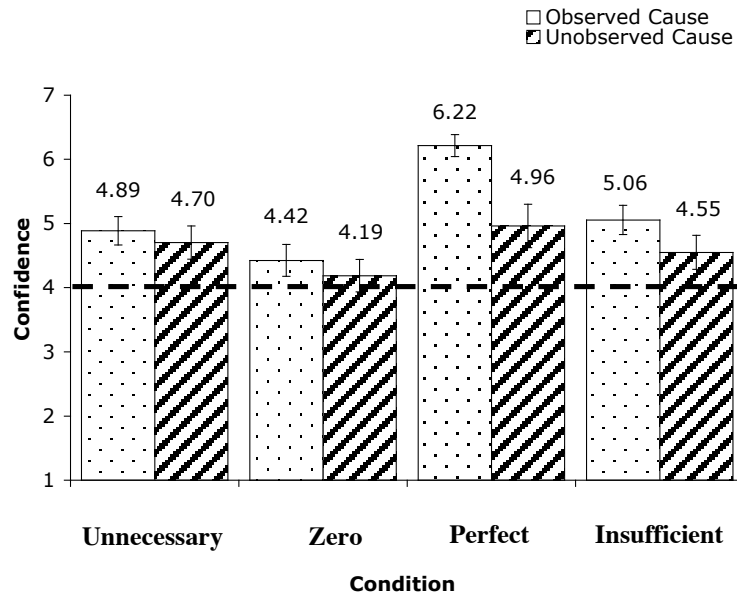
Figure 11 - Confidence judgments from Experiment 2B.
Error bars indicate standard error.

Thus, the absence of $\overline{OE}$ observations led participants to perceive the unobserved cause

as weak and to be less confident when doing so.

Simulating with the Constraint-satisfaction Model. We used the constraint-

satisfaction model to simulate Experiment 2B by constructing a different network for

each of the four conditions. For each condition, the network included one node

representing each type of observation included in that condition. The nodes representing

OE and $\overline{O}\,\overline{E}$ observations were always linked to the node representing the observed cause

explanation with positively weighted links. The nodes representing $\overline{O}E$ and $O\overline{E}$

observations (when included) were always linked to the node representing the observed

cause explanation with negatively weighted links. The two explanation nodes (one for

the observed cause, one for the unobserved cause) were linked to each other with a

negatively weighted link. Additionally, the observation nodes were linked to a special node whose activation was clamped at 1 (i.e., maximal).

The weights used were identical to those used in previous simulations and have allowed such models to account for a range of both causal and non-causal judgments (e.g., Thagard, 2000; Hagmeyer & Waldmann, 2002). Observation nodes and explanation nodes were connected with links taking on positive weights of 0.05 or negative weights of –0.05. The weight of the link between the explanation nodes was –0.2. The activation of all nodes was then updated for 200 iterations (as in Thagard, 2000). The resulting activation of each explanation node (multiplied by 100 to match the scale used by participants) was then taken as the judged strength of the cause.

The results of this simulation are displayed in the top panel of Figure 10. As expected, the unobserved cause was predicted to be strongest in the Zero condition. In all other conditions the model predicted that the unobserved cause would be perceived as weaker. Unfortunately, these predictions are drastically different than the results of Experiment 2B and provide a poor fit to the data (R^2=.33, RMSD=88.84)[10]. The two biggest errors made by the constraint-satisfaction model are not predicting an influence of unexplained effects and the fact that the final activations tended towards the extremes (i.e., 100 and -100).

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[10] In the C~E present/~CE present condition, all activation change is entirely due to the initial, random activations (positive initial weights lead to positive final activations, negative initial weights lead to negative final activations). In the absence of these initial activations, there are no changes in activation; the two explanations are in perfect equilibrium. Thus, in the absence of these random activations and assuming that people begin the experiment believing that the causal strengths are zero (as BUCKLE and RW do), the resulting activation of each explanation node should be zero.

Constraint-satisfaction models are traditionally able to take any beliefs a reasoner might entertain and represent the complex interdependencies that related them (see Thagard, 2000 for a wide array of applications). Why then does the constraint-satisfaction model fail to account for the results of Experiment 2B? The constraint-satisfaction model assumes that causes compete. This can be seen in the results of the simulation. The unobserved cause explanation is maximally active in the Zero condition in which there is an equal amount of positive (i.e., OE, $\overline{OE}$) and negative (i.e., $\overline{O}E$, $O\overline{E}$) support for the observed cause. In all other conditions, the unobserved cause explanation is rejected because there is more evidence in favor of the observed cause explanation than there is evidence against it. The adversarial relationship between causes/explanations is useful for explaining causal judgments (see Morris & Larrick, 1995; Price & Yates, 1993), but it apparently cannot explain beliefs about unobserved alternative causes. For example, participants believed both the observed and unobserved causes to be moderately causal in both the Unnecessary and Insufficient conditions. This behavior leads us to conclude that, without substantial additions, constraint-satisfaction models do not accurately describe participants' beliefs about unobserved causes.

Simulating with RW. Though Experiments 4-6 are more tailored to test RW, we briefly summarize simulations conducted with RW. RW was assumed to learn about two cues in the current experiment. The first, cue_{obs} represents the observed cause. The other, cue_{bg}, represents an unobserved cause that is assumed to be present on every trial. We simulated each of the conditions used in Experiment 2B (which used the methodology appropriate to test RW) by presenting the model with the exact same set of observations in the exact same order that participants received them. The final associative strength of

cue$_{obs}$ (multiplied by 100 to match the scale used by participants) was taken as the judged strength of the observed cause. The final associative strength of *cue*$_{bg}$ (again multiplied by 100) was taken as the judged strength of the unobserved cause. Because the predictions of RW are quite sensitive to the learning rate, the learning rate, α, was fitted to the data (again using the Hooke-Jeeves method). Additionally, to allow for the possibility that the two cues were differentially salient, we fit a separate learning rate, α, to each. The learning rate parameter associated with the outcome, β, was set to 0.5 for all simulations. The best fitting parameters were 0.86 for *cue*$_{obs}$ and 0.62 for *cue*$_{bg}$. The estimates can be seen in the bottom panel of Figure 10. These estimates fit the data quite well, (R^2=.77, RMSD=21.97). These results will be discussed more thoroughly later.

Experiment 3

Experiments 2A and 2B demonstrated the importance of unexplained effects in causal learning. The influence of unexplained effects was the most critical aspect of BUCKLE's predictions and set it apart from the constraint-satisfaction model. One remaining question, however, involves the extent to which our methodology led participants to be sensitive to unexplained effects. Participants in Experiment 2 were given explicit information about the potential existence and influence of an unobserved cause on every trial. As explained earlier, this measure was taken to equate participants' assumptions about the learning environment with those of BUCKLE. Yet, this prior knowledge may have inadvertently changed participants' learning strategies with respect to unobserved causes. That is, it is not clear from these experiments the extent to which people make spontaneous inferences about unobserved causes. To explore how general

the influence of unexplained effects is, we conducted an additional experiment in which

participants were given no prior information about the existence of an unobserved cause.

Method

Fifty-two Yale University undergraduates participated for partial fulfillment of

course credit or $10/hour. Participants were randomly assigned to either the Explicit

condition (N=26) or the Implicit condition (N=26).

Stimulus materials and procedure was the same as in Experiment 2A except for

the following changes. Stimuli consisted of electrical systems like those used in previous

experiments. Instead of using all four contingencies, only two critical conditions were

used. These were the Unnecessary and Insufficient conditions (see Table 1).

Participants in the Explicit condition were told that there were two buttons and a

light as in Experiment 2. These participants were told that nothing else could affect the

light. In contrast, participants in the Implicit condition were told that the system included

one button and a light. Participants in the Implicit condition were not given any

information about the existence (or nonexistence) of alternative causes until they were

asked to evaluate their causal strengths after all observations were completed. All

participants were told that they would be asked to evaluate the causal strength of the

observed button. After viewing the entire set of trials participants in the Implicit

condition were told that the initial description of the system was incomplete. They were

told that the system actually contained a second button whose information had been lost

and that in each presented test any combination of these two buttons could have been

pressed (i.e. either of the buttons alone, both of the buttons together, or neither of the

buttons). All participants then evaluated each button as in Experiment 2A.

Results and Discussion

Mean causal ratings are presented in Figure 12. The pattern of results was

identical to that found in Experiments 2A and 2B. Judgments of the unobserved cause

varied depending on whether unexplained effects were included or not. Most

importantly, this effect was found even in the Implicit condition where participants had

no prior information about an alternative cause until they were asked to make

judgments[11]. A 2 (contingency structure: $\overline{O}E$ present/$O\overline{E}$ absent vs. $\overline{O}E$ absent/$O\overline{E}$
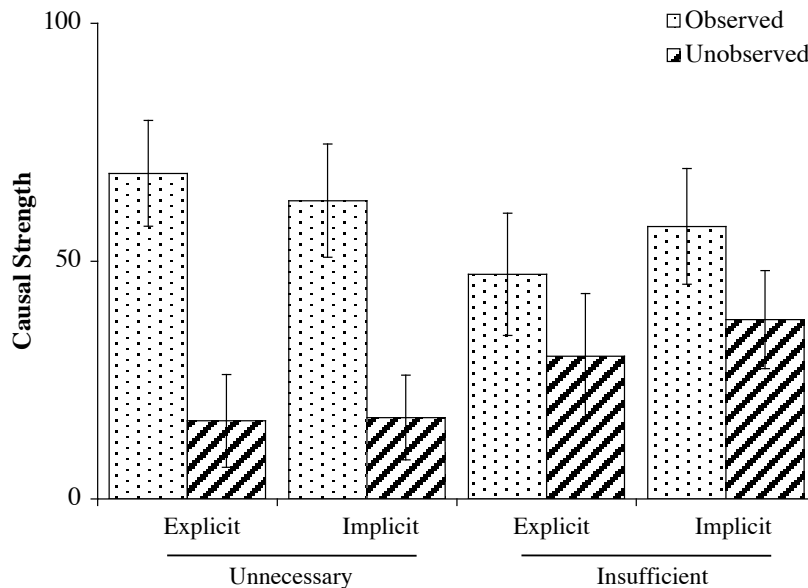
Figure 12 - Causal strength judgments from Experiment 3.
Error bars indicate standard error.

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[11] One might argue that participants made judgments about the unobserved cause
retrospectively when they were asked about the unobserved cause, rather than

present) X 2 (explicit / implicit) ANOVA was performed on the ratings of the unobserved cause. This analysis revealed a significant main effect of contingency structure, $F(1, 46)=21.28$, $p<.0001$, because participants gave much higher ratings in the Unnecessary condition ($M = 65.58$, $SD = 40.75$) than in the Insufficient condition ($M = 16.75$, $SD = 31.53$). Both the main effect of the instructional manipulation (i.e. level of implicitness) and the interaction effect were non-significant (both F's < 1).

The absence of an interaction between implicitness and contingency structure is theoretically important because it suggests that people naturally learn about unobserved causes[12]. Because of its importance, this effect was further subjected to a power analysis. While our sample provided sufficient power (.81) to detect a "large" effect (i.e. $f = .4$), the observed effect size was significantly smaller, $f = .045$. Achieving similar power to detect an effect of this size would require an additional 3800 participants.

Summary

Experiments 2 and 3 demonstrate that judgments of unobserved causes are systematic and vary depending on the contingency between the observed cause and the effect. Unexplained effects were particularly influential. The unobserved cause was perceived as strong only when participants encountered unexplained effects. Otherwise,

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spontaneously making inferences about the unobserved cause during learning. Experiment 6 provides evidence against this interpretation.

[12] What exactly learners represent remains unclear. For example, it is unclear whether people represent a single unobserved cause, as BUCKLE does, or whether they represent more than one. To explore this possibility, we modified BUCKLE so as to represent multiple unobserved causes. Though these modified versions of BUCKLE do make different quantitative predictions, the effect is too subtle to draw any reasonable conclusions given the current result.

the unobserved cause was perceived as weak. BUCKLE, which is developed to be sensitive to unexplained effects, obviously could explain such an effect.

These experiments did not, however, provide a critical comparison of BUCKLE and RW. As explained earlier, RW represents an unobserved cause and assumes that it is present at all times. The associative strength accrued by this cause matched Experiment 2B's results quite well. Because of the apparent accuracy of RW's account, we wish to conduct a more thorough exploration into the details. We plan to test RW in three ways. First, Experiment 4 and 5 will explore the difference between BUCKLE's and RW's representation of unobserved causes. RW makes the assumption that the unobserved cause is present at all times whereas BUCKLE computes the probability that the unobserved cause is present for each observation. Second, RW is indifferent to the causal role of events, whereas BUCKLE utilizes information about causal role. This critical difference will be explained and tested in more detail in Experiment 6.

Experiment 4: Judging a Constant Cause

Though the unobserved cause in our experiments is certainly part of the background, it seems unnatural to actually equate the two. The experimental context includes the unobserved cause as well as other less obvious elements (e.g., the color of the background during stimuli presentation). Thus, while causal judgments of the unobserved cause should be related to the associative strength of the background, it is unclear whether the two should be equal.

To explore this idea, we designed an experiment to evaluate the predictions of RW more directly. Instead of being presented with an observed cause and an unobserved

cause as in previous experiments, participants in the experimental condition will be presented with two observed causes. One cause will vary (i.e., sometimes present, sometimes absent), whereas the other cause will always be present. We will then compare participants' judgments about the constantly present cause to their judgments of an unobserved cause in the control condition. If people's judgments of the unobserved cause are based on the associative strength of the background context, then judgments of the constant cause should be equal to judgments of the unobserved cause.

Method

Participants

Thirty-six Yale University undergraduates participated for partial fulfillment of course credit or $10/hour.

Materials and Design

Stimuli consist of electrical systems like those used in previous experiments. The experiment uses a 2 (contingency) X 2 (Unobserved vs. Constant cause) within-subject design. Instead of using all four contingency structures, only two critical structures were used. The contingencies will be the Unnecessary and Insufficient conditions (see Table 1), but will include ten trials of each type instead of seven as in Experiment 2. In the Constant condition, one cause will be observed and vary according to the particular contingency while the other will be observed and was present on every trial. In the Unobserved condition one cause will be observed and vary according to the contingency of the particular condition while the other cause will be unobserved (as in previous experiments).

Procedure

The procedure was identical to that used in Experiment 2B.

Results

Results can be seen in Figure 13. The critical question in Experiment 4 is whether

people would evaluate the constant cause in the same manner as the unobserved cause.

We performed a 2 (condition: Unobserved vs. Constant) x 2 (contingency: $\overline{OE}$ present/

absent vs. $O\overline{E}$ present/ absent) repeated measures ANOVA on judgments of the

constant/unobserved causes. We observed a significant effect of condition ($F(1,34)=8.39$,

$p<.01$) because the causes were perceived as stronger in the Unobserved condition

(M=44.53) than in the Constant condition (M=13.42). We also observed a significant

effect of contingency ($F(1,34)=27.45$, $p<.0001$) because the causes were perceived as

stronger in the Unnecessary condition (M=57.42) than in the Insufficient condition

(M=0.53). The interaction between factors was not significant.

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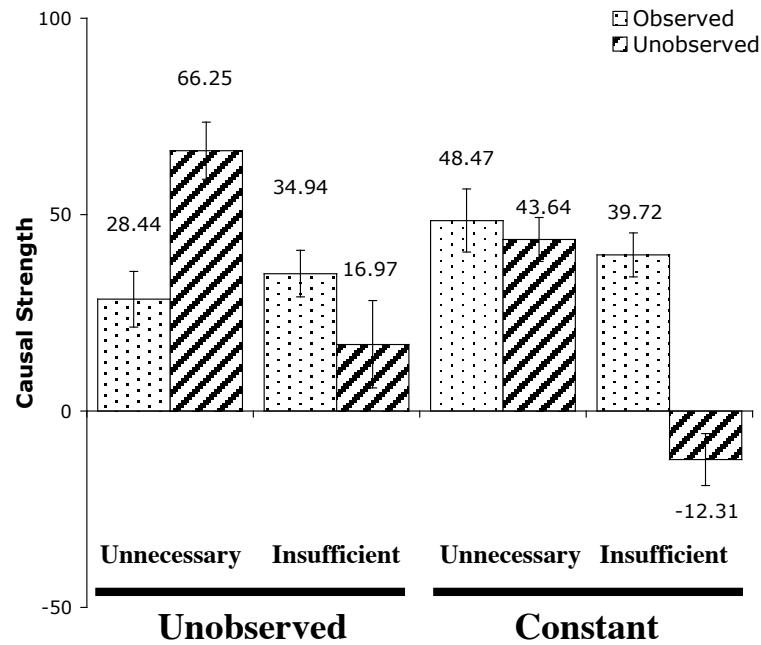
Figure 13 - Causal strength judgments from Experiment 4.
Error bars indicate standard error.

The above analyses suggest that the only difference between conditions was in the overall magnitude of participants' causal judgments. This effect results in several findings of note. First, judgments of the constant cause in the Insufficient contingency (M=-12.31) were marginally less than zero (t(36)=1.86, p=.07). This implies that participants believed the constant cause to be preventative in nature, a result not observed in the Unobserved condition (M=16.97). Looking back at the bottom panel of Figure 10, RW predicted a negative associative strength for the background cause in the Insufficient contingency. A second finding is that the constant cause in the Unnecessary contingency was judged to be just as strong as the varying cause (t(35)<1). This finding also differs from the Unobserved condition in which the unobserved cause was judged to be significantly stronger than the observed cause in the Unnecessary contingency (t(35)=3.81, p<.001 in the Unobserved condition). Looking again at the bottom panel of

Figure10, RW predicted equivalent associative strength for the two causes in the

Unnecessary contingency.

Taken together, these analyses suggest that judgments of the unobserved cause are

not equivalent to the associative strength of the experimental background. RW makes

clear predictions about the associative strength of constantly present causes (it is equal to

the associative strength of the background). Though the pattern of judgments for

unobserved and constant causes is similar (which allows RW a decent fit to the data),

there are important differences. For example, the unobserved cause was always judged to

be generative whereas the constant cause was judged to be preventative under certain

conditions. Thus, RW's context cue seems unable to account for people's judgments of

unobserved causes.

Of course, one advocates of RW could (and should) argue that judgments of the

unobserved cause are still some (unknown) function of the associative strength of the

background. We acknowledge that the unobserved cause is one part of the larger

experimental background. Perhaps we have simply mischaracterized the how learners

map the background cue to judgments of the unobserved cause. Due to this uncertainty,

we decided to explore additional predictions made by RW in Experiment 5 and 6.

Experiment 5: Evaluating Beliefs about the Occurrence of Unobserved Causes

According to BUCKLE, participants believe that unobserved causes are present

with some probability on every trial. RW makes the simpler assumption that the

unobserved cause is constantly present. Experiment 5 directly assesses people's beliefs

about the presence of unobserved causes, which will allow us to better evaluate BUCKLE

and RW. Experiment 5 was similar to Experiment 2; on every trial, participants were presented with information about the presence or absence of one of the causes and the effect while the second cause remained unobserved. However, unlike Experiment 2, participants were asked to rate the probability that the unobserved cause was present on that trial.

As explained in the introduction, BUCKLE predicts that the probability of the unobserved cause will vary as a function of type of evidence (e.g., OE or $\overline{OE}$) as well as q_u and q_o. More detailed predictions of BUCKLE will be presented later. In short, the main goal of Experiment 5 was to demonstrate that (1) people's estimates of P(u|o,e) in a given learning situation vary as a function of type of evidence and (2) the same type of observation can lead to different estimates of P(u|o,e) as a function of q_u and q_o.

In addition, because Experiment 5 provides direct assessments of P(u|o,e), it allows us to examine whether people are making the no-confounding assumption (i.e., P(u|o) = P(u|~o)) required by the Power PC theory. If participants do not believe this assumption met, the Power PC theory cannot estimate causal power and people should be unable to provide causal strength judgments (Cheng, 1997). In contrast, BUCKLE does not require such a constraint in learning of observed causes. Hagmeyer and Waldmann (2004) demonstrated that learners make causal judgments even when they believe the no-confounding assumption to be violated. Unfortunately, Hagmeyer and Waldmann use only a small variety of contingencies and they do not report data relevant to our test of BUCKLE (e.g., P(u|o) and P(u|~o)). Thus, Experiment 5 seeks to use a wider range of contingencies and will allow a better test of BUCKLE.

Participants

Twenty-four Vanderbilt University undergraduates participated for partial fulfillment of course credit.

Materials and Procedure

Stimuli consisted of four electrical systems used in Experiment 2. The design was the same as in Experiment 2 (Table 1) except that, in order to increase the number of measurements taken, we presented each cell 10 times instead of the original 7.

The procedure of Experiment 5 was the same as in Experiment 2A except for the following addition. After the trial information was presented and the spacebar was pressed to continue, a question appeared on the screen below the depiction of that trial. The question was, "How likely is it that the [color] button was pressed in this test?" where the button referred was the unobserved cause. Below this was an eleven-point scale ranging from 0 (Definitely NOT pressed) to 10 (Definitely pressed). After typing their answer on the computer keyboard, the next trial commenced. It should be noted that asking participants about the probability of the unobserved cause on every trial did not appear to have created an unusually disruptive learning situation, because the pattern of causal strength judgments (see Figure 14) mirrored that of Experiment 2[13].

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[13] A 2 ($\overline{OE}$ present / absent) X 2 ($O\overline{E}$ present / absent) repeated measures ANOVA on causal judgments of unobserved causes revealed a significant main effect of $\overline{OE}$ information, $F(1, 22) = 26.59$, $p<.0001$, because participants gave much higher ratings on conditions with $\overline{OE}$ (M = 72.60, SD = 28.77) than on conditions without $\overline{OE}$ (M = 41.47, SD = 35.69). No other main effects or interactions were significant.
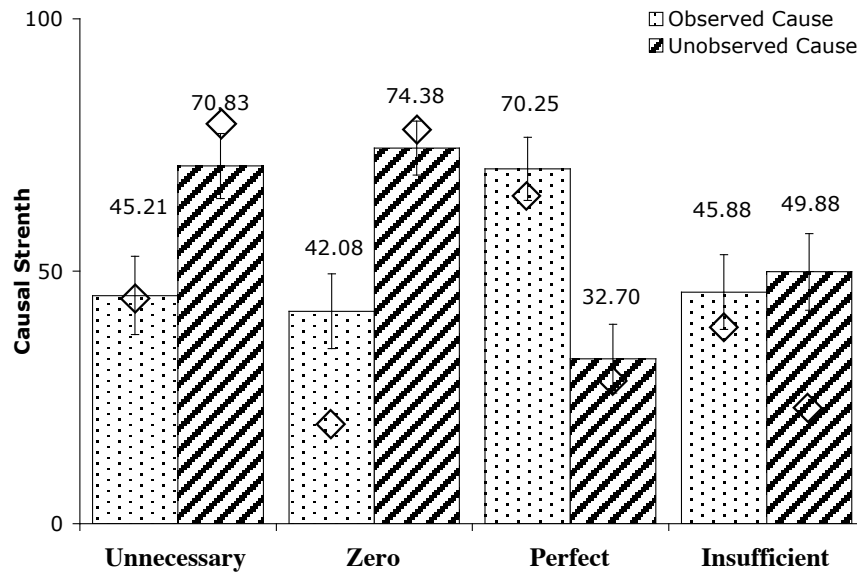
Figure 14 - Causal strength judgments from Experiment 5. Error bars indicate standard error. The diamonds represent BUCKLE's estimates.

Results

Table 2 shows mean ratings of P(u|,o,e) broken down by the four conditions and trial types. Figure 15 shows participants' mean ratings of P(u|,o,e) as a function of the given trial type's position during learning. The first finding to note is that participants did not believe that the unobserved cause was constant. Probability judgments varied

Table 2 - Average trial-by-trial probability judgments for the various trial types in each condition of Experiment 4. Marginal averages are also provided.

Condition	Unnecessary			Zero			Perfect			Insufficient		
	E	$\overline{E}$		E	$\overline{E}$		E	$\overline{E}$		E	$\overline{E}$	
Likelihood(u) O	5.8		5.8	5.38	**3.41**	4.4	4.1		4.1	5.25	**3.76**	4.51
$\overline{O}$	7.51	2.15	4.83	7.84	1.82	4.83		3.16	3.16		2.7	2.7
	6.66	2.15		6.61	2.62		4.1	3.16		5.25	3.23	

considerably and systematically. These results cannot be accounted for by RW because RW does not allow the probability of the unobserved cause to vary.

For example, participants' probability judgments varied as a function of type of observation. Individual one-way repeated measures ANOVAs were performed on each of the four conditions with the trial type as a variable. The main effect of trial type was significant in three of the four conditions (all p's < .05) and marginally significant in the Perfect condition ($F(1,23) = 3.67$, p=.068). Thus, as would be expected given BUCKLE, participants appear to be making varied, but systematic inferences about the presence of the unobserved cause.

Participants also expected that the presence of the unobserved cause to co-vary with the effect. This can be seen by looking at the marginal averages below each matrix in Table 2; P(*u*|,*o*,*e*) was judged to be higher when *e* was present than when *e* was absent. This finding makes sense given that participants' causal strength judgments for the unobserved cause were greater than zero in all four conditions; positive causal judgments should imply positive covariation.

Taking this a step further, participants should have believed the unobserved cause covaried with the effect more in the two conditions that elicited high causal judgments of the unobserved cause (Unnecessary and Zero) than the two conditions that elicited low causal judgments of the unobserved cause (Perfect and Insufficient). To evaluate this prediction, we compared OE trials and $\overline{O}\overline{E}$ trials because these were the only trial types shared across the four conditions. If participants believed the unobserved cause varied with the effect, participants should believe the unobserved cause to be more likely present on OE trials and less likely on $\overline{O}\overline{E}$ trials. If participants do not believe the unobserved

cause covaries with the effect, they may believe that the likelihood of the unobserved cause being present is more similar on these two trial types.

For each participant, their average rating for $\overline{O}\overline{E}$ trials was subtracted from their average rating for $O\overline{E}$ trials. This composite score served as an index of the degree to which participants believed the unobserved cause to covary with the effect on these trials. A 2 ($\overline{O}E$ present / absent) X 2 ($O\overline{E}$ present / absent) repeated measures ANOVA was performed on this composite. This analysis revealed a significant main effect of unexplained effects ($\overline{O}E$ trials), F(1, 23)=8.77, MSE=84.52, p<.01, because the composite was higher in conditions with $\overline{O}E$ (M = 3.60, SD = 3.46) than on conditions without $\overline{O}E$ (M = 1.72, SD = 3.29). The main effect of $\overline{O}E$ trials and the interaction between the two factors were both marginally significant (p=.073 and p=.063 respectively).

The above analyses were based the average judgments collapsed across the 10 occasions each trial type was encountered. One might assume, given that the current experiments are designed to measure learning, that judgments would change gradually as experience accumulated (see Shanks, Holyoak, & Medin, 1996 for a variety of results detailing how judgments change during causal learning). In fact, our participants' trial-by-trial judgments changed very little. As illustrated in Figure 15, participants' average ratings of each of the trial types in each of the conditions each time they were asked did not vary greatly as learning proceeded. Regression analyses were performed on each participant's ratings for each of the trial types (i.e. OE, $O\overline{E}$, $\overline{O}E$, $\overline{O}\overline{E}$) in each condition using trial number as the sole regressor. This analysis allows us to see the extent to which likelihood judgments increased or decreased as learning progressed.

Figure 15 - Participants' estimates of P(u|,o,e) from Experiment 5. Each of the four conditions is illustrated in its own graph.

The results show that none of the slopes were significantly different from zero except

for $\overline{OE}$ trials in the Unnecessary condition (slope = –0.14).

We were also interested in the degree to which participants believed the

unobserved cause varied with the observed cause. As mentioned in the Introduction,

some models of causal learning (Cheng, 1997; Pearl, 2000) avoid making specific

inferences about unobserved causes by assuming that unobserved causes are independent

of observed causes (i.e., P(o|u)=P(o|~u)). In contrast, recent work (Hagmeyer &

Waldmann, 2004) suggests that participants do not necessarily share this assumption. To

evaluate these possibilities, we averaged likelihood ratings for trials in which the

observed cause was present (i.e., P(u|o)) and for trials in which the observed cause was

absent (i.e., P(u|~o)). This was done separately for each subject and each condition. The

difference between these quantities was significant in the Unnecessary condition (mean

difference = .97, $t(23)=2.18, p<.05$), the Zero condition (mean difference = -.43,

$t(23)=2.12, p<.05$), and the Insufficient condition (mean difference = 1.80,

$t(23)=4.38, p<.001$) and was marginally significant in the Perfect condition (mean

difference = .94, $t(23)=2.02, p=.055$). Note that it was not simply the case that

participants believed the two causes to be correlated. In the Zero condition, the

unobserved cause was significantly less likely in the presence of the observed cause.

These findings mirror those of Hagmeyer and Waldmann (2004) and suggest that learners

are not making the simplifying assumptions required by some models (i.e., Cheng, 1997;

Pearl, 2000).

We used BUCKLE to simulate each of the conditions used in Experiment 5 by presenting the model with the exact same set of observations in the exact same order that participants received them. The parameters were fit or set as in Experiment 2. BUCKLE's predictions accounted for 76% of the variance in participants' causal judgments and resulted in an RMSD of 13.65.

The results of BUCKLE's likelihood computations (multiplied by 10 to match the scale used by participants) can be seen in Figure 16. We note several important features of these simulations. First, as shown by separate lines in each graph in Figure 16, BUCKLE predicts that likelihood estimates should differ depending on type of evidence (e.g., OE vs. $\overline{O}\overline{E}$). To quantitatively evaluate the fit between participants' estimates and BUCKLE's predictions about the influence of trial type and condition on likelihood judgments, we averaged likelihood estimates separately for each trial type in each condition for all participants and for BUCKLE. The likelihood estimates generated by BUCKLE provided a good fit, accounting for a significant amount of variance in participants' likelihood judgments (R^2=.91, RSMD=1.04).

BUCKLE also mirrors participants' belief that the unobserved cause (which is estimated to be generative) is more likely in the presence of the effect than in its absence. As can be seen in Figure 16, the likelihood of u being present is much higher when the effect is present (e.g., OE and $\overline{O}E$) than when the effect is absent (e.g., $\overline{O}\overline{E}$ or $O\overline{E}$), illustrating this co-variation. Note also that the difference between P(u|o,e) for OE trials

and P($u|o,e$) for $\overline{OE}$ trials is fairly large in these two conditions. In contrast, the bottom
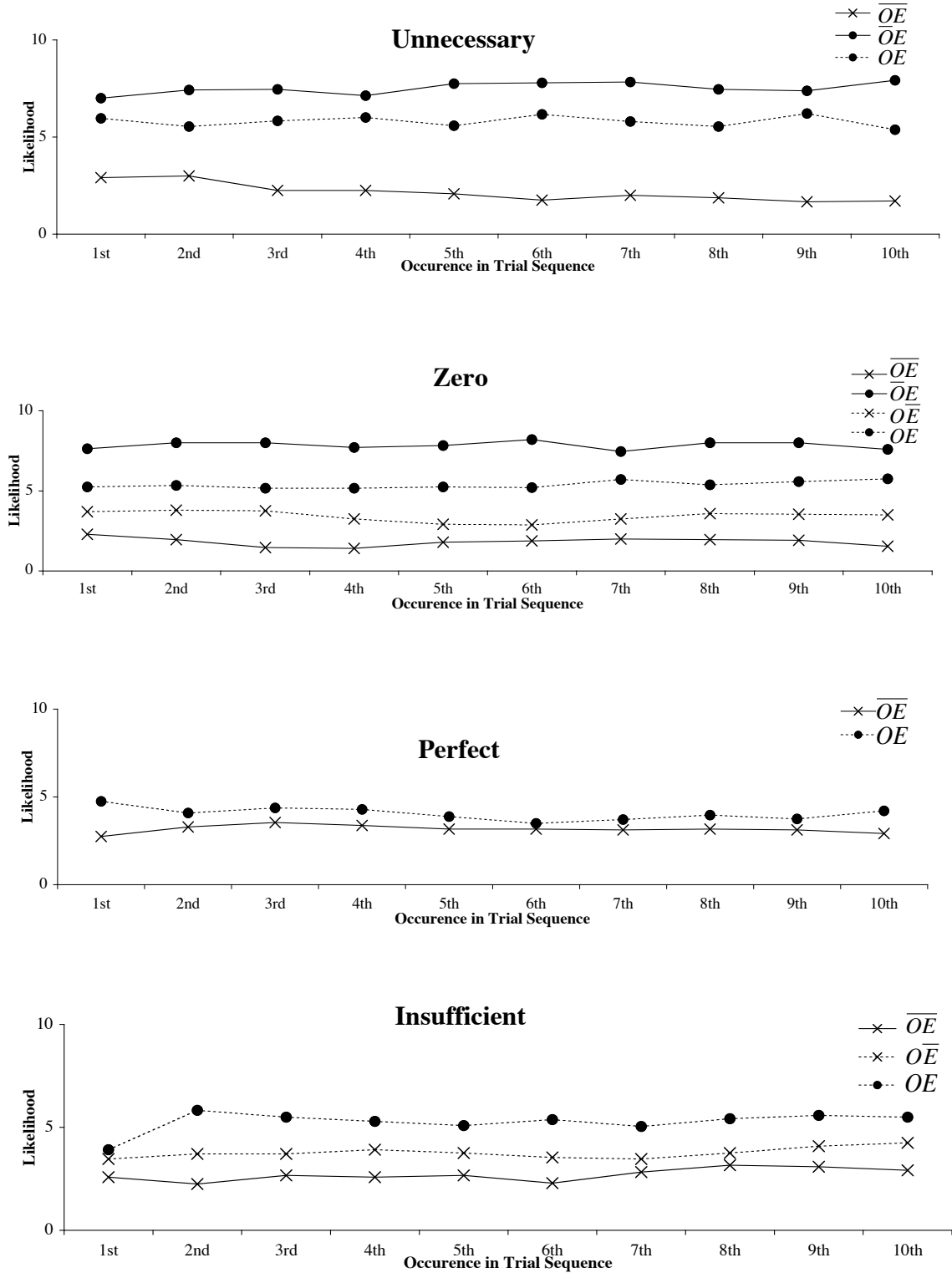
two panels show conditions where q_u was only somewhat positive.
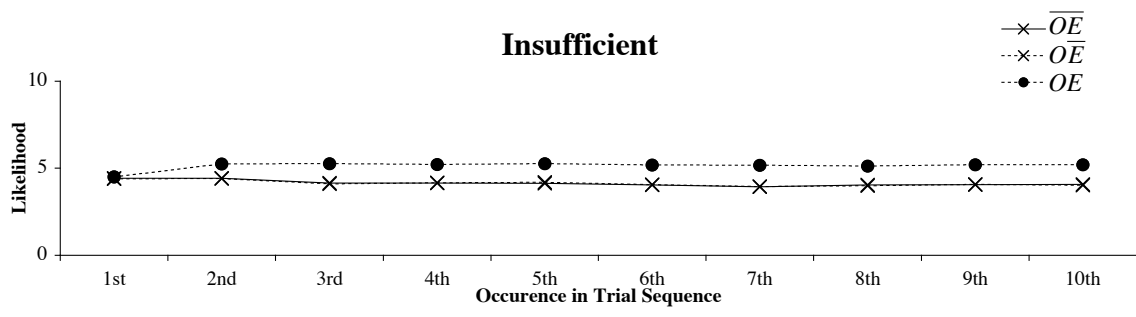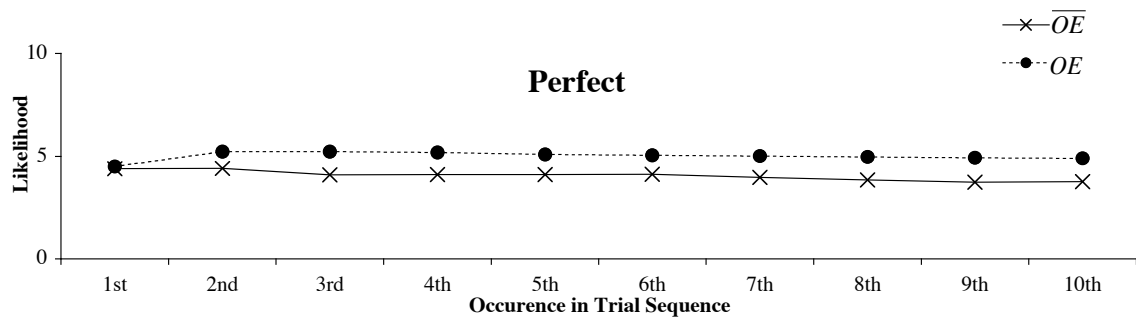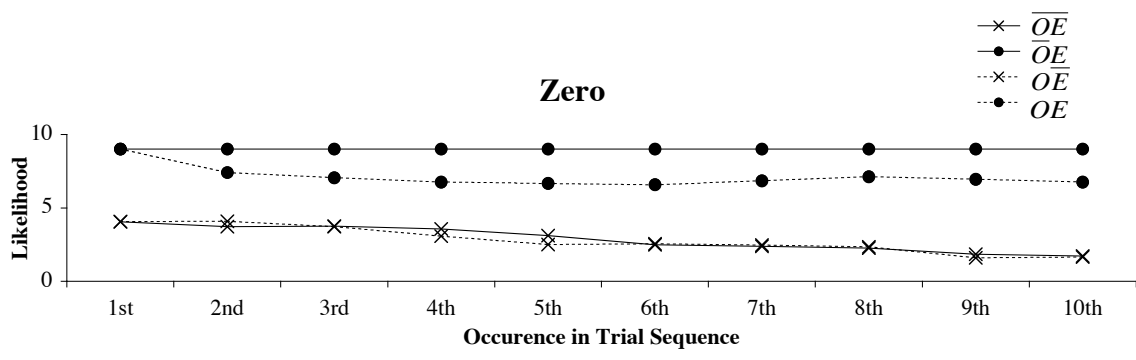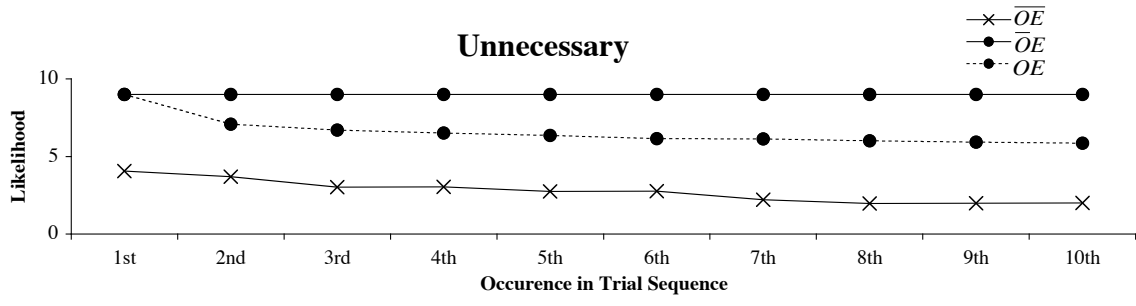
Figure 16 - BUCKLE's estimates of P(*u*|,*o*,*e*) from Experiment 5. Each of the four conditions is illustrated in its own graph.

Here, the difference between P($u|o,e$) for *OE* trials and P($u|o,e$) for $\overline{OE}$ trials is much

smaller, indicating only moderate co-variation. To test this more thoroughly, we

computed a composite score (as before) using BUCKLE's likelihood estimates during

$\overline{OE}$ and *OE* trials. BUCKLE's composite scores (3.82, 4.22, .96, and 1.0 for the

Unnecessary, Zero, Perfect, and Insufficient conditions respectively) accounted for 72%

of the variation in our participants' composite scores (RMSD = .85).

Lastly, we should note that, just as in our participants' judgments, BUCKLE's

likelihood estimates do not generally demonstrate substantial learning effects. The

likelihood of the unobserved cause being present for a particular observation in a

particular condition remains relatively constant and certainly doesn't demonstrate

changes of the magnitude seen in causal strength. As discussed in the introduction,

parameters such as q_o and q_u (i.e., those that change during the course of the experiment)

have a much smaller effect on the likelihood judgment than do the state of the observed

cause and effect. Thus, BUCKLE argues that changes in the likelihood judgments should

necessarily be smaller than those in causal strength. Nonetheless, there are small changes

in both BUCKLE's and participants' likelihood judgments over the course of learning.

We again fit lines to BUCKLE's likelihood inferences for each of the trial types in each

of the four contingencies. Despite the highly truncated range of slopes, the slopes of

these lines accounted for 24 percent of the variance in participants' mean slopes (p=.10).

Summary

The results of Experiment 5 illustrate that people do not make the simplifying

assumption about unobserved, alternative causes as RW does, nor do they believe the no-

confounding assumption as some models require (Cheng, 1997; Pearl, 2000). Instead

learners make sophisticated inferences about the likelihood of unobserved, alternative

causes. Judgments about the likelihood of the unobserved cause varied greatly as a

function of whether the observed cause and the effect were present. Judgments also

varied, even for identical observations, according to the contingency used. For example,

the two conditions that elicited strong causal judgments for the unobserved cause lead

participants to believe that the unobserved cause varied with the effect more than the two

conditions that elicited weaker unobserved cause judgments. These findings suggest that

beliefs about causal strength (e.g., the perceived strength of the unobserved cause) work

to shape beliefs about the likelihood of the unobserved cause beyond the relatively strong

influence of observations type.

One might argue that it is overly simplistic to always assume that all unobserved

causes are constantly present, and RW's extra cause node should only represent those

events that are actually constantly present (e.g., color of the walls), and not the alternative

causes that might vary across learning trials. However, this leaves RW unable to deal

with the very issue that we set out to examine, namely, how learners deal with

unobserved alternative causes.

Experiment 6: Manipulating Causal Role

Another important difference between BUCKLE and RW is that BUCKLE is a

model of causal learning, whereas RW is a model of associative learning, which does not

distinguish between the learning of causal relationships and other, non-causal

relationships. Because of this, RW assigns events to nodes in its network using the order

in which those events are presented to the learner. When presented with information about two events, the event that occurs first is mapped to the input and the event that follows is mapped to the output. This ordering may map onto the concepts of cause and effect in typical situations, but there is no principled relationship between presentation order and causal role. For example, when confronted with new medical disorders, scientists often learn about the symptoms (i.e., effects) of the disorder before discovering the cause of those symptoms. In such situations, the predictions of associative models such as RW diverge from those of models that acknowledge the cause-effect distinction. For example, Waldmann and his colleagues have shown that cue interaction (blocking, overshadowing, etc.), a well-known learning phenomenon, is sensitive to causal role but not presentation order (Waldmann & Holyoak, 1992; Waldmann, Holyoak, & Fratianne, 1995; Waldmann, 1996, 2000, 2001; but see Shanks and Lopez, 1996 as well as Tangen and Allan, 2004).

Experiment 6 is designed to test whether learning about unobserved causes is sensitive to causal role (as BUCKLE assumes) or presentation order (as RW assumes). To do so, we used two conditions as illustrated in Figure 17. In the Predictive condition, the causal structure is the same as the previous experiments reported so far; there are two causes – one observed and one unobserved – and one observed effect. To ensure an unambiguous presentation order, information about the presence of causes was always presented to participants before information about the presence of the effect. In the Diagnostic condition, there are two effects – one observed and one unobserved – and one observed cause. In this condition, information about the presence of effects was always presented to participants before information about the presence of the cause.

Predictive

Blue button.

Light did not turn on.

Red button was not pressed.

Diagnostic

Blue light.

Button was not pressed.

Red light did not turn on.
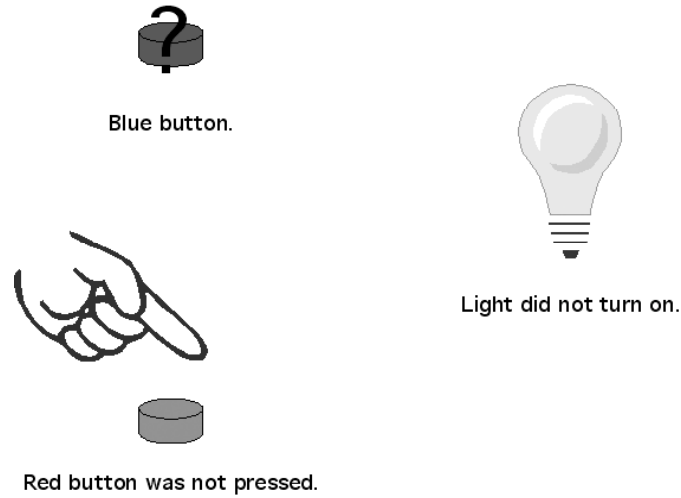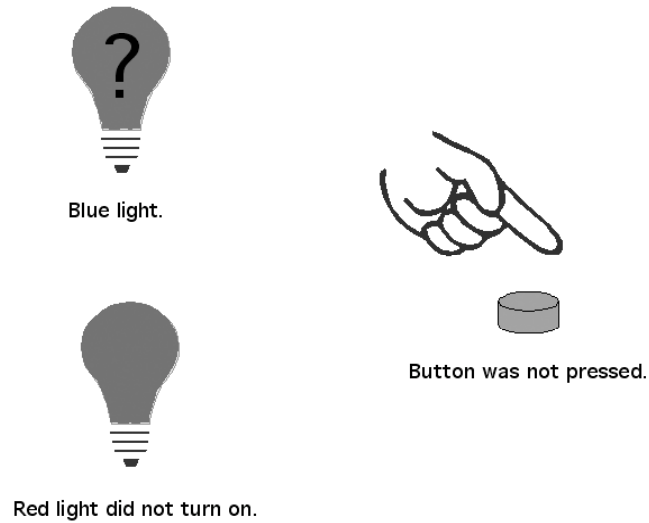
Figure 17 - Illustrating the difference between the Predictive and Diagnostic conditions used in Experiment 6. In the Predictive condition, the cues are the buttons (i.e., causes). As in previous experiments, one is observed and one is unobserved. In the Diagnostic condition, the cues are the lights (i.e., effects). In this condition, one of the lights is observed and one is unobserved.

According to RW, these two conditions are identical; "for an associative account, however, the real-world interpretation of the events is immaterial" (Shanks, Lopez, Darby, Dickinson, 1996, p. 281). Therefore, if people's judgments in the Diagnostic differ from those in the Predictive conditions, RW will be unable to explain why this is the case.

Yet, an attempt has been made in the associative camp to devise a way to be sensitive to causal role. Shanks and Lopez (1996) proposed that diagnostic learning takes place by running two associative networks in parallel. One network would learn according to presentation order (effects to causes) and one according to causal role (causes to effects). On this account, which network's weights are used to generate judgments is based entirely on the judgment being elicited. If the learner is asked to make a judgment based on presentation order, then the learner uses the network that tracked presentation order as traditionally modeled. If, on the other hand, the learner is asked to make a judgment based on causal role (a diagnostic judgment), then the learner uses the network that tracked causal roles. To avoid this possibility, Experiment 6 elicited judgments based on presentation order following Waldmann (2001): In both the Diagnostic and Predictive conditions, participants were asked about the degree to which each of the first events predicted the second. This methodological detail disallows participants from using their knowledge of causal roles. In the Diagnostic condition in particular, participants were asked about the degree to which the presence of the effect

predicts the presence of the cause, and thus they can only rely on the presentation order.

Thus, RW's predictions are truly equated across to two conditions[14].

Method

Participants

Forty-eight Yale University undergraduates participated for partial fulfillment of course credit or $10/hour. Each participant was randomly assigned to either the Predictive (N=24) or the Diagnostic condition (N=24).

Materials and Design

Stimuli for the Predictive condition consisted of four electrical systems identical to those used in previous experiments. Stimuli for the Diagnostic condition also consisted of four electrical systems except that there were two effects (i.e., lights) and one cause (i.e., button). Each of the lights used in this condition was a different color to aid in their memory and to ensure that subjects did not confuse different systems. Each system in the Diagnostic condition contained exactly one light whose state (lit or not) was observable, one light whose state was unobservable, and a single button. The unavailable state of the unobserved light was denoted via a large question mark superimposed over the light (see Figure 17). The state of the button (pressed or not) was always observable in this condition.

The contingencies of the four systems used were identical to those used in Experiment 2 (see Table 1) and were identical across the two conditions. However, since

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[14] Unfortunately, using this methodology makes it impossible to derive BUCKLE's predictions for the Diagnostic condition, because BUCKLE estimates the likelihood of an effect given a cause. It should be noted, however, that the main purpose of Experiment 4 is to test RW's predictions rather than to provide further support for BUCKLE.

the causal role is reversed in the Diagnostic condition, instead of naming the independent

variables in terms of *o* and *e* (which stand for observed cause and effect) we will refer

them in terms of Cue and Outcome (i.e., $\overline{Cue} \cdot Outcome$ present or absent, and

$Cue \cdot \overline{Outcome}$ present or absent). Thus, the experiment is a 2 ($\overline{Cue} \cdot Outcome$:

present/absent) X 2 ($Cue \cdot \overline{Outcome}$: present/absent) X 2 (Predictive versus Diagnostic

conditions) mixed design with the last factor being manipulated between subjects.

Procedure

The procedure was similar to that used in Experiment 2. Participants saw all four

systems in a counterbalanced order. Trials for each system were presented in quasi-

random order. One difference between this and previous experiments was the

presentation of individual trials. Because RW uses presentation order to determine the

difference between input and output, we wanted to make the presentation order as

unambiguous as possible. Thus, for all trials, the first event (i.e., causes in the Predictive

condition and effects in the Diagnostic condition) was presented in isolation for 250ms.

After this delay, the second event (i.e., the effect in the Predictive condition and the cause

in the Diagnostic condition) was presented alongside the first event. At this point the

participant was able to press a key to proceed to the next trial at her own pace.

After viewing the entire set of trials for a single system, participants were asked to

provide responses. To allow identical dependent variables for the two conditions, we

elicited predictive judgments (i.e., how well one of the first events predicted the second;

Waldmann, 2001). Thus, in the Predictive condition, participants were asked, "How well

does the [color] button being pressed predict whether or not the light turned on?" In the

Diagnostic condition, participants were asked, "How well does the [color] light turning

on predict whether or not the button had been pressed?" Judgments were made on a scale

ranging from 0 (labeled as "Not at all a predictor") to 100 (labeled as "A perfect

predictor"). These responses were obtained for both the observed and the unobserved

events.
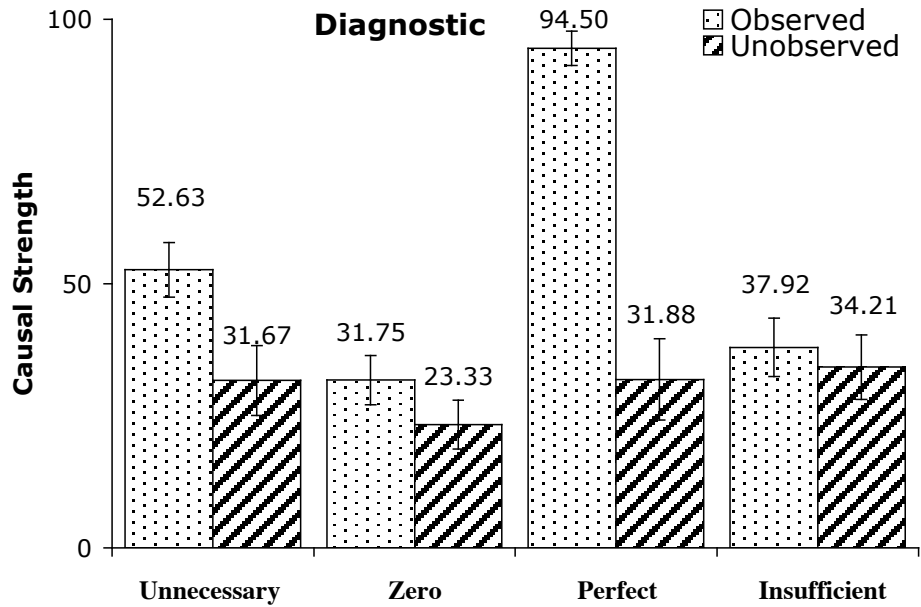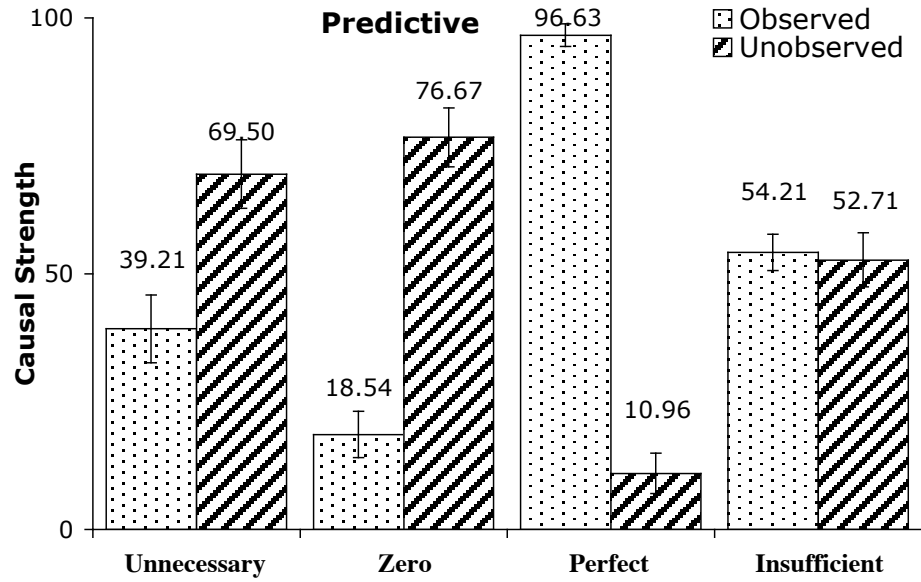
Figure 18 - Results from Experiment 6. The results in the Predictive condition mirror those found in the other experiments. The results in the Diagnostic condition look quite different. RW cannot account for this difference.

Participants' mean ratings for both the observed and the unobserved events can be seen in Figure 18. To investigate how causal role influenced judgments of the unobserved event, we performed a 2 (Condition: Predictive / Diagnostic) X 2 ($\overline{Cue} \cdot Outcome$: present/absent) X 2 ($Cue \cdot \overline{Outcome}$: present/absent) ANOVA with repeated measures on the last two factors over participants' ratings on the unobserved event. The critical question was whether the Predictive /Diagnostic manipulation would influence judgments. This analysis revealed a significant main effect of condition (F(1,46)=16.28, p<.0005) and this factor interacted with both the presence of $\overline{Cue} \cdot Outcome$ (F(1,46)=46.12, p<.0001) and $Cue \cdot \overline{Outcome}$ (F(1,46)=14.06, p<.001). That is, the causal role of events had a significant effect on judgments.

To better understand this pattern of results, we performed individual 2 ($\overline{Cue} \cdot Outcome$: present/absent) X 2 ($Cue \cdot \overline{Outcome}$: present/absent) ANOVAs for each condition. For the Predictive condition, the main effect of $\overline{Cue} \cdot Outcome$ observations was significant (F(1,23)=59.83, p<.0001) because systems including $\overline{Cue} \cdot Outcome$ observations elicited higher unobserved cause judgment (M = 73.08, SD = 30.38) than those not including $\overline{Cue} \cdot Outcome$ observations (M = 31.83, SD = 30.97). In other words, the previous effect of unexplained effects was replicated. The main effect of $Cue \cdot \overline{Outcome}$ observations was also significant (F(1,23)=18.78, p<.0005) because conditions that included $Cue \cdot \overline{Outcome}$ observations elicited higher unobserved cause judgments (M =64.69, SD = 29.42) than those that did not (M = 40.23, SD = 39.76). The interaction between these factors was also significant (F(1,23)=15.48, p<.001). For the

Diagnostic condition, we again performed a 2 ($\overline{Cue \cdot Outcome}$: present / absent) X 2 ($Cue \cdot \overline{Outcome}$: present / absent) ANOVA, but found no significant effects, all p's > .2.

These analyses make it clear that, contrary to the predictions of RW, judgments of unobserved events are greatly influenced by the manipulated causal role. In the Predictive condition, judgments of the unobserved cause varied with the contingency as predicted by BUCKLE. In the Diagnostic condition, however, no such effect was found. Instead, judgments were uniformly low regardless of observed contingency, suggesting that people do not spontaneously make inferences about unobserved effects in the same way they do about unobserved causes. Indeed, the lack of learning about unobserved effects is reasonable given that there appears to be no basis for making educated judgments about the unobserved effects.

To summarize, Experiment 6 investigated whether learning about unobserved events is a general feature of learning, as one would assume given RW's representation, or whether inferences about likelihood and strength are sensitive to causal role. Confronted with a diagnostic learning situation (i.e., effects predicting their causes), participants' behavior deviated significantly from that in the Predictive condition (and from the results of experiments reported above). From an associative perspective, this difference is inexplicable because causal role should be irrelevant for learning. In contrast, causal role is critical for BUCKLE's inferences. For example, the likelihood of an effect occurring after its cause cannot be equated with the likelihood of a cause preceding its effect. The results of Experiment 6 suggest that causal role is an important determinant of learning about unobserved, alternative causes.

One critical aspect of the causal learning process described by BUCKLE is that causal strength estimates are updated in a sequential manner as each observation is made. This approach contrasts with approaches that compute causal strength over all available trials once enough observations have been accumulated (e.g., Cheng, 1997; Busemeyer, 1991; White, 2002). According to BUCKLE, however, people's inferences about the likelihood of the unobserved cause changes as a function of the variables q_u, and q_o, which change over the course of learning. An interesting consequence of this is that the order in which observations are encountered should influence the final causal strength estimates because identical observations will be interpreted differently depending on q_u and q_o, which in turn would change beliefs about q_u and q_o (e.g., Dennis & Ahn, 2001).

To demonstrate this effect, we used the set of trials summarized in the top panel in Figure 19. This set of trials was divided into two blocks. One of the blocks contained unexplained effects ($\overline{OE}$) and the other did not. These two blocks could be ordered in one of two ways; the block containing unexplained effects could be presented either first (early-unexplained-effect condition) or second (late-unexplained-effect condition) as shown in the bottom panel in Figure 19. Note that, because the only manipulation was the order of the two blocks, participants always saw the same set of observations by the end of the sequence. Thus, any differences between conditions cannot be a result of the number or type of trials observed.

BUCKLE predicts that the causal strength judgments will differ between the two orderings. To see why this is, consider the early-unexplained-effect condition. During the first block of this condition, the unexplained effects will lead to the unobserved cause

being perceived as strong (as illustrated in Experiment 2). When the second block

(without unexplained effects) is encountered, the now-strong unobserved cause will be

interpreted as covarying with the effect (as illustrated in Experiment 5). For instance, a

learner would believe that the unobserved cause would likely be present during OE trials

but absent in $O\overline{E}$ trials. These inferences would further increase the strength of the

unobserved cause.

In contrast, consider the late-unexplained-effect condition in which the

	E	$\overline{E}$
O	8	4
$\overline{O}$	4	8

Early-unexplained-effect Condition				Late-unexplained-effect Condition		
	E	$\overline{E}$			E	$\overline{E}$
O	4	0		O	4	4
$\overline{O}$	4	4		$\overline{O}$	0	4

	E	$\overline{E}$			E	$\overline{E}$
O	4	4		O	4	0
$\overline{O}$	0	4		$\overline{O}$	4	4
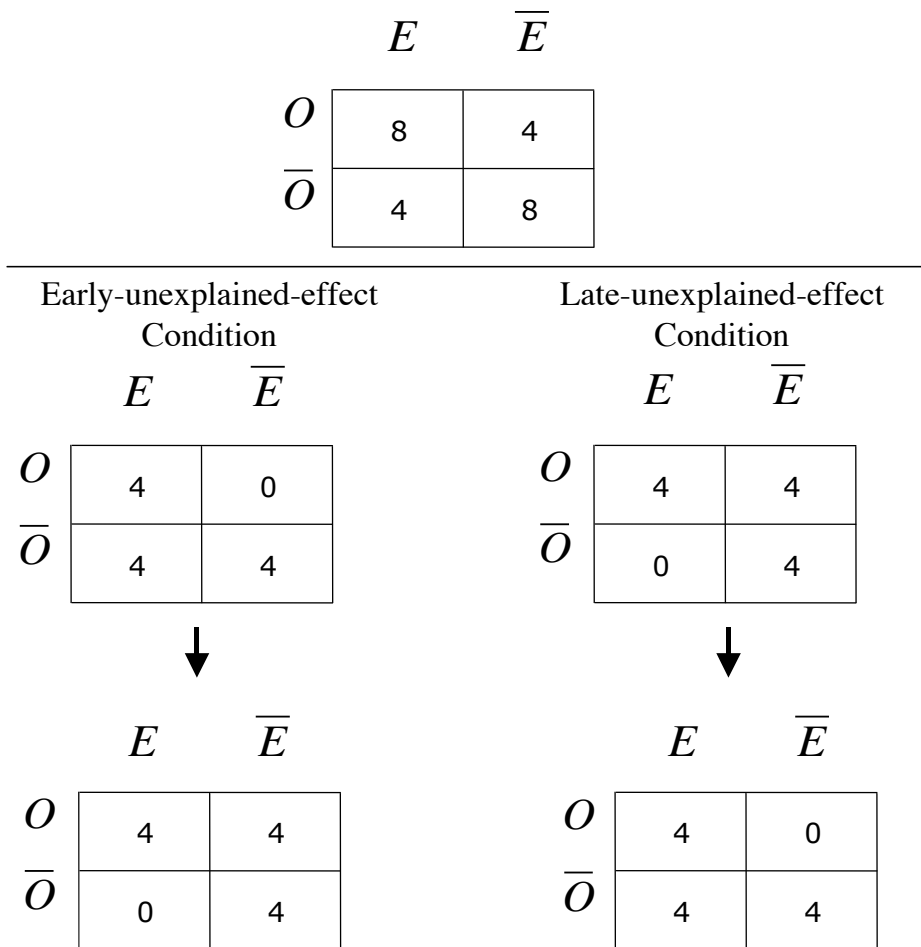
Figure 19 - Illustration of the design used in Experiment 7. The two sets of contingency tables in the bottom panel each show the order in which participants received two blocks of trials in each condition. Each ordering results in the identical contingency as described by the table at the top.

unexplained effects are encountered at the end. In this situation, at the end of the first half, the unobserved cause will be perceived as weak (as illustrated in Experiment 2). Only when the second block (with unexplained effects) is encountered will the perceived strength of the unobserved cause begin to increase. Thus, only the second block in this situation will lead to the unobserved cause being perceived as strong. However, compared to the early-unexplained-effect condition, there are far fewer trials remaining, and thus the belief in a strong unobserved cause will be reinforced less. Thus, the unobserved cause should be perceived as stronger when encountering unexplained effects in the first block than when encountering them in the second block.

Method

Participants

Fifty Vanderbilt University undergraduates participated for partial fulfillment of course credit.

Materials

The stimulus materials were similar to Experiment 2. The statistical properties of the system are summarized by the cell frequencies illustrated in Figure 19.

Design and procedure

The sole manipulation in this experiment was the order in which trials were presented to participants. There were two orderings used, each of which consisted of two blocks. One block contained $O\overline{E}$ trials but not $\overline{O}E$ trials. The other contained $\overline{O}E$ trials but not $O\overline{E}$ trials. In the early-unexplained-effects condition, participants first saw the block containing $\overline{O}E$ trials followed by the block containing $O\overline{E}$ trials. In the late-

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unexplained-effects condition, participants saw the two blocks in the reverse order. Although the set of trials was made of two blocks, there was nothing noting the change from one block to the other, and as far as participants were concerned, they were experiencing a continuous stream of observations.

The procedure of Experiment 7 was the same as in Experiment 2A. Thus, after completing observations, participants were asked to estimate on how many of 100 new tests in which a target button was pressed they would expect the light to turn on. Each subject saw both orders instantiated in different colored buttons, and the orders were counterbalanced across participants.

Results and Discussion

As summarized in Figure 20, participants gave a significantly higher rating in the unobserved cause in the early-unexplained-effects condition (M = 73.50, SD = 25.90) than in the late-unexplained-effects condition (M = 61.66, SD = 27.79), t(49)=2.89, p < .01, even though they observed identical contingency.

We used BUCKLE to simulate each of the conditions used in Experiment 7 by presenting the model with the exact same set of observations in the exact same order that participants received them. As expected, BUCKLE predicts a difference between the perceived strength of the unobserved cause (see Figure 20). The unobserved cause was predicted to be higher in the early-unexplained-effects condition (q_u=69.19) than in the late-unexplained-effects condition (q_u=64.28). These estimates accounted for 91% of the variance in participants' causal judgments and resulted in an RMSD of 5.79.

In terms of methodology, Experiment 7 also provides converging evidence for BUCKLE. Except for in Experiment 1, participants in the previous experiments were forced to provide estimates on unobserved causes. Although Experiment 1 provided strong evidence that participants were willing to provide judgments of unobserved causes, Experiment 7 demonstrates, using a different method, that people make
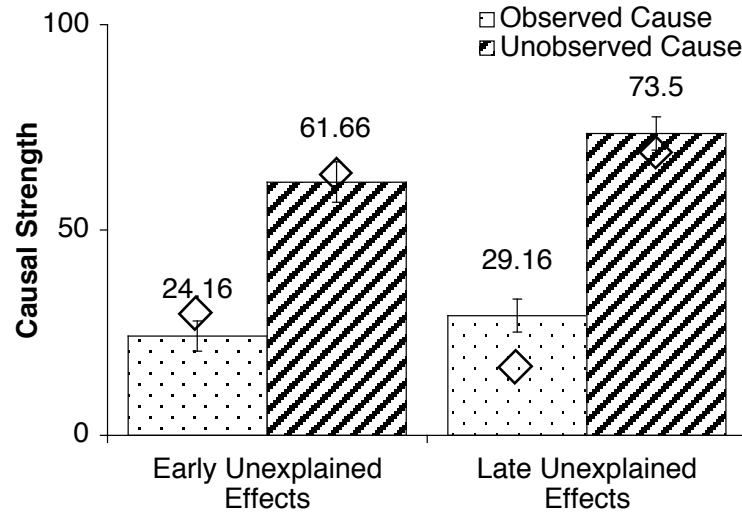
Figure 20 - Causal strength judgments from Experiment 7. Error bars indicate standard error. The diamonds represent BUCKLE's estimates.

spontaneous inferences about unobserved causes. That is, the results from Experiment 7 imply that people spontaneously made inferences about the unobserved cause during the first block even when they were not prompted to make such judgments. These covert judgments (e.g., beliefs) then influenced their interpretations of later observations, resulting in the order effect.

Experiment 8: The Influence of $O\overline{E}$ Observations

As demonstrated so far, $\overline{O}E$ observations (or unexplained effects) act to increase the perceived strength of the unobserved cause, because $\overline{O}E$ observations necessarily imply that the unobserved cause has caused the effect on that occasion. One might suppose that $O\overline{E}$ observations would act in the opposite manner. When encountering an $O\overline{E}$ observation, the learner might conclude that the unobserved cause prevented the effect. Such an interpretation should act to decrease the perceived strength of the unobserved cause.

According to BUCKLE, this is not necessarily the case for the following reason. Assuming that o is generative and u is preventative, the likelihood computations used by BUCKLE assume that $O\overline{E}$ observations occur for two separate reasons (see Equation A11 in Appendix A). First, as just mentioned, $O\overline{E}$ observations could occur when the effect is prevented from occurring by a preventative unobserved cause. Second, $O\overline{E}$ observations could occur because the observed cause itself is not entirely sufficient to bring about the effect. This latter interpretation should leave the state (i.e., present vs. absent) of the unobserved cause relatively ambiguous and thus reduce the amount of learning taking place during $O\overline{E}$ observations.

The suggestion that $O\overline{E}$ observations do not necessarily lead to the perception of a preventative unobserved cause has already been supported by the results of Experiment 2. Though the Insufficient condition included $O\overline{E}$ observations, our participants judged the unobserved cause to be somewhat generative (M = 27.25, SD = 35.56 in Experiment 2A) rather than preventative. However, BUCKLE predicts that, if it were established that the observed cause was sufficiently strong cause, then $O\overline{E}$ observations should indicate the influence of a preventive unobserved cause. More specifically, the term, $\{P(o) \bullet (1 - q_o) \bullet [1 - P(u \mid o)]\}$, from the denominator of Equation A11 in Appendix A will decrease, and increase the likelihood of the unobserved cause being present. Because the unobserved cause is likely present in the absence of the effect, q_u will decrease (i.e., it will become a stronger preventative cause; see the simulations below).

To test this prediction, Experiment 8 utilized two phases of training. During the first phase, participants learned that the observed cause was a strong cause of the effect. In the second phase, participants were exposed to $O\overline{E}$ observations. According to BUCKLE, the first phase should decrease the likelihood that the observed cause was insufficient to bring about the effect and increase the likelihood that the unobserved cause exerted a preventative influence in the second phase. Thus, this paradigm should allow $O\overline{E}$ result in preventative judgments of the unobserved cause.

This prediction is consistent with the results from Schulz et al. (2005) discussed earlier in the introduction. To reiterate, they found that preschoolers perceived an unobserved cause as preventative after encountering $O\overline{E}$ observations. Schulz et al. argued that this occurs because preschoolers believe that whenever a cause is present, an effect must occur. Under this belief, the only way $O\overline{E}$ could be possible is when there is

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a preventive alternative cause. This belief is essentially what we intended to induce from adult participants during the first phase of training in Experiment 8.

Method

Participants

Forty-four Yale University undergraduates participated for partial fulfillment of course credit. Each participant was randomly assigned to either the Unnecessary condition (N=22) or the Insufficient condition (N=22).

Design and Procedure

The set of trials for each system was divided into two phases. The first phase was designed to increase the perceived strength of the observed cause without changing beliefs about the unobserved cause. During the first phase (20 trials), both of the causes were observable. The cause that was to be unobserved in the second phase (see below) was explicitly noted to be absent on every trial (see Figure 21), whereas the other cause varied across trials with ΔP of .8.

The second phase of each system was similar to previous experiments; the unobserved cause was unobserved and the observed cause remained observed. This phase consisted of 12 trials. The contingencies between the observed cause and the effect in the second phase differed depending on the conditions as follows.

FKR-813 not taken.

KDN-568 taken.

Weight increased.
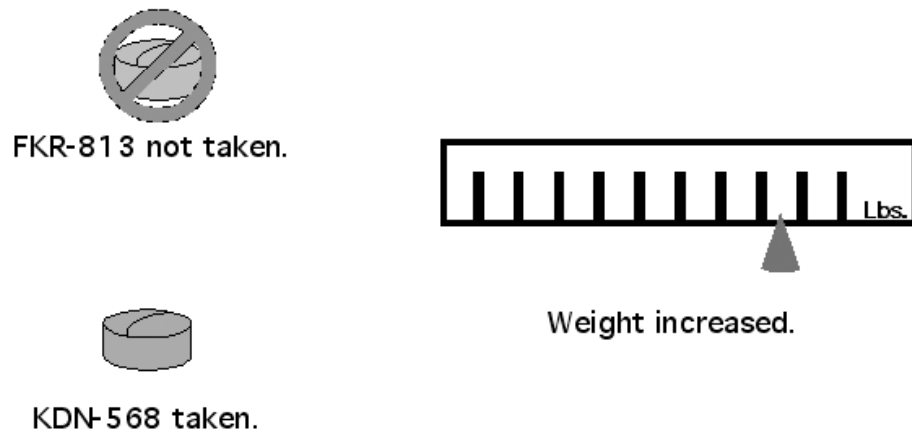
Figure 21 - A sample trial used in the first phase of Experiment 8. One of the causes (the bottom one) is observed throughout the entire experiment. The other cause (the top one) is observed and constantly absent in the first phase. In the second phase, this cause will become unobserved just as in previous experiments.

In the Insufficient condition, there were four trials of each of $O\overline{E}$, OE, and $\overline{OE}$ observations. In the Unnecessary condition, there were four trials of each of $\overline{OE}$, OE, and $\overline{OE}$ observations. As explained earlier, it is predicted that after having learned in the first phase that the observed cause is a strong generative cause, participants in the Insufficient condition would be more likely to believe that the unobserved cause is a preventative cause. The Unnecessary condition was included to rule out the possibility that any "probabilistic" observation (i.e., $\overline{OE}$ or $O\overline{E}$) results in preventative unobserved cause judgments. To the contrary, $\overline{OE}$ in the second phase of the Unnecessary condition is predicted to make participants believe that the unobserved cause is a generative cause.

In addition, all participants were presented with a system called First-Phase Only that only included the trials from the first phase only. This condition allowed us to measure causal beliefs resulting from the first phase of trials per se and to better interpret

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the results of the second phases. Because the First-Phase Only condition was the only condition to not include a second phase (the "unobserved" cause was always observed) it was always presented last so as to not disrupt participants' expectations about the subsequent conditions.

After viewing the entire set of trials for a system (i.e., both the first and the second phases), participants were asked to rate the causal strength of the observed and unobserved cause separately. The response scale ranged from –100 (e.g., prevents the effect) to 100 (e.g., causes the effect) with zero labeled as "Has no influence". Once participants finished making all responses for the first system, they were presented with the second system in the same condition and proceeded as before. The two systems in the same condition differed only with respect to the stimulus materials.

Stimuli

The electric systems used as stimuli previously are more appropriate for learning generative causal relations (i.e., buttons normally cause lights to turn on rather than preventing lights from being turned on). To create a more plausible preventative scenario, stimuli in Experiment 8 consisted of novel medications and physical side effects. The medications names were novel strings of letters and digits (e.g., DJE-143). The effects were "salivation increased/did not increase" and "dilation increased/did not increase" in the Insufficient condition and "blood pressure increased/did not increase" and "weight increased/did not increase" in the Unnecessary condition. During the trials in the second phase, the unobserved cause was indicated by a large question mark over the medication as in the previous experiments.

Participants' causal judgments can be seen in Figure 22. There was no difference between two systems in the same condition, so the analyses reported below are based on the average ratings across the two systems in the same condition. The critical finding was that the unobserved cause was judged to be significantly preventative (negative) in the Insufficient condition (M = –12.27, SD = 23.51; one-sample test against zero: t(21)=2.45, p<.05; paired test against the First-Phase Only condition: t(19)=2.57, p<.05[15]). This result is compatible with those of Schulz, et al. (2005). In contrast, judgments of the unobserved cause in the Unnecessary condition were significantly positive (M = 34.77, SD = 23.25; one-sample test against zero: t(21)=7.01, p<.0001;
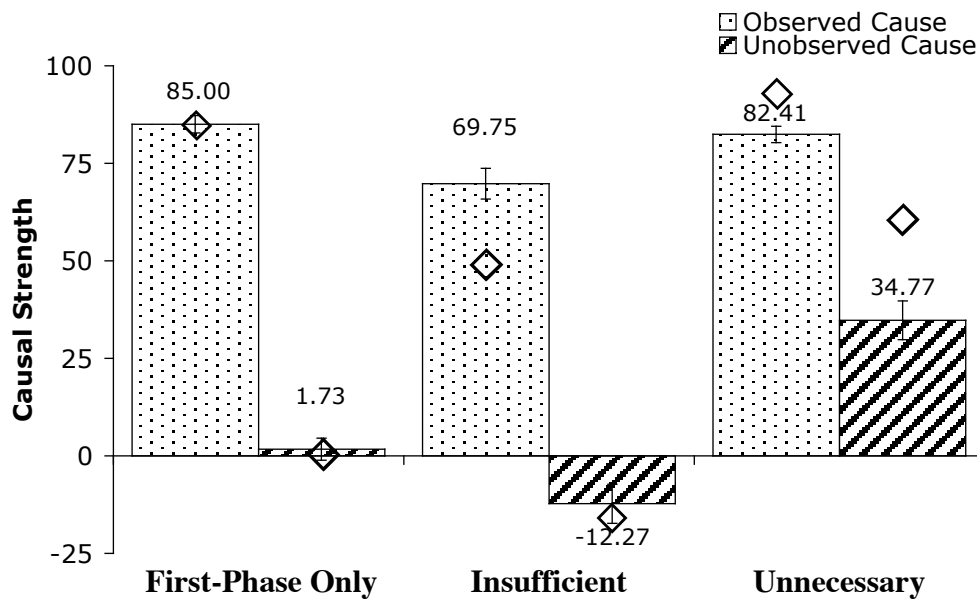
Figure 22 - Causal strength judgments from Experiment 8. Error bars indicate standard error. The diamonds represent BUCKLE's estimates.

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[15] A computer error resulted in the loss of data from the First-Phase Only condition for two subjects.

paired test against the First-Phase Only condition: t(19)=3.04, p<.01). Furthermore, judgments of the observed cause in the Insufficient condition were significantly lower than that in the Unnecessary condition (t(42)=2.82, p<.01), indicating that $O\overline{E}$ observations decreased judgments of the observed cause.

Simulating Experiment 8

We used BUCKLE to simulate Experiment 8 by presenting the model with the exact same set of observations in the exact same order that participants received them. We fit α_o to participants' judgments from the First-Phase Only condition (because causal strength judgments of u were near zero, attempting to fit α_u would have resulted in overfitting). The best fitting value of α_o was then used for both α_o and α_u when simulated the Insufficient and Unnecessary conditions. The results can be seen in Figure 22. BUCKLE accounted for 91% of the variance and resulted in an RMSD of 12.36. The first result of note is that BUCKLE accounts for the difference in unobserved cause's strength in the two conditions. The final value for q_u was –15.96 in the Insufficient condition but 60.18 in the Unnecessary condition. The second finding is that, unlike all previous simulations, BUCKLE's causal strength estimate was preventative (i.e. negative) in the Insufficient condition.

It should also be noted that, like participants' judgments, BUCKLE's estimate of the unobserved cause was only weakly preventative in the Insufficient condition. According to BUCKLE, during an $O\overline{E}$ observation, if o is generative and u is preventative (i.e., $q_o>0$, $q_u<0$), P($u|o,e$) increases as q_o increases or q_u decreases (i.e., becomes increasingly preventative). BUCKLE assumes that q_u is zero at the beginning of

Experiment 8's second phase and thus q_u does not act to substantially increase the likelihood of u. However, q_o should be high at the beginning of the second phase. Shouldn't this increase the likelihood of u (and thus decrease q_u)? According to BUCKLE's likelihood computation (see Appendix A), even when q_u is small (e.g., -.01), and q_o is large (e.g., 0.9), the likelihood of u will be only slightly greater than chance (e.g., .52). It is only when the perceived strength of the observed cause is near maximal that $O\overline{E}$ observations will lead to large negative values of q_u (e.g., when q_o=0.999 and q_u=-.01, the likelihood of u is .92). Thus, BUCKLE intuitively suggests that the strength of the observed cause will predict the magnitude of the influence of $O\overline{E}$ observations on unobserved cause judgments.

Thus, according to BUCKLE, even when the observed cause is reasonably strong, $O\overline{E}$ observations remain somewhat ambiguous. Because of this, even in the Insufficient condition BUCKLE allows the possibility that the observed cause was insufficient to bring about the effect. This interpretation causes BUCKLE to decrease the perceived strength of the observed cause. Thus, just as was found in the current study, BUCKLE argues that the observed cause in the Insufficient condition ($q_u = 49.05$) should be perceived as weaker than in the Unnecessary condition (q_u =93.04; see Figure 22).

Summary

Experiment 8 illustrated that $O\overline{E}$ observations do not necessarily lead learners to perceive the unobserved cause as preventative. BUCKLE argues that this is because $O\overline{E}$ observations have ambiguous implications for unobserved causes. The current experiment included a pre-training phase designed to minimize this ambiguity. As

predicted, with the pre-training, participants were able to use $O\overline{E}$ observations to infer a preventative influence of an unobserved cause. In addition, BUCKLE mirrored more subtle elements of participants' behavior. The $O\overline{E}$ observations led to small decreases in the perceived strength of the observed cause. Perhaps more surprising is the fact that $O\overline{E}$ observations led to only weakly preventative causal judgments. BUCKLE argues that this again occurs because of the ambiguity inherent in $O\overline{E}$ observations. Only when the observed cause is near-absolutely sufficient (perhaps as in the beliefs of preschoolers, Schulz, et al., 2005) will the unobserved cause be perceived as strongly preventative.

CHAPTER VI

GENERAL DISCUSSION

The model proposed here, BUCKLE, describes a simple two-step process for learning in the presence of unobserved causes. Each of the two steps involves a probabilistic inference about the likelihood of an event occurring. Each of these steps occurs on every learning occasion and together allows causal beliefs to be modified over the course of learning. The first step is to compute how likely the unobserved cause is to be present. This inference is made using both information available in the environment (e.g., the state of observed causes and effects), as well as beliefs and assumptions (e.g., the belief about the current strength of the observed cause). After this inference is made, there is no longer any missing information; all causes are believed to be present with some likelihood. The second step is to learn about the underlying casual relationships. To do this, BUCKLE makes a prediction about how likely the effect is to be present given the currently available information (e.g., the state of observed and unobserved causes and effects and beliefs and assumptions). This inference allows BUCKLE to compare its beliefs about how the world operates (i.e., the likelihood inference) with the actual operation of the world (i.e., whether the effect actually occurred or not). Errors in this step's inference then form the basis of learning. Despite its relative simplicity, BUCKLE appears to accurately capture a significant variety of aspects of people's cause learning.

Given a variety of statistical environments, BUCKLE successfully accounted for participants' causal strength judgments. In particular, BUCKLE anticipated the substantial influence of unexplained effects ($\overline{OE}$ observations) on judgments of the unobserved cause, which in turn influenced judgments of the observed cause. BUCKLE also accounted for more subtle behavior. For example, when participants encountered $\overline{OE}$ observations, they did not necessarily judge the unobserved cause to be preventative. This finding is contrary to the predictions of several models and appears to contradict previously reported findings in children (Schulz, et al., 2005). However, given the details of BUCKLE's operation, we were able to speculate about why our result was obtained and what the underlying developmental difference might have been. This allowed us to design a situation that elicited preventative judgments, providing further evidence in support of BUCKLE's account and eliminating the apparent contradiction.

The ability to predict causal strength judgments is an obvious first requirement for any model of causal learning. Indeed, fits to such judgments have been the primary method of validating causal learning models (e.g., Buehner, et al. 2003; Lober & Shanks, 2000). Of course, the point of modeling any psychological process is to allow a deeper understanding of its operation. Fortunately, BUCKLE details a rich process that can be investigated in its own right. The most obvious byproduct of BUCKLE's process is the result from BUCKLE's first step: the inference about the likelihood of the unobserved cause. Again, using a variety of situations, our participants' estimates consistently mirrored those of BUCKLE on several different dimensions. For example, likelihood estimates were greatly influenced by the state of the observed cause and effect. However, as predicted by BUCKLE, even identical observations elicited reliably different

likelihood judgments under appropriate circumstances. This insight allowed us to produce what may be the most dramatic illustration of BUCKLE's process-oriented nature. While presenting participants with identical sets of trials, we were able to elicit predictably different causal strength judgments.

The Interchangeable Nature of BUCKLE

As with any model, there are many assumptions built into BUCKLE. Some of these assumptions are captured by the values of various unchanging parameters (see Table A1). However there are other, less obvious assumptions behind BUCKLE's operation demonstrated in this paper. For example, BUCKLE, as described so far, assumes that each cause has an independent influence on its effect. Such an assumption disallows causal interactivity that is often (if not always) present in the real world. For example, individually, gasoline and key-turning do not cause your car to start; both causes are needed together before the effect will occur.

Independent influence and causal interactivity describe qualitatively different ways in which causes may operate. In the language of causal maps (see Pearl, 2000 for a thorough review), this is a difference in parameterization. Independence and interactivity are only two possible parameterizations. In theory, there are an infinite number of parameterizations for any given set of causes and effects.

Fortunately, different parameterizations are easy to implement in BUCKLE. For example, given the assumption that causes exert independent influence on their effects, the expressions described in Appendix A and Equations 2-7 are appropriate to compute the desired likelihood. However, if causes do not exert independent influence on their

effects, different expressions are needed to compute the same likelihood. Take the example of computing the likelihood of the unobserved cause (BUCKLE's first step). Equation 1 can still be used, coupled with a specification of the different ways in which each type of observation can occur and their associated probabilities. This information is precisely what is described by a given parameterization. Thus, if BUCKLE were used to simulate learning in a domain where causal interactivity were assumed, the expressions used to elaborate Equation 1 (i.e., Appendix A) would need to be rewritten to reflect the change in parameterization. The same is true of BUCKLE's second step. Equations 2-7 provide the appropriate way to compute the likelihood of the effect under a noisy-OR/noisy-AND-NOT parameterization. However, if it were discovered that a different parameterization better described people's beliefs, the relevant expressions could be substituted.

Causal interactivity is not the only alternative parameterization that is psychologically plausible. Recent work by Beckers and colleagues (Beckers, De Houwer, Pineno, & Miller, 2005; Beckers, Miller, De Houwer, & Urushihara, in press; see also Griffiths & Tenenbaum, 2005) has shed light on the inferences that are only allowable when the effect is a continuous variable (e.g., temperature). BUCKLE, as described here, assumes that effects (all events actually) are discrete. Clearly entirely different parameterizations are required when dealing with continuous effects. In such a situation, the likelihood of the effect is a distribution (or distributions) over possible values of the effect rather than single probabilities. In particular, Beckers argues that people generally assume that multiple effective causes lead to a stronger effect than a single effective cause. This assumption implies a particular class of parameterizations

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and, again, such parameterizations would imply a different set of expressions for the inferences prescribed by BUCKLE.

Note that BUCKLE is not alone in this flexibility. Any model that explicitly adopts a causal parameterization (e.g., MLE; Tenenbaum & Griffiths, 2005) will need to change its specific computations if the parameterization changes. As causal models have gained in popularity, more models are explicitly acknowledging their parameterization assumptions. What these models share is a commitment to describe inferences that are specifically causal. This means that the assumptions about causality are explicit in their computation. The same cannot be said for non-causal models of learning such as the constraint-satisfaction and RW models. For example, it is unclear why RW uses the sum of associative strengths as the basis of its error-correction algorithm[16]. However, the reason for this choice certainly does not have anything to do with how causes behave, because RW is claimed to be a general (i.e., non-causal) model of learning. Thus, models such as RW cannot be adapted to deal with different, specifically causal, assumptions. This rigidity formed the basis of Waldmann's (1996, 2000, 2001) studies as well as our own Experiment 6. Thus, unlike BUCKLE, these non-causal models have no way of predicting, a priori, that beliefs about parameterization should influence causal learning.

In this section, we have discussed various ways in which BUCKLE can be extended. Of course, arbitrary changes to BUCKLE could lead to an ultimately unfalsifiable model. Instead, we have attempted to illustrate that there is a principled

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[16] "The associative strength of the compound, V_{AX}, must somehow be specified in terms of the strengths of the components. The simplest assumption, and the one we will make here, is $V_{AX} = V_A + V_X$," (Rescorla & Wagner, 1972). Of course, if RW is treated as a model of causal learning, then its assumptions about parameterization may be revealed (Tenenbaum & Griffiths, 2005). However, as its advocates claim (Shanks, et al., 1996), RW is not a model of causal learning.

relationship between assumptions about particular learning environments and the details of BUCKLE's operation. To reiterate the core principles, BUCKLE predicts that learners typically represent an unobserved cause and learn about it using a two-step process. The first step is to infer the likelihood that the unobserved cause is present. Using the result of this inference, the strength of all causes, observed and unobserved, is adjusted.

Similarity between BUCKLE and Other Models of Learning

As briefly mentioned in the introduction, BUCKLE shares theoretical foundations with several existing models. The idea of error-correction learning has a long history in the psychology of learning (e.g., Widrow & Hoff, 1960). In particular, Rescorla and Wagner (1972) used error-correction to model behavior (as opposed to neural activity). The success of RW in accounting for learning behavior has led to its continued use even more than thirty years later. Of course, as just discussed, we believe that RW has features that limit its ability to account for a variety of causal learning phenomena.

To remedy these limitations, BUCKLE uses a slightly different model of causal learning based on suggestions of Danks, et al. (2003). This model has two related features that set it apart form RW. First, as just discussed, Danks, et al. (2003) method uses a noisy-OR parameterization to compute the likelihood of the effect. BUCKLE elaborates this by using either a noisy-OR parameterization when dealing with generative causes and a noisy-AND-NOT parameterization when one cause is preventative. This has two advantages. First, recent work (Cheng, 1997; Novick & Cheng, 2004; Griffiths & Tenenbaum, 2005; Danks, Griffiths, & Tenenbaum, 2003; Steyvers, Tenenbaum, Wagenmakers, & Blum, 2003) suggests that the noisy-OR/AND-NOT parameterization

better describes people's causal beliefs. Second, and perhaps more critically, building a model around the explicit notion of parameterization allows flexibility otherwise unthinkable. If noisy-OR/AND-NOT parameterizations only work in certain domains of causality, the model can simply be changed based on the new assumptions.

The second, related difference between RW and the proposal of Danks, et al. (2003) has to do with the quantity being computed. RW computes associative strength, a quantity that has been related to measurements of learning, but who's exact meaning is unclear. Associative strength is often related to, "magnitude or probability of conditioned responding" (Rescorla & Wagner, 1972) in conditioning experiments. In human causal learning studies, associative strength has been mapped onto a scale that includes preventative cause (negative associative strengths), ineffective causes (associative strengths of zero), and generative cause (negative associative strengths). Associative strength also tends to capture both aspects of a cause's sufficiency and its necessity (see Cheng, 1997 for details on the relationship between ΔP and RW). BUCKLE and the model of Danks, et al. (2003) do not learn associative strength. Instead, they learn a quantity that describes a cause's sufficiency. As mentioned in the introduction, this quantity is essentially what Pearl (2000) calls PS and what Cheng (2000) calls contextual causal power. The reason we chose this quantity actually has more to do with methodological cleanliness than any deep theoretical motivation. When eliciting judgments of associative strength, a vague question about "the degree to which A causes B" has traditionally been used (e.g., Wasserman, Kao, Van Hamme, Katagiri & Young, 1996). Recent work (Griffiths & Tennenbaum, 2005; White, 1992) has noted that the ambiguity of this question could elicit several, qualitatively different quantities. To elicit

the quantity computed by BUCKLE, a more concrete question is required, and thus we can be more assured that our participants' judgments reflect the quantity of interest.

The models discussed so far have all been related to BUCKLE's learning process (the second step) and not the first, inference step. More broadly, the idea of combining the two steps (filling in missing data and learning) has a long history in the field of machine learning. For example, Dempster, Laird, and Rubin (1977) describe an algorithm called EM (see also McLachlan and Krishnan, 1997; Tanner, 1996) that is able to successfully estimate parameters even when there are missing data. Like BUCKLE, EM consists of two, repeated steps. The first step (the E step) is to compute a likelihood distribution over possible values of the missing data. In the current studies, this step would compute the likelihood that the unobserved cause was present or absent on all trials. This step essentially acts to "fill in" missing data. The second step (the M step) is to find the set of parameters that maximizes the distribution computed in the first step. In the current studies, this would be equivalent to computing the MLE of q_u and q_o given the observed data as well as the data inferred in the first step. These two steps are repeated until parameter estimates converge. EM has been shown to successfully converge on locally maximal parameter estimates (Dempster, Laird, and Rubin, 1977 provided the proof).

Though BUCKLE and EM share a conceptual approach to the problem of missing data, there are several differences. The largest difference is that EM performs its two steps over the entire dataset simultaneously whereas BUCKLE performs its two steps on one observation at a time. As a result, EM cannot begin learning until all the data are collected. Thus, EM describes an inference that requires multiple iterations before

settling (much like the constraint-satisfaction network utilized in the current study) whereas BUCKLE describes a trial-by-trial learning that allows beliefs to be modified in a piecemeal fashion. This essentially prohibits EM from dealing with the dynamics of learning found in several of the current experiments. For example, because EM ignores trial order, it would not demonstrate the order effects found in Experiment 7.

There have been attempts to modify EM to be incremental (i.e., dealing with one data item at a time). The motivation for this effort generally comes, not from a desire to be psychologically plausible, but from a need to find efficient algorithms in the face of massive datasets (e.g., data mining large databases). For example, Neal and Hinton (1998) demonstrate that a modified EM algorithm requiring only one data item at a time is equivalent to the canonical version of EM and is thus guaranteed to converge in the same manner (though faster). However, even this incremental variant was designed to be run on a complete dataset. For example, Neal and Hinton (1998) state that, "data items might be selected for updating in the E step cyclically, or by some scheme that gives preference to data items for which [the likelihood distribution] has not yet stabilized". Thus, the advantage of this algorithm is not in allowing for incremental learning, but in its ability to learn from complete datasets more efficiently.

Others have suggested EM algorithms that are truly incremental (i.e., require only one forward pass through the data; Bradley, Fayyad, & Reina, 1998; Suematsu, Maebashi, Hayashi, 2004). However, these algorithms uniformly require considerable computation and complex storage (e.g. clustering). BUCKLE is certainly a member of this family of algorithms. BUCKLE's certainly two steps mirror those of EM. However, BUCKLE is also certainly a simplistic variant of EM. The most obvious example of this

is the fact that BUCKLE does not maximize its parameter estimates in the second step. Instead, it uses the more obviously psychologically plausible notion of error-correction. Nor does BUCKLE labor over the inference of its first step. It simply makes its best guess given the available information and moves on. These features may no doubt lead BUCKLE's behavior to deviate from more complex EM variants, however, future work is required to more thoroughly explore this speculation.

Conclusion

Knowledge about alternative causes is crucial in inducing causation from covariation. Previous models of causal learning have acknowledged the importance of alternative causes but have only made simplified assumptions about learning of unobserved alternative causes. The current paper presents empirical evidence suggesting that people willingly make sophisticated inferences about unobserved causes. We have presented a new model of causal induction that delineates bases of such inferences and demonstrated that BUCKLE could explain unobserved causal learning better than existing models. As a result, BUCKLE could explain observed causal learning under confounded situations better than existing models.

Because the current paper is the first thorough investigation of a model that is specifically designed to deal with unobserved causal learning, most of our discussion and experiments were focused on unobserved causal learning. Future research can further examine other possible consequences of unobserved causal inferences on learning of observed causes. We have also discussed many ways in which BUCKLE can be flexibly expanded to accommodate causal environments different from those used in our current

experiments. Further research is needed to test the psychological validity of these

extensions.

Appendix A: Likelihood Computations for BUCKLE

- o represents the observed cause and equals 1 when present, 0 when absent.
- u represents the unobserved cause and equals 1 when present, 0 when absent.
- e represents the effect and equals 1 when present, 0 when absent.
- q_u and q_o are the causal strengths of u and o respectively.
- $P(u|o=0)$ and $P(u|o=1)$ are the prior likelihoods of u occurring.
- If a likelihood equation ever results in an undefined value (i.e., when $P(o,e)=0$), then $P(u|o,e)$ is assigned the value of the prior (e.g., $P(u|,o)$).

Assuming $q_o \geq 0$, $q_u \geq 0$:

When $o=0$ and $e=0$, it may be because:
- o is absent, u is absent
- o is absent, u is present and fails to produce e

$$P(u|o=0,e=0) = \frac{[1-P(o)] \bullet P(u|o=0) \bullet (1-q_u)}{\{[1-P(o)] \bullet [1-P(u|o=0)]\} + \{[1-P(o)] \bullet P(u|o=0) \bullet (1-q_u)\}} \quad \text{(A1)}$$

When $o=0$ and $e=1$, it may be because:
- o is absent, u is present and produces e

$$P(u|o=0,e=1) = \frac{[1-P(o)] \bullet P(u|o=0) \bullet q_u}{[1-P(o)] \bullet P(u|o=0) \bullet q_u} = 1 \quad \text{(A2)}$$

When $o=1$ and $e=0$, it may be because:
- o is present and fails to produce e, u is absent
- o is present and fails to produce e, u is present and fails to produce e

$$P(u|o=1,e=0) =$$

$$\frac{P(o) \bullet (1-q_o) \bullet P(u|o=1) \bullet (1-q_u)}{\{P(o) \bullet (1-q_o) \bullet [1-P(u|o=1)]\} + [P(o) \bullet (1-q_o) \bullet P(u|o=1) \bullet (1-q_u)]} \quad \text{(A3)}$$

When $o=1$ and $e=1$, it may be because:
- o is present and produces e, u is absent
- o is present, u is present, either o or u produces e

$$P(u|o=1,e=1) =$$

$$\frac{P(o) \bullet P(u|o=1) \bullet [q_o + q_u - (q_o \bullet q_u)]}{\{P(o) \bullet q_o \bullet [1-P(u|o=1)]\} + \{P(o) \bullet P(u|o=1) \bullet [q_o + q_u - (q_o \bullet q_u)]\}} \quad \text{(A4)}$$

Assuming $q_o < 0$, $q_u \geq 0$:

When $o=0$ and $e=0$, it may be because:
- o is absent, u is absent
- o is absent, u is present and fails to produce e

$$P(u \mid o = 0, e = 0) =$$
$$\frac{\left[1 - P(o)\right] \bullet P(u \mid o = 0) \bullet (1 - q_u)}{\left[(1 - P(o)) \bullet (1 - P(u \mid o = 0))\right] + \left[(1 - P(o)) \bullet P(u \mid o = 0) \bullet (1 - q_u)\right]} \quad (A5)$$

When $o=0$ and $e=1$, it may be because:
- o is absent, u is present and produces e

$$P(u \mid o = 0, e = 1) = \frac{\left[1 - P(o)\right] \bullet P(u \mid o = 1) \bullet q_u}{\left[1 - P(o)\right] \bullet P(u \mid o = 1) \bullet q_u} = 1 \quad (A6)$$

When $o=1$ and $e=0$, it may be because:
- o is present, u is absent
- o is present, u is present, either u fails to produce e or produces e but o prevents e

$$P(u \mid o = 1, e = 0) =$$
$$\frac{P(o) \bullet P(u \mid o = 1) \bullet \left[(1 - q_u) + (q_u \bullet q_o)\right]}{\left[P(o) \bullet (1 - P(u \mid o = 1))\right] + \left\{P(o) \bullet P(u \mid o = 1) \bullet \left[(1 - q_u) + (q_u \bullet q_o)\right]\right\}} \quad (A7)$$

When $o=0$ and $e=1$, it may be because:
- o is absent, u is present and produces e

$$P(u \mid o = 0, e = 1) = \frac{P(o) \bullet P(u \mid o = 0) \bullet q_u}{P(o) \bullet P(u \mid o = 0) \bullet q_u} = 1 \quad (A8)$$

Assuming $q_o \geq 0$ and $q_u < 0$:

When $o=0$ and $e=0$, it may be because:
- o is absent, u is present
- o is absent, u is absent

(i.e., observation is non-diagnostic)

$$P(u \mid o = 0, e = 0) =$$
$$\frac{(1 - P(o)) \bullet P(u \mid o = 0)}{\left[(1 - P(o)) \bullet P(u \mid o = 0)\right] + \left[(1 - P(o)) \bullet (1 - P(u \mid o = 0))\right]} = P(u \mid o = 0) \quad (A9)$$

When o=0 and e=1, it may be because:
- Impossible occurrence (i.e., observation is non-diagnostic)

$$P(u|\ o{=}0,\ e{=}1) \ = \ P(u\,|\,o=0) \qquad \text{(A10)}$$

When o=1 and e=0, it may be because:
- o is present and fails to produce e, u is absent
- o is present, u is present, o fails to produce e or produces e but u prevents e

$$P(u\,|\,o=1,e=0) =$$

$$\frac{P(o) \bullet P(u\,|\,o=1) \bullet \left[(1-q_o)+(q_u \bullet q_o)\right]}{\left[P(o) \bullet (1-q_o) \bullet (1-P(u\,|\,o=1))\right] + \left\{P(o) \bullet P(u\,|\,o=1) \bullet \left[(1-q_o)+(q_u \bullet q_o)\right]\right\}} \qquad \text{(A11)}$$

When o=1 and e=1 it may be because:
- o is present and produces e, u is absent
- o is present and produces e, u is present and fails to prevent e

$$P(u\,|\,o=1,e=1) =$$

$$\frac{P(o) \bullet q_o \bullet P(u\,|\,o=1) \bullet \left[1-(-q_u)\right]}{\left\{P(o) \bullet q_o \bullet \left[1-P(u\,|\,o=1)\right]\right\} + \left\{P(o) \bullet q_o \bullet P(u\,|\,o=1) \bullet \left[1-(-q_u)\right]\right\}} \qquad \text{(A12)}$$

Assuming $q_o < 0$, $q_u < 0$:

When o=0 and e=0, it may be because:
- o is absent, u is present
- o is absent, u is absent
(i.e., observation is non-diagnostic)

$$P(u\,|\,o=0,e=0) =$$

$$\frac{\left[1-P(o)\right] \bullet P(u\,|\,o=0)}{\left\{\left[1-P(o)\right] \bullet P(u\,|\,o=0)\right\} + \left\{\left[1-P(o)\right] \bullet \left[1-P(u\,|\,o=0)\right]\right\}} = P(u\,|\,o=0) \qquad \text{(A13)}$$

When o=0 and e=1, it may be because:
- Impossible occurrence (i.e., observation is non-diagnostic)

$$P(u|\ o{=}0,\ e{=}1) \ = \ P(u\,|\,o=0) \qquad \text{(A14)}$$

When o=1 and e=0, it may be because:
- o is present, u is present
- o is present, u is absent
(i.e., observation is non-diagnostic)

$$P(u \mid o = 1, e = 0) =$$

$$\frac{P(o) \bullet P(u \mid o = 1)}{\left[P(o) \bullet P(u \mid o = 1) \right] + \left\{ P(o) \bullet \left[1 - P(u \mid o = 1) \right] \right\}} = P(u \mid o = 1) \qquad \text{(A15)}$$

When $o=1$ and $e=1$, it may be because:
- Impossible occurrence (i.e., observation is non-diagnostic)

$$P(u \mid o=1, e=1) = P(u \mid o = 1) \qquad \text{(A16)}$$

Table A1

This table contains a list of the parameters used by BUCKLE along with descriptions of their nature and use.

Name	Initial Value	Source	Description
o	N/A	Input	Presence/absence of the observed cause on the current observation.
e	N/A	Input	Presence/absence of the effect on the current observation.
u	N/A	Input	Presence/absence of the unobserved cause on the current observation.
q_o	0	Learned	Causal sufficiency of the observed cause.
q_u	0	Learned	Causal sufficiency of the unobserved cause.
$P(u \mid o = 1)$	.5	Static	Prior likelihood of the unobserved cause being present during O observations.
$P(u \mid o = 0)$	.5	Static	Prior likelihood of the unobserved cause being present during $\overline{O}$ observations.
$P(o)$	.5	Static	Likelihood of the observed cause being present (does not influence computation).
α_o	N/A	Fit to data	Learning rate associated with the observed cause.
α_u	N/A	Fit to data	Learning rate associated with the unobserved cause.
β	.5	Static	Learning rate associated with the effect.

Table A2

This table contains the best fitting values of α_o and α_u for each experiment.

Experiment	α_o	α_u
2A	0.260	0.239
2B	0.280	0.199
5	0.204	0.185
7	0.119	0.235
8	0.413	0. 413
Mean (S.D)	0.249 (0.124)	0.268 (0.100)

Appendix B: Likelihood Computations for MLE

The MLE computes the combination of parameters most likely to have resulted in the observed data. To do so, we calculate the likelihood of encountering the set of observations. If the condition includes the set of observations, D, each containing N observations, the likelihood can be computed as follows:

$$L(D \mid q_o, q_u) = \prod_{i=1}^{N} P(D_i \mid q_o, q_u) \qquad \text{(B1)}$$

The likelihood of all possible combinations of q_o and q_u can then be computed and the pair that results in the greatest likelihood is the MLE.

To calculate $P(D_i \mid q_o, q_u)$, we will assume (as we did for BUCKLE) that causes combine in the manner of a noisy-OR/AND-NOT gate. The likelihood of each type of observation (i.e., $P(D_i \mid q_o, q_u)$) may be computed by combining the likelihood of each possible way in which that observation (i.e., D_i) could occur (see below). We assumed that $P(u \mid q_o, q_u)$ is a uniform distribution equal to 0.5, indicating complete uncertainty about the likelihood of the unobserved cause and that o is independent of u (i.e., $P(e \mid u=1) = P(e \mid u=0)$). As shown in the equation above, by iterating over the observed trial types and multiplying the individual likelihoods we can compute the overall likelihood of each pair of parameter values. Doing so for all possible combinations of parameter values allows us to find the pair that is most likely given the data.

When $o=0$ and $e=0$ it may be because:
- o is absent, u is absent
- o is absent, u is present and fails to produce e

$$P(o = 0, e = 0 \mid q_o, q_u) =$$
$$P(o = 0 \mid q_o, q_u) \bullet \left\{ P(u = 0 \mid q_o, q_u) + \left[P(u = 1 \mid q_o, q_u) \bullet (1 - q_u) \right] \right\} \qquad \text{(B2)}$$

When $o=0$ and $e=1$ it may be because:
- o is absent, u is present and produces e

$$P(o = 0, e = 1 \mid q_o, q_u) = P(o = 0 \mid q_o, q_u) \bullet P(u = 1 \mid q_o, q_u) \bullet q_u \qquad \text{(B3)}$$

When $o=1$ and $e=0$ it may be because:
- o is present and fails to produce e, u is absent
- o is present and fails to produce e, u is present and fails to produce e

$$P(o = 1, e = 0 \mid q_o, q_u) =$$
$$P(o = 1 \mid q_o, q_u) \bullet$$
$$\left\{ \left[(1 - q_o) \bullet P(u = 0 \mid q_o, q_u) \right] + \left[(1 - q_o) \bullet P(u = 1 \mid q_o, q_u) \bullet (1 - q_u) \right] \right\} \qquad \text{(B4)}$$

When $o=1$ and $e=1$ it may be because:
- o is present and produces e
- o is present, u is present and produces e

$$P(o = 1, e = 1 \mid q_o, q_u) =$$

$$P(o = 1 \mid q_o, q_u) \bullet \qquad \qquad \text{(B5)}$$

$$\left[q_o + \left(P(u = 1 \mid q_o, q_u) \bullet q_u \right) - \left(q_o \bullet \left[P(u = 1 \mid q_o, q_u) \bullet q_u \right] \right) \right]$$

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