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#### Beyond Markov: Accounting for Independence Violations in Causal Reasoning

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#### Abstract

Although many theories of causal cognition are based on causal graphical models, a key property of such models—the independence relations stipulated by the Markov condition—is routinely violated by human reasoners. Two accounts of why people violate independence are formalized and subjected to experimental test. Subjects' inferences were more consistent with a *dual prototype model* in which people favor network states in which variables are all present or all absent than a *leaky gate model* in which information is transmitted through network nodes when it should normatively be blocked. The article concludes with a call for theories of causal cognition that rest on foundations that are faithful to the kinds of causal inferences people actually draw.

The last 25 years has seen a dramatic increase in research asking how causal knowledge influences many acts of cognition, including reasoning, categorization, decision making, and learning. Theory in this area has been advanced by use of the formalism known as *causal graphical models* that provides a good first order approximation of human abilities. Nevertheless, there is now considerable evidence that a defining feature of these models—the Markov condition—is routinely violated by reasoners. This article describes these violations and then compares two new models that account for them, albeit in different ways. The diverging predictions of these models are then tested in a new experiment.

#### **Independence Violations in Causal Reasoning**

The causal networks in Figs. 1 and 2 provide two prominent examples of how human reasoners violate the independence relations associated with causal graphic models. Starting with the common cause network in Fig. 1 in which X is a common cause of  $Y_1$  and  $Y_2$ , whereas  $Y_1$  and  $Y_2$  are unconditionally dependent (e.g., one can reason from the presence of  $Y_1$  to the likely presence of X and then to the likely presence of  $Y_2$ ) the Markov condition stipulates that they are independent conditioned on X (i.e.,  $Y_1 \perp Y_2 | X$ ). In this case X "screens off" the Ys from one another (Pearl, 2000; Spirtes, 2000). Because it will be useful to characterize the sign and magnitude of the dependence between two variables, I define a function D (for "delta") that characterizes the difference in the probability of one variable given the presence or absence of another. Moreover, I define that difference in terms of probabilities transformed into log odds. For example,  $D(Y_1, Y_2)$  is defined as,

$$D(Y_1, Y_2) = logit (p(y_1^1 | y_2^1)) - logit (p(y_1^1 | y_2^0))$$
(1)

where  $y_i^x$  denotes  $Y_i = x$  (e.g.,  $y_1^1$  means  $Y_1$  is present). Because within-network causal relations will have the same

properties (e.g., have the same causal strength) in this work, symmetry entails that  $D(Y_1, Y_2) = D(Y_2, Y_1)$ , which will therefore be abbreviated D(Y).

Analogously,  $D(Y_1, Y_2 | x^1)$ —the difference in (the logit of) the probability of  $Y_1$  given the presence or absence of  $Y_2$ , conditioned on  $x^1$ —is defined as,

$$D(Y_1, Y_2 | x^1) = logit (p(y_1^1 | y_2^1, x^1)) - logit (p(y_1^1 | y_2^0, x^1))$$
(2)

and is abbreviated  $D(Y|x^1)$ .

The normative predictions for D(Y) and  $D(Y|x^1)$  are presented in the left chart of Fig. 1 assuming that the common cause graph is parameterized such that  $p(x^1) = .50$ , that the generative "causal power" associated with both  $X \rightarrow Y_1$ and  $X \rightarrow Y_2$  is .80, that there are weak alternative causes of  $Y_1$  and  $Y_2$  such that  $p(y_1^1|x^0) = p(y_2^1|x^0) = .20$ , and that multiple causal influences integrate according to a standard noisy-or function (Cheng, 1997). The figure illustrates the independence relations that characterize a common cause network: Whereas D(Y) > 0,  $D(Y|x^1) = 0$ , that is, the Ys are independent conditioned on  $x^1$ .



*Figure 1*. Normative predictions for the common cause model on the left and the corresponding empirical rating from Rehder & Waldmann (2015). Error bars are 95% confidence intervals.

The right chart of Fig. 1 illustrates how that independence relation is typically violated. Rehder & Waldmann (2015) instructed subjects on causal relations that formed a common cause model in the domain of economics, meteorology or sociology. For example, for economics subjects were told that "low interest rates causes small trade deficits"  $(X \rightarrow Y_1)$ and that "low interest rates causes high retirement savings"  $(X \rightarrow Y_2)$ . The variables senses involved in the causal links were varied (e.g., *high* rather than low interest rates sometimes played the role of X). Subjects were then presented with a series of inferences in which they were asked to rate (on a 0-100 scale) the probability of one variable conditioned on others. We found that the empirical analog of  $D(Y|x^1)$  (the difference between  $rating(y_i^1|y_j^0, x^1)$ ) and  $rating(y_i^1|y_j^0, x^1)$ ) was about 18 points (Fig. 1).



and the corresponding empirical ratings from Rehder & Waldmann (2015). Error bars are 95% confidence intervals.

Fig. 2 presents a common effect network in which X is now a common effect of  $Y_1$  and  $Y_2$ . For this network the independence relations are the reverse of those for a common cause network: Whereas  $Y_1$  and  $Y_2$  are unconditionally independent  $(Y_1 \perp Y_2)$  they become dependent conditioned on X. And, assuming that  $Y_1 \to X$  and  $Y_2 \to X$  are generative and operate independently then  $Y_1$  and  $Y_2$  exhibit an *explain*ing away relationship when X is present (the presence of  $Y_1$ makes  $Y_2$  less likely and vice versa). These predictions-D(Y) = 0 and  $D(Y|x^1) < 1$ -are presented in the left chart of Fig. 2 assuming the same parameterization as in Fig. 1 (i.e.,  $p(y_i^1) = .50$ , causal powers of .80, and  $p(x^1|y_1^0y_2^0) =$ .20). And the right chart exhibits how those relations are typically violated: Rehder & Waldmann found that D(Y) > 0. As an aside, note that, as predicted,  $D(Y|x^1)$  was significantly negative, although see Rottman & Hastie (2014) for evidence that explaining away is usually weaker than predicted by the normative model. The pattern of independence violations in Figs. 1 and 2 was also observed by Rehder (2014a) using a forced-choice task.

A number of rationalizations of these independence violations have been offered. For instance, Park & Sloman (2013) showed that those that arise with a common cause network are sometimes partly due to subjects' beliefs that the two causal links could be disabled by a common factor (also see Ali, Chater, & Oaksford, 2011; Lagnado & Sloman, 2004; Fernbach & Rehder, 2013; Mayrhofer & Waldmann, 2015; Rehder, 2014b; Walsh & Sloman, 2008). However, this account fails to explain the violations that occur with the common effect network in Fig. 2 (although it may explain why those violations were numerically about half the size of those in Fig. 1). Relatedly, Rehder & Burnett (2005) explained the large variety of Markov violations they observed by assuming that all variables were related by an underlying common cause (an assumption justified on the basis of the fact that the variables were features of a category; also see Rehder, 2014b). However, this account also fails to explain the results from Rehder & Waldmann (2015), which tested materials that were not category features.

To address these descriptive failures of the normative model, I present two new models of causal reasoning—the *leaky gate model* and the *dual prototype model*. For each I first show that it accounts for the independence violations shown in Figs. 1 and 2. Because the models make the same predictions for those simple networks, I describe two new causal networks for which their predictions diverge. An experimental test of those networks is then reported.

#### The Leaky Gate Model

The leaky gate model is based on the intuition that information flows along the directed edges of a causal network in a manner that is reminiscent of "spreading activation" accounts of memory—even in situations where that flow is normatively blocked. It accomplishes this by introducing additional statistical structure associated with every triple of variables A, B, and C in the network that are related such that A - B - C (where the edges represents a directed edge in either direction).

 $Y_1$ , X, and  $Y_2$  are so related in the networks in Figs. 1 and 2. The joint distribution specified by the leaky gate model for those networks is derived from the joint specified by the normative model under parameterization  $\theta$ , referred to as  $p_N(X, Y_1, Y_2 | \theta)$ . The leaky gate model's joint distribution,  $p_{LG}$ , augments  $p_N$  with an additional *energy function*,  $\epsilon_{XY_1Y_2}$  (Koller & Friedman, 2009),

$$log(p_{LG}(X, Y_1, Y_2|\theta, a)) \propto \epsilon_{XY_1Y_2} + log(p_N(X, Y_1, Y_2|\theta))$$
(3)

$$\epsilon_{XY_1Y_2} = \begin{cases} a, & X = Y_1 = Y_2 \\ 0, & \text{otherwise} \end{cases}$$
(4)

where *a* is a free parameter  $\ge 0$ .  $\epsilon_{XY_1Y_2}$  represents an expectation (whose magnitude is represented by *a*) that *X*, *Y*<sub>1</sub>, and *Y*<sub>2</sub> will tend to have the same value, that is, to be all present or all absent. Eq. 3 yields a proper joint distribution after exponentiation and normalization.

The introduction of  $\epsilon_{XY_1Y_2}$  is sufficient to reproduce the pattern of independence violations shown in Figs. 1 and 2. When a = .75 and the common cause network is instantiated with same parameters as in Fig. 1, the leaky gate model predicts  $D(Y|x^1) = .75$  rather than 0. For the common effect network in Fig. 2, it predicts D(Y) = 1.37 rather than 0.

The leaky gate model generalizes to more complex networks. Consider the elaborated common cause network in Fig. 3 in which the two effects  $(Y_1 \text{ and } Y_2)$  are themselves the causes of two other variables  $(Z_1 \text{ and } Z_2)$ . In this network, there are three triples of connected variables:  $Y_1 - X - Y_2$ ,  $X - Y_1 - Z_1$ , and  $X - Y_2 - Z_2$ . Thus, the leaky gate model's joint distribution for this network is,

$$log(p_{LG}(X, Y_1, Y_2, Z_1, Z_2 | \theta, a))$$
(5)  

$$\propto \epsilon_{XY_1Y_2} + \epsilon_{XY_1Z_1} + \epsilon_{XY_2Z_2}$$

$$+ log(p_N(X, Y_1, Y_2, Z_1, Z_2 | \theta))$$

$$\epsilon_{XY_1Z_1} = \begin{cases} a, & X = Y_1 = Z_1 \\ 0, & \text{otherwise} \end{cases}$$
(6)

$$\epsilon_{XY_2Z_2} = \begin{cases} a, & X = Y_2 = Z_2 \\ 0, & \text{otherwise} \end{cases}$$
(7)

and  $\epsilon_{XY_1Y_2}$  is given by Eq. 4.

The predictions for the elaborated common cause network are shown in Fig. 3 for both the normative and leaky gate model and a number inference types (it also includes the predictions of the dual prototype model presented in the next section). The top row of charts presents predictions regarding the independence of the  $Y_S - D(Y)$  and  $D(Y|x^1)$ -but also those regarding the independence of the  $Z_S -$ D(Z) and  $D(Z|x^1)$ . The bottom charts compare  $D(Z|x^1)$ with a new kind of inference, namely,  $D(Z|x^1y_1^1y_2^1)$  in



*Figure 3.* Predictions of the normative model, the leaky gate model, and the dual prototype model for the elaborated common cause network. The "•" in, say,  $D(\bullet)$  represents either Y or Z. Empirical results are shown in the last column. Error bars are 95% confidence intervals.

which  $Z_1$  and  $Z_2$  are screened off from one another by *three* variables  $(X, Y_1, \text{ and } Y_2)$  rather than one.

Using the same parameters as in Fig. 1, the normative model predicts that D(Y) > D(Z). Because the causal relations are probabilistic, the Ys provide more information about each other than the Zs. Nevertheless,  $D(Y|x^1) = D(Z|x^1) = 0$ , because knowledge of X renders the Zs independent along with the Ys. In contrast, the leaky gate model (a = .75) predicts that the Ys and Zs are no longer independent. Nevertheless, the magnitude of the dependence between the Ys $-D(Y|x^1)$ —is greater than that between the Zs $-D(Z|x^1)$ —which in turn is close to 0. This is so because the probabilistic links between the Ys and Zs further attenuates the flow of information.

Whereas the normative model predicts  $D(Z|x^1) = D(Z|x^1y_1^1y_2^1) = 0$ , the leaky gate model predicts  $D(Z|x^1) > D(Z|x^1y_1^1y_2^1) \approx 0$ . When the flow of information is blocked by three variables, virtually no information gets transmitted between the Zs even when the gates are leaky.

Eqs. 5-7 can also be applied to the elaborated common effect model in Fig. 4 in which the causes  $Y_1$  and  $Y_2$  are the effects of  $Z_1$  and  $Z_2$ . That figure also shows that whereas the normative model predicts that D(Y) = D(Z) = 0 (i.e., the causes are unconditionally independent), the leaky gate model predicts instead that D(Y) > D(Z) > 0. It also predicts that explaining away will be weaker (i.e.,  $D(Y|x^1)$  and  $D(Z|x^1)$  will be less negative) than in the normative model.

In summary, the key predictions of the leaky gate model are that  $D(Y|x^1) > D(Z|x^1) \ge 0$  for the elaborated common cause model, D(Y) > D(Z) > 0 for the elaborated common effect model, and  $D(Z|x^1y_1^1y_2^1) \approx 0$  for both.

#### **The Dual Prototype Model**

The dual prototype model is based on the intuition that causal inferences reflect a bias to expect that the variables in a network are either all present or all absent. Like the leaky gate model, the joint distribution it defines,  $p_{DP}$ , modifies the one specified by the normative model,  $p_N$ . For the simple common cause and common effect networks in Figs. 1 and 2,  $p_{DP}$  is the same  $p_{LG}$  and so reproduces the independence violations in Figs. 1 and 2 in the same manner. Differences between  $p_{DP}$  and  $p_{LG}$  arise for the networks in Figs. 3 and 4, however. In particular, Eq. 10 is analogous to Eq. 7 but elaborated with an alternative energy function,

$$log(p_{DP}(X, Y_1, Y_2, Z_1, Z_2 | \theta, a))$$
(10)  

$$\propto \epsilon_{XY_1Y_2Z_1Z_2} + log(p_N(X, Y_1, Y_2, Z_1, Z_2 | \theta))$$

$$\epsilon_{XY_1Y_2Z_1Z_2} = \begin{cases} a, \quad X = Y_1 = Y_2 = Z_1 = Z_2 \\ 0, \quad \text{otherwise} \end{cases}$$
(11)

 $\epsilon_{XY_1Y_2Z_1Z_2}$  represents the expectation that all five variables will tend to be all present or all absent.

Unlike the leaky gate model, the predictions of the dual prototype model are less sensitive to the distance between two variables in a network. For the extended common cause network (Fig. 3) it predicts a smaller difference between D(Y) and D(Z), that  $D(Y|x^1) \approx D(Z|x^1)$ , and that  $D(Z|x^1y_1^1y_2^1)$  will be greater than 0 (and about equal to  $D(Z|x^1)$ ). For the extended common effect network (Fig. 4) it predicts that  $D(Y|x^1y_2^1) > 0$  and  $D(Z|x^1y_1^1y_2^1) > 0$ . The prediction that  $D(Z|x^1y_1^1y_2^1) > 0$  in both networks is especially notable because they imply the presence of independence violations between the Zs even when they are separated by multiple "blockers."

#### **Overview of Experiment**

These predictions are tested in an experiment in which subjects learned either the common cause network in Fig. 3 or the common effect network in Fig. 4 and then drew a number of causal inferences, including those necessary to



*Figure 4.* Predictions of the normative model, the leaky gate model, and the dual prototype model for the elaborated common effect network. The "•" in, say,  $D(\bullet)$  represents either Y or Z. Empirical results are shown in the last column. Error bars are 95% confidence intervals.

compute the key quantities D(Y), D(Z),  $D(Y|x^1)$ ,  $D(Z|x^1)$ , and  $D(Z|x^1y_1^1y_2^1)$ . On the basis of previous research I expected that the independence relations stipulated by the normative model would be violated. The key question is whether those violations match the pattern predicted by the leaky gate model or the dual prototype model.

#### Method

**Materials**. Three domains were tested: economics, meteorology, and sociology. The five variables in each domain are shown in Table 1. In the database of materials each variable had the two values shown in Table 1 ("high" or "low" for interest rates) plus a third "normal" value. However, the variables were described as binary to subjects (e.g., interest rates were either high or normal) and the causal relations were described as obtaining between the non-normal values (e.g., low interest rates  $\rightarrow$  small trade deficits). To control for any domain knowledge that subjects might have brought to the experiment, a between-subject factor controlled which variable states were described as causally related and took on the values ++++, ----, -+-+-, and +-+-+, where each +/picks out the value in Table 1 for variables *A*, *B*, *C*, *D*, and *E*, respectively.

As a further safeguard against subjects' potential domain knowledge, a second between-subject factor controlled the assignment of these variables to the causal roles in Figs. 3 and 4. In the common cause condition, the roles of, X,  $Y_1$ ,  $Y_2$ ,  $Z_1$ ,  $Z_2$  were played by A, B, C, D, and E for half the subjects and by A, D, E, B, and C for the other half. In the common effect condition, they were played by D, B, C, A, and E for half the subjects and by D, A, E, B, and C for the other half. This scheme balances the assignment of the variables to the roles of Y and Z so that any differences between inferences involving Ys and those involving Zs cannot be attributed to the particular variables involved.

The description of each causal relation consisted of two sentences, one that stated that one variable caused another and a second that described the mechanism responsible for that relationship. Table 2 presents an example of one of the sets of causal relations that formed a common cause network in the domain of economics. Subjects also viewed a diagram of the causal relations and a third between-subject variable controlled which of four versions of that diagram was presented. The four versions of the common cause diagram are presented in Fig. 5 (names of the actual variables replaced  $X, Y_1$ , etc.). These layouts were chosen to ensure that the spatial distance between the two Ys on the screen was the same as between the Zs and that the Ys and Zs appeared an equal number of times in each quadrant of the screen. The four common effect diagrams were the same as those in Fig. 5 with the arrows reversed.

		Table I	
Variable	Economics	Meteorology	Sociology
A	Interest rates	Ozone levels	Urbanization
	(low+/high-)	(high+/low-)	(high+/low-)
В	Trade deficits	Air pressure	Interest in religion
	(small+/large-)	(low+/high-)	(low+/high-)
С	Retirement	Humidity	Socio-economic
	savings	(high+/low-)	mobility
	(high+/low-)		(high+/low-)
D	Job mobility	Wind direction	Interest in sports
	(high+/low-)	(up+/ down-)	
Ε	Income taxes	Air temperature	Commitment to
	(low+/high-)	(low+/high-)	the rule of law
			(strong+/weak-)

**Procedure**. Subjects first studied several screens of information about the domain and then performed the inference test. The initial three screens presented a cover story and a description of the domain's five variables and their two values. A fourth screen presented the four causal links and a fifth the presented the diagram of those links. When ready, participants took a multiple-choice test of this knowledge. While taking the test, participants could return to the information screens they had studied; however, doing so obligated them to retake the test.

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Causal	
relation	Description
$X \to Y_1$	Low interest rates cause small trade deficits. The low cost of
	borrowing money leads businesses to invest in the latest manu-
	facturing technologies, and the resulting low-cost products are
	exported around the world

- $X \rightarrow Y_2$  Low interest rates cause high retirement savings. Low interest rates stimulate economic growth, leading to greater prosperity overall, and allowing more money to be saved for retirement in particular.
- $Y_1 \rightarrow Z_1$  Small trade deficits cause high job mobility. The intense demand for exports means that entrepreneurs are forming many new companies, and those new companies must hire workers away from existing companies.
- $Y_2 \rightarrow Z_2$  High retirement savings causes low income taxes. When retirement savings are high, states are likely to lower income taxes to encourage spending and so stimulate the economy.

Subjects were then presented with a test that included those inferences needed to compute  $D(Y), D(Z), D(Y|x^1)$ ,  $D(Z|x^1)$ , and  $D(Z|x^1y_1^1y_2^1)$ . Each question presented a particular economy (or society or weather system) whose variables were shown in a layout like one of those in Fig. 5. The values of variables whose state was known appeared in its corresponding box, boxes for variables whose values were unknown were blank, and the unknown variable whose value was being requested was filled with "?????." Subjects were asked "What's the probability that [this economy] has X?" where X was the to-be-inferred variable (e.g., high interest rates). Subjects entered their response by moving a tick on a rating scale whose ends were labeled "0%" and "100%". Each inference type was asked in both directions, e.g., subjects were asked both  $p(y_1^1|y_2^1)$  and  $p(y_2^1|y_1^1)$ . The order of the 36 questions was randomized for each subject. Subjects could refer to a printed sheet of the causal diagram during the entire inference test.

**Design and participants**. The experiment consisted of a 2 (network type: common cause or common effect)  $\times$  3 (domain: economics, meteorology, or sociology)  $\times$  2 (assignment of variables to roles) between-subject design. 96 New York University undergraduates received course credit for participating and were randomly assigned to these 12 cells subject to the constraint that an equal number appeared in each cell. The four level factors that controlled which variable states were described as causally related and which version of the diagram was presented were both randomly assigned for each subject.

#### Results

Initial analyses revealed no effects of the between-subject counterbalancing variables and so the results are presented in Figs. 3 and 4 collapsed over those factors. The common cause and common effect conditions are reported separately.



#### Figure 5

Common cause results. Consistent with previous research, the Ys were treated as dependent even when the state of their common cause X was known: Conditioned on  $x^1$ , one Y was rated about 15 points more probable when the other Y was present versus absent. One new finding is that the Zs were also treated as dependent and that the magnitude of those effects,  $D(Y|x^1)$  and  $D(Z|x^1)$ , were about equal. Another is that the magnitude of the dependence between the Zs was the same regardless of whether they were separated by one or three blockers,  $D(Z|x^1) \approx D(Z|x^1y_1^1y_2^1)$ . These three differences scores were each significantly greater than zero, ps < .0001, and not significantly different from one another, ts(47) < 1. This pattern of results was predicted by the dual prototype model but not the leaky gate model (Fig. 3). One unexpected result is that whereas all three models predicted that D(Y) > D(Z), the corresponding empirical judgments (29 and 26, respectively) did not differ from one another significantly, t < 1. Notably, it was the dual prototype model that predicted the smallest difference between D(Y) and D(Z).

Common effect results. Also consistent with previous findings, the causes of a common effect network were treated as unconditionally dependent: One Y was rated about 10 points more probable when the other Y was present versus absent, p < .001. Although the degree of dependence between the Zs - D(Z) - was a bit smaller (7 points), it was significantly different than 0, p < .01, and not significantly different than D(Y), t(47) = 1.27, p = .29. In addition,  $D(Z|x^1y_1^1y_2^1)$  was significantly greater than 0, p < .001. These results  $-D(Y) \approx D(Z)$  and  $D(Z|x^1y_1^1y_2^1) > 0$ -were predicted by the dual prototype model but not the leaky gate model (Fig. 4). One unexpected finding is that  $D(Y|x^1) \approx$  $D(Z|x^1), t < 1$ , whereas the dual prototype model predicted  $D(Z|x^1) > D(Y|x^1)$ . Notably, although these measures of explaining away were negative, neither differed significantly from zero, ps > .14.

A model fitting exercise in which the three models were fit to the subjects' inferences corroborated the apparent superiority of the dual prototype model. Although space prohibits a full reporting of these results, I found that the common cause inferences were fit best by the dual prototype model (AIC = 75.4), followed by the normative (90.0), and leaky gate models (92.0). Common effect inferences were also fit best by the dual prototype model (AIC = 63.4), followed by the leaky gate (70.8) and normative models (77.1). The predictions of the fitted dual prototype model deviated from the empirical ratings by an average of 4.1 and 3.1 points (on a 0-100 scale) in the common cause and common effect conditions, respectively, and the correlations with those ratings were .96 and .98.

#### Discussion

Empirically, this article has both replicated and extended

previous findings. Once again, the two effects of a common cause network were not treated as independent conditioned on their cause. The new finding is that the same is true of the effects of those effects: The Zs were treated as dependent just like the Ys. The two causes of a common effect network (the Ys) were again not treated as unconditionally independent and, in a new result, so too were the causes of those causes (the Zs). Remarkably, in both networks the Zs were treated as dependent even when separated by multiple "blockers" ( $D(Z|x^1y_1^1y_2^1) > 0$ ).

Theoretically, this article has advanced our understanding of such effects via the testing of two formal models. One might have expected that independence violations arise because information flows along a network in cases where it shouldn't. Yet this intuition—formalized as the leaky gate model—predicts that the magnitude of independence violations should decrease as the number of blockers between two variables increases. The fact that the common cause subjects treated the Zs as dependent to the same degree regardless of whether they were separated by three blockers or one provides striking evidence against "spreading activation" sorts of accounts.

Instead, reasoners behaved as if they overestimated the likelihood of two prototypical networks states, one in which all variables were present and another in which they were all absent, a view which explains the absence of an effect of either distance (*Y*s vs. *Z*s) or the number of blockers on the magnitude of independence violations. Because this tendency works in the opposite direction of explaining away in a common effect network, the dual prototype model also accounts for the lack of significant explaining away in this and past work (Rehder & Waldmann, 2015; Rehder, 2014a; Rottman & Hastie, 2013).

It is important to note that the dual prototype model does not imply that reasoners ignore the direction of causality in their causal inferences, because that model's joint distribution modifies rather than replaces the joint derived from the normative model. Common cause and common effect networks are theoretically important because they are identical ignoring the direction of causality. Yet in every experimental test of which I am aware (including this one) differences between those networks obtain (compare Figs. 3 and 4). The conclusion to be drawn from this work is not that people aren't sophisticated causal reasoners but rather that their understanding of the statistics implied by causal networks differs from that of network theorists.

One might be tempted to interpret the dual prototype model's success as reflecting subjects' beliefs that the causal links operated deterministically (and that the effect variables had no alternative causes), a situation that would normatively predict that variables were either all present or all absent (at least for the common cause network). Yet when these subjects were asked to infer X given 0, 1, or 2 Ys (i.e.,  $p(x^1|y_1^0, y_2^0), p(x^1|y_i^0, y_j^1)$ , or  $p(x^1|y_1^1, y_2^1)$ ) their ratings (27, 57, and 82 in the common cause condition; 29, 64, and 97 in the common effect condition) reflected a belief that the causal links were probabilistic rather than deterministic (and the fitted causal power parameter was < .86 in both conditions). Nevertheless, an experimental test of the dual

prototype model in which causal links are described as probabilistic is called for.

Cognitive theories based on causal graphical models have enjoyed great success accounting for causal-based judgments. Yet, the fact that people fail to honor the independence relations that such graphs express means that the theories rest on shaky foundations. It is time to move them onto on a firmer foundation, one that embodies fundamental empirical facts regarding how people draw causal inferences. Let the formalisms described herein represent modest proposals in that direction.

#### References

- Ali, N., Chater, N., & Oaksford, M. (2011). The mental representation of causal conditional reasoning: Mental models or causal models. *Cognition*, 119, 403-418.
- Cheng, P. (1997). From covariation to causation: A causal power theory. *Psychological Review*, *104*, 367-405.
- Fernbach, P. & Rehder, B. (2012). Toward an effort reduction framework for causal inference. Argument and Computation, 4, 1-25
- Koller, D. & Friedman, L. (2009). Probabilistic Graphical Models: Principles and Techniques. Cambridge, MA: MIT Press.
- Lagnado, D.A. & Sloman S.A. (2004). The advantage of timely intervention. *Journal of Experimental Psychology: Learning, Memory, & Cognition, 30*:856–76
- Mayrhofer, R. & Waldmann, M. R. (2015). Agents and causes: Dispositional intuitions as a guide to causal structure. *Cognitive Science*, 39, 65-95.
- Park, J. & Sloman, S. A. (2013). Mechanistic beliefs determine adherence to the Markov property in causal reasoning. *Cognitive Psychology*, 67, 186-216.
- Pearl, J. (2000). Causality: models, reasoning, and inference. Cambridge, UK: Cambridge University Press.
- Perales, J., Catena, A., & Maldonado, A. (2004). Inferring non-observed correlations from causal scenarios: The role of causal knowledge. *Learning and Motivation*, 35, 115– 135.
- Rehder, B. (2014a). Independence and dependence in human causal reasoning. *Cognitive Psychology*, 72, 54-107.
- Rehder, B. (2014b). The role of functional form in causalbased categorization. *Journal of Experimental Psychology: Learning, Memory, and Cognition.*
- Rehder, B., & Burnett, R. C. (2005). Feature inference and the causal structure of object categories. *Cognitive Psychology*, 50, 264-314.
- Rehder, B., & Waldmann, M. (2015). Failures of Explaining Away and Screening Off in Described versus Experienced Causal Learning Scenarios. Submitted for publication.
- Rottman, B., & Hastie, R. (2014). Reasoning about causal relationships: Inferences on causal networks. *Psychological Bulletin*, 140, 109-139.
- Spirtes, P., Glymour, C., & Scheines, R. (2000). Causation, prediction, and search. New York: Springer-Verlag.
- Walsh, C. R., & Sloman, S. A. (2008). Updating beliefs with causal models: Violations of screening off. In G. H. Bower, et al. (Eds.), *Memory and Mind: A Festschrift for Gordon Bower* (pp. 345-358).