Differences in the Weighting and Choice of Evidence for Plausible versus Implausible Causes

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Abstract

Individuals have difficulty changing their causal beliefs in light of contradictory evidence. We hypothesized that this difficulty arises because people facing implausible causes give greater consideration to causal alternatives, which, because of their use of a positive test strategy, leads to differential weighting of contingency evidence. Across four experiments, participants learned about plausible or implausible causes of outcomes. Additionally, we assessed the effects of participants' ability to think of alternative causes of the outcomes. Participants either saw complete frequency information (Exp. 1 and 2), or chose what information to see (Exp. 3 and 4). Consistent with the positive test account, participants given implausible causes were more likely to inquire about the occurrence of the outcome in the absence of the cause (Exp. 3 and 4) than those given plausible causes. Furthermore, they gave less weight to Cells A and B in a 2 x 2 contingency table and gave either equal or less weight to Cells C and D (Exp. 1 and 2). These effects were inconsistently modified by participants' ability to consider alternative causes of the outcome. The total of the observed effects are not predicted by dominant models of normative causal inference, nor by the particular positive test account proposed here, but they may be commensurate with a more broadly-construed positive test account.

Differences in the Weighting and Choice of Evidence for Plausible versus Implausible Causes

Across domains it is commonly observed that prior theories and beliefs influence how people interpret evidence. For example, individuals have difficulty changing their causal beliefs in light of contradictory data. This failure to revise beliefs has concerned experimental psychologists (e.g., Alloy & Tabachnik, 1984; McKenzie & Mikkelsen, 2007; Taylor & Ahn, 2012) and philosophers of science (e.g., Fodor, 1984; Giere, 1994), with much work addressing its putative normativity. Normative or not, the entrenchment of prior beliefs is increasingly seen as a problem by science educators, with even the brightest students holding onto their naïve conceptions about scientific phenomena (e.g., Chinn & Brewer, 1993; Schauble, 1990; Taber, 2003; Treagust, Chitleborough, & Mamiala, 2002). Furthermore, it is a problem for society when people maintain erroneous causal beliefs despite repeated demonstrations to the contrary (e.g., the belief that vaccines cause autism; Lewandowsky, Ecker, Seifert, Schwarz, & Cook, 2012).

Work on both covariation assessment and on causal inference from contingency data has demonstrated that judgments about the relation between two events are biased in the direction of prior expectations (e.g., Billman, Bornstein, & Richards, 1992; Dennis & Ahn, 2001; Freedman & Smith, 1996; Fugelsang & Thompson, 2000; Fugelsang & Thompson, 2003; López, Shanks, Almaraz, & Fernández, 1998; Marsh & Ahn, 2006; Mutter, Strain, & Plumlee, 2007; Schulz, Bonawitz, & Griffiths, 2007; Wright & Murphy, 1984). For example, after seeing contingency data, people judge two variables to be more strongly related when they are linked by a plausible causal mechanism (e.g., severed brake lines causing car accidents) rather than an implausible one (e.g., flat tires causing car start failures; Fugelsang & Thompson, 2000). This effect obtains not only for beliefs participants have prior to entering the experiment but also those acquired as part of the experiment (e.g., Garcia-Retamero, Müller, Catena, & Maldonado, 2009, Exp. 1; Marsh & Ahn, 2006; Taylor & Ahn, 2012). In particular, for simple causal relations, a primacy effect is observed in the interpretation of contingency evidence that favors participants' initial hypotheses (Marsh & Ahn, 2006; Taylor & Ahn, 2012).

How **Does Prior Belief Influence Causal Inference from Contingency Evidence?**

This paper is primarily concerned with the mechanisms via which prior belief influences causal inference from contingency data. Prior beliefs take on many forms. One may have generic knowledge of object kinds, domain-specific knowledge about the form of relations between causes and outcomes, or prior beliefs about whether the causal mechanism produces its outcome deterministically versus probabilistically (e.g., Cheng, 1997; Griffiths, Sobel, Tenenbaum, & Gopnik, 2011; Lien & Cheng, 2000; Novick & Cheng, 2004; see Griffiths & Tenenbaum, 2009, for a review). Our interest, however, is in beliefs concerning the plausibility of specific causal mechanisms.

Some formal theories of rational causal inference are either silent with respect to the role of specific causal mechanism information (ΔP; Jenkins & Ward, 1965) or emphasize the importance of purely covariation-based causal learning because it can occur in the absence of such information (causal power; Cheng, 1997). From a Bayesian perspective, however, prior beliefs *should* influence the interpretation of new evidence (e.g., Koslowski, 1996; McKenzie & Mikkelsen, 2007; Schulz et al., 2007).

According to Bayesian inference, current belief in a hypothesis is a product of prior belief in that hypothesis and the current evidence (Pearl, 2000). Thus, prior beliefs are expected to affect the interpretation of evidence as part of rational belief updating.

Yet, existing models of Bayesian causal learning (e.g., Griffiths & Tenenbaum, 2005; Lu, Yuille, Liljeholm, Cheng, & Holyoak, 2008) do not explicitly address *how* prior knowledge regarding the believability of a causal mechanism is integrated with contingency evidence. The closest approximation is a model of causal inference in the blicket detector paradigm (Griffiths et al., 2011), which accounts for prior knowledge of the cause's base rate and whether the causal mechanism is deterministic or probabilistic. However, this model neither generalizes across domains nor captures the effects of the believability of the causal mechanism. Variability in the base rate of the causal objects is not the same as variability in the believability of a causal mechanism: Infrequently occurring events do not necessarily imply implausible causal mechanisms (e.g., nuclear fission causing a nuclear explosion). Similarly, both deterministic and probabilistic relations may vary in their degree of believability.

Fugelsang and Thompson (2003) demonstrated that learners' prior beliefs indeed affect how new covariation evidence is interpreted, although the effect varies with the form of those beliefs. When they established a prior expectation by manipulating the causal mechanism's plausibility, participants' causal judgments varied more with new contingency evidence for plausible as opposed to implausible mechanisms (see also Mutter et al., 2007). In contrast, when the expectation took the form of prior covariation information, participants' judgments suggested that it was simply added to the new covariation evidence to form an impression of the strength of the causal relationship.

To account for these data, Fugelsang and Thompson introduced a dual process model of belief-evidence integration, which bears several similarities to the interactional framework model of Alloy and Tabachnik (1984). This model stipulates that belief-updating in the face of new covariation evidence occurs in two stages. The first involves recruiting prior knowledge regarding the relation between the candidate cause and outcome. This process, which occurs outside of conscious awareness, could yield knowledge of a causal mechanism and of how the two events covaried in the past. In the second stage, individuals evaluate new covariation evidence and make an inference. Fugelsang and Thompson proposed that the weighting of covariation information increases as a function of the plausibility of the causal mechanism.

A Positive Test Strategy Account of Plausibility Effects on Evidence Weighting

Why is this interaction observed with causal mechanism information? Going a step beyond Fugelsang and Thompson (2003), we suggest that an implausible mechanism leads learners to attend to different types of co-occurrence information. This information—represented by the familiar 2 x 2 table in Figure 1—consists of the number of cases where the candidate cause and outcome are both present (Cell A), the cause is present and outcome is absent (B), the cause is absent and the outcome is present (C), and both are absent (D). We hypothesized that participants faced with plausible versus implausible causal mechanisms would weight information in these cells differently.

Research employing cover stories with plausible or neutral causal mechanisms demonstrates that participants do not weight the four cells equally. When judging the effects of a putative generative cause participants generally demonstrate the cell-weight inequality A > B ≥ C > D (Mandel & Lehman, 1998). This inequality is observed in participants' explicit rankings of cell importance (Levin, Wasserman, & Kao, 1993; Wasserman, Dorner, & Kao, 1990), and in the correlation between participants' causal judgments and the cell frequencies (Levin et al., 1993; Mandel & Lehman, 1998; Mutter & Plumlee, 2009). Importantly, this inequality changes for putative preventative causes such that B is considered the most important cell (B > A > D > C; Mandel & Vartanian, 2009; see also Levin et al., 1993, Exp. 2).

These results have been interpreted as reflecting a *positive test strategy* (Mandel & Vartanian, 2009). For generative causes, cells A and B (and especially A) provide positive tests and cells C and D provide a negative one; for preventative causes, Cell B provides the most positive test and it becomes more important than A. The primacy effect observed when contingency information is presented trial-by-trial has also been interpreted as reflecting a positive test strategy, in which a hypothesis established early in learning affects the processing of subsequent trials (Marsh & Ahn, 2006). Consistent with this idea, the interpretation of each cell depends upon participants’ prior learning experience (Luhmann & Ahn, 2011).

Here, we extend the positive test strategy[1] account to the situation of implausible causes. Whereas Fugelsang and Thompson (2003) suggested that implausible causes lead learners to place less weight on the data overall, we propose that it also leads to a shift in cell weights. When faced with a plausible causal mechanism, learners’ preference for few or even single causes (e.g., Dougherty, Gettys, & Thomas, 1997; Lombrozo, 2007) means they are likely to adopt that plausible cause as their focal hypothesis, in which case a positive test strategy (Klayman & Ha, 1987; McKenzie,

2004) yields the usual cell weight inequality (Levin et al., 1993; Mandel & Lehman, 1998). But when faced with an implausible cause, learners may fail to adopt it as their focal hypothesis and so the normal overweighting of Cells A and B will be eliminated. Even further, an implausible cause might encourage a learner to focus on alternative causes, in which case Cells C and D become the “positive tests.” Cell C may be of particular importance, because a non-zero frequency in that cell confirms the action of unobserved alternative causes (e.g., Hagmayer & Waldmann, 2007; Luhmann & Ahn, 2007; Luhmann & Ahn, 2011; Rottman, Ahn, & Luhman, 2011).

Plausibility and the Consideration of Alternative Causes

Although individuals generally prefer few or even single causes of an outcome (Dougherty et al., 1997; Lombrozo, 2007; Lu et al., 2008; McKenzie, 1994), there are a number of circumstances in which they do consider causes in addition to those explicitly presented in an experiment (Cummins, 1995; Fernbach, Darlow, & Sloman, 2010; Luhmann & Ahn, 2007; see Rottman et al., 2011, for a review). Of particular relevance here, individuals differ in the number of alternative causes of an outcome they think of (Dougherty et al., 1997; Hirt & Markman, 1995; Sprenger & Dougherty, 2012). Causal scenarios also differ, with some supporting more alternative causes than others (e.g. Cummins, 1995). For example, there are many causes of stress, but few causes of nuclear explosions. Thus, we hypothesize that the weighting of cause absent information (e.g., cell C) will increase not only for implausible causes but also as a function of the number of causes that individuals consider.

Predictions of Extant Models

What do extant models predict with regards to the effects of plausibility on data weighting? Recall that, in contrast to our positive test hypothesis, Fugelsang and Thompson's (2003) model of belief-evidence integration predicts that less weight overall will be given to contingency evidence for implausible causal scenarios. Formal models of rational causal inference either do not incorporate priors regarding the plausibility of a specific causal relation (Cheng, 1997; Lu et al., 2008)[2] or employ uniform priors (Griffiths & Tenenbaum, 2005). To determine what these models would predict for causal relations differing in plausibility, we performed a modeling exercise adding plausibility-based priors to these models (see Appendix A for a full description of this exercise). In brief, we employed three different priors to represent differences in the plausibility of the causal mechanism (.9, .5, and .1 for high, moderate and low plausibility, respectively). We calculated posterior causal strength as a weighted average of the prior and the model-specific estimate of causal strength calculated over randomly-generated 2 x 2 contingency data.[3] To calculate cell weights, we regressed the posterior causal strength estimates on the frequencies for cells A, B, C and D. Figure 2 excerpts graphs from Appendix A for modified causal power and causal support when causes are moderately common [p(C) = p(E) = .50], because these best-illustrate the differing predictions of the two models.

Predictions of the causal strength version of Griffiths and Tenenbaum's (2005) causal support model are consistent with that of Fugelsang and Thompson (2003): As plausibility decreases from .9 to .1, there is decreased weighting of the evidence from all cells of the contingency table (Figure 2B). In contrast, the modified version of

Cheng’s (1997) causal power predicts differential weighting of the cells as a function of plausibility (Figure 2A): As plausibility decreases from .9 to .1, there is increasing weight on confirming evidence (Cells A and D) and decreasing weight on disconfirming evidence (Cells B and C). Thus, in contrast to the positive test hypothesis, the modified causal power model predicts that evidence disconfirming a prior expectation will be given more weight.

Finally, while the predictions of the modified causal support and causal power models were directionally stable across changes in the base rates, predictions of Lu et al.’s (2008) sparse and strong (SS) priors model varied. With low base rates, those predictions aligned more with causal power, but with high base rates, they aligned more with causal support.

In sum, extant models predict either an across the board decrease in cell weights for implausible causes [Fugelsang & Thompson (2003); our modification of Griffiths & Tenebaum’s (2005) causal support; and Lu et al.’s (2008) SS priors with high base rates] or they predict an increase in weighting of confirming evidence, with a decrease in weighting of disconfirming evidence for implausible causes [modification of causal power (Cheng, 1997) and Lu et al.’s (2008) SS priors with low base rates]. These predictions differ from those of the positive test account posed here, which predicts a decrease in the weighting of cause-present information (Cells A and B) and an increase in the weighting of cause-absent information (Cells C and D) for implausible causes.

Overview of Current Experiments

Across four experiments we assessed how participants’ use of data varied with the plausibility of the causal mechanism. Each experiment manipulated the plausibility of causes: participants learned about either highly plausible causal relations (e.g., severed brakes causing car accidents) or implausible ones (e.g., leather car seats causing car accidents).

Experiments 1 and 2 tested whether plausibility affects learners' cell weights. On each trial, participants received complete frequency information corresponding to the four cells of the contingency table and made a causal judgment. Experiments 3 and 4 tested whether effects of plausibility extended to participants' choice of information. Instead of complete frequency data, they received opportunities to select either a cause-present case (e.g., car *with* severed brakes) or a cause-absent case (e.g., car *without* severed brakes). Participants then saw the outcome (e.g., whether or not that car was in an accident). After observing a limited number of cases, participants made causal judgments. Our primary interest, however, was in participants' choices. At the end of Experiments 2 through 4, participants listed as many causes as they could think of for each of the outcomes.

The positive test strategy account predicts that, relative to those given plausible causes, participants given implausible causes would place less weight on Cells A and B, greater weight on Cells C and D, and more frequently choose to inspect cause-absent cases. Additionally, in Experiments 2 through 4 we tested whether participants' weighting and choice of evidence varied with their ability to list causal alternatives.

Experiment 1

Method

Participants. Undergraduate students (*N* = 166; 117 female) attending either the University of Cambridge or Seton Hall University completed the experiment in partial fulfillment of a course requirement or for ₤10. They ranged in age from 18 to 35 years (*M* = 20.12, *SD* = 2.46).

Procedure. Participants completed the experiment at a computer running E-Prime 2.0 (Psychology Software Tools, Pittsburgh, PA). All participants learned about the causes of skin rashes and car accidents in separate randomly-ordered blocks (cover stories modified from Fugelsang & Thompson, 2000). Different participants received the plausible and implausible causes (see left half of Table 1). For example, in the plausible skin rash condition, participants imagined that they were a doctor testing whether hiking in the woods causes skin rashes. They tested this hypothesis in 16 different doctors' offices by observing data about whether children went hiking in the woods and whether they experienced a skin rash. When assessing the causes of car accidents, participants imagined that they were a police officer investigating whether severed brake lines (plausible condition) or leather seats (implausible condition) caused car accidents for cars in 16 different county garages. For both cover stories, participants received complete frequency data on each of 16 randomly-ordered trials (see rows of Table 2 for cell frequencies on each trial). For example, on a single trial, some participants saw the following:

<u>Dr. Gibson's Office</u>

10 children WENT HIKING IN THE WOODS

8 of the 10 developed a skin rash.

5 children DID NOT GO HIKING IN THE WOODS

1 of the 5 developed a skin rash

On each trial, participants made a causal judgment between -100 and + 100 (-100 indicating the cause completely prevents the effect, 0 indicating no effect and +100 indicating the cause completely produces the effect). We instructed participants to base their judgment on the information from that particular garage (or doctor’s office), and to disregard other trials.

Design and Data Analysis. We estimated participants’ cell weights by calculating Pearson correlation coefficients between each participant's judgments (within cover story) and each of the cell frequencies (i.e., separate correlations for cells A, B, C, and D). We transformed the correlations into Fisher's *z* so that they could be used as measures in the analyses (Mandel & Lehman, 1998; Mandel & Vartanian, 2009). Previous researchers used the absolute value of participants' observed correlations, changing the sign on the correlations for cells B and C to negative, because these cells are normatively considered evidence against the hypothesis that the cause produced the effect (Mandel & Lehman, 1998; Mandel & Vartanian, 2009). In contrast, we left the sign on participants' observed correlations unaltered when performing analyses, because participants do not interpret the cell information in the expected normative way (Luhmann & Ahn, 2011). For ease of interpretation, however, we present absolute values of the mean cell weights in tables and graphs.

Prior to analysis we screened the data for multivariate outliers based on robust estimates of Mahalanobis’ distance. The critical value for Mahalanobis’s distance with two variables and an alpha of 0.05 is 5.992. We calculated separate estimates of Mahalanobis’s distance for each of the cells. Cases for which Mahalanobis’s distance exceeded 5.992 for at least one of the cells were counted as outliers and excluded from the analysis. In all experiments, each participant contributed more than one case. Thus, exclusion of individual cases did not necessarily eliminate whole participants, but we note when it did. Across experiments, similar patterns of results emerged in the screened and unscreened data.

We performed 2-level mixed linear model analyses (MLM), with a single random effect (participants' random intercepts). For the cell weight analyses, we analyzed the full factorial of the manipulated factors as fixed effects – i.e., 4 (cell: A, B, C, D) x 2 (outcome: skin rashes, car accidents) x 2 (plausibility: plausible, implausible). We determined the optimal structure for the residual covariance matrix with preliminary MLM analyses, using likelihood ratio tests to compare models assuming homogeneous versus heterogeneous variances and covariances, retaining the best-fitting residual covariance structure. We used maximum likelihood estimation because of our primary interest in the fixed effects (Singer & Willett, 2003, p.90) and the *F* distribution with between-within degrees of freedom (West, Welch, & Galecki, 2007; Rabe-Hesketh & Skrondal, 2008, p. 111). We followed significant interactions involving the factor of plausibility with single degree of freedom simple main effects tests (Keppel & Wickens, 2004), testing the effect of plausibility at each level of the variable with which it interacted. Final covariance structures and results for the random effects are presented in Appendix B.

Results

Two participants gave causal judgments outside the range of the scale and three gave judgments of zero on all 32 trials. These five participants were removed prior to analysis, leaving *N* = 161. Although the effect of sample did not reach significance, we observed a qualitatively different pattern of cell-weighting among the two samples (Cambridge/UK vs. Seton Hall/USA). Because the US sample was larger (*N* = 113; *n* = 54 for implausible and *n* = 55 for plausible) and because Experiments 2 through 4 employ US participants, we report the results from the US sample here and report the results from the smaller UK sample in Appendix C.

Cell Weights. As can be seen in Figure 3, compared to those receiving plausible causes, implausible participants gave less weight to Cells A and B but similar weight to C and D. This impression is confirmed by the significant plausibility by cell interaction, *F*(3, 761) = 10.45, *p* < .001. While the implausible group gave less overall weight to the data than did the plausible group, *F*(1, 111) = 5.96, *p* = .016, the difference between the groups only reached significance for Cells A and B [*p* < .001, *d* = 0.57 for Cell A; *p* < .001, *d* = 0.50 for B; *p* = .523, *d* = 0.12 for C; *p* = .081, *d* = 0.29 for D].

We also observed a main effect of cell, *F*(3,761) = 10.45, *p* < .001, whose interpretation is tempered by the interaction discussed above. No other effects approached significance [*F*(3, 761) = 1.66, *p* = .174, on the plausibility by cell by outcome interaction; all other *F*s < 1].

Discussion

In Experiment 1 we observed that the effect of plausibility differed depending on the cell. Having an implausible cause lowered participants' weights for Cells A and B, relative to having a plausible cause. While directionally this was also true for Cells C and D, the effect was much smaller and failed to reach significance for these cells.

The results are partially consistent with Fugelsang and Thompson (2003), in that having an implausible cause lowered reliance on the data. However, the differential weighting of the cells as a function of plausibility is not predicted by Fugelsang and Thompson (2003), nor by our modification of Griffiths and Tennebaum’s (2005) causal support (Appendix A), both of which predict lower overall cell weights for the implausible group. It is also not predicted by the modified causal power model, which predicts an increase in Cells A and D and a decrease in B and C as the prior probability of the cause decreases (Appendix A).

The results are partially consistent with our positive test account. We predicted that relative to plausible participants, those facing implausible causes would give less weight to Cells A and B and more weight to C and D. Consistent with this hypothesis, we observed a reduction in the positive test strategy for the experimentally-introduced causes among the implausible group – as observed in their reduced weights on Cells A and B. However, we did not observe any evidence that these participants adopted an alternative focal hypothesis, which we hypothesized would be demonstrated by them giving more weight to Cell C in particular.

While Experiment 1 yielded partial support for a positive test account, it has limitations. First, the objective contingencies, which varied between zero and 0.58, were low and positive; indeed, contingency was zero on half of the 16 trials (Table 2). Experiment 2 tested a wider range of contingencies.

Second, the standard deviations of the cell frequencies differed (i.e., looking down the columns in Table 2, *SD*s ranged from 2.50 to 2.74). Differences in the variability of cell frequencies might have produced artificial cell weight differences because higher variability implies a higher maximum correlation with participants’ judgments. Of course, differences in variability cannot explain the differences in cell weights between the plausible and implausible conditions. Nonetheless, in Experiment 2 we equated the cells' standard deviations.

A third potential limitation was how the frequency information was presented. Indicating how many times the outcome occurred for the cause present and cause absent cases gave participants the Cell A and C frequencies but required them to perform subtraction to determine the frequencies for B and D. For example, participants told "8 of the 10 children who went hiking in the woods developed a skin rash," needed to subtract to determine that 2 of the 10 did not develop a skin rash. Experiment 2 addressed this issue as well.

Experiment 2

Experiment 2 investigated the effects of plausibility and the number of causes listed on participants' cell weighting with the aforementioned changes: (a) we explicitly indicated the numbers corresponding to the frequency in each of the cells (avoiding the need for subtraction), (b) a broader range of contingencies was tested, and (c) the standard deviations of the four cells were equated (*SD*s = 6.68). Because equal standard deviations entail both positive and negative contingencies, those contingencies varied between -0.85 and 0.85 (see Table 3).

Experiment 1 tested two outcomes: car accidents and skin rashes. Although Experiment 1 did not yield outcome effects, to better test for this possibility, and to extend our results to additional cover stories, we performed pilot testing to identify two additional outcomes (see Appendix D). The number of causes listed for car accidents and stress were about the same (*M* = 7.01, *SD* = 4.06 and *M* = 7.07, *SD* = 3.70, respectively), as were the number for plant growth and skin rashes (*M* = 4.33, *SD* =

1.98 and *M* = 4.04, *SD* = 2.04). Thus, Experiment 2 tested two outcomes for which people listed more causes (stress and accidents) and two for which people listed fewer (plant growth and skin rashes). Finally, at the end of the experiment participants listed all the possible causes they could think of for these outcomes.

Method

Participants. Seton Hall University undergraduates (*N* = 125; 86 female) participated in partial fulfillment of a course requirement (*n* = 62 implausible and *n* = 63 plausible). They ranged in age from 18 to 26 years old (*M* = 18.83, *SD* = 1.24).

Procedure. The procedure was similar to that of Experiment 1, with a few exceptions. All participants made causal judgments on 16 trials for each of the four outcomes in Table 1, for a total of 64 trials. When assessing the causes of plant growth, participants imagined they were a botanist testing whether fertilizer (plausible) or being in a blue pot (implausible) led to healthy plant growth for plants in 16 different greenhouses. When assessing the causes of stress, participants imagined they were a clinical psychologist testing whether having lots of school deadlines (plausible) or eating lots of fruits and vegetables (implausible) leads to complaints of stress among students visiting a school's counseling center (in 16 different schools).

Order of presentation of the four outcome types was counterbalanced across participants and trials were presented in a random order. Each row in Table 3 corresponds to one trial and these cell frequencies were explicitly indicated to participants. For example, on a single trial, a participant may have seen the following:

<u>Dr. Gibson's Office</u>

10 children WENT HIKING IN THE WOODS

8 of the 10 developed a skin rash.

2 of the 10 did not develop a skin rash.

5 children DID NOT GO HIKING IN THE WOODS

1 of the 5 developed a skin rash.

4 of the 5 did not develop a skin rash.

After completing all four blocks of trials, participants wrote down all the possible causes they could think of for each of the four outcomes (order counter-balanced across participants). In all other respects, the procedure for Experiment 2 was that same as that for Experiment 1.

Design and Data Analysis. We calculated cell weights and performed MLM analyses as in Experiment 1, analyzing the full factorial of the manipulated variables and the number of listed alternatives as fixed factors in the MLM – i.e., a 4 (outcome: skin rashes, accidents, plant growth, stress) x 2 (plausibility: implausible, plausible) x 4 (cell: A, B, C, D) x number of listed alternatives.

In this and subsequent experiments we observed that the range on the number listed alternatives often differed for the implausible and plausible groups. Because a difference in range could alter the size of the coefficient for predicting cell weights, we performed the analysis over the smaller range of cases. Prior to analyses we screened for outliers as in Experiment 1.

Results

One participant from the implausible condition was excluded from the analysis for giving causal judgments of zero on all 64 trials, leaving *N* = 124 (*n* = 61 and 63 in implausible and plausible, respectively).

Number of Causes Listed. While one might suspect that participants given implausible causes would be induced to think of more alternatives, participants in the implausible (*M* = 6.74, *SD* = 3.77) and plausible (*M* = 6.89, *SD* = 3.81) conditions listed a similar number of alternatives, *F* < 1, an effect replicating Fugelsang and Thompson (2000, Exp. 3). However, the number of alternatives listed varied by outcome, *F*(3, 496) = 33.17, *p* < .001. Consistent with the pilot study, participants listed more causes for car accidents (*M* = 8.23, *SD* = 3.87) and stress (*M* = 8.68, *SD* = 4.69) than for skin rashes (*M* = 5.27, *SD* = 2.53) and plant growth [*M* = 5.09, *SD* = 1.82; *p*s < .001 for Bonferroni comparisons]. Car accidents and stress did not differ from each other (*p* = .893), nor did skin rashes and plant growth (*p* = 1.00). Outcome and plausibility did not interact, *F* < 1.

Cell Weights. There were no outliers, but the range of listed alternatives was smaller for the implausible (2, 26) than the plausible (1, 26) group. We analyzed cases falling within the smaller range.

As seen in Figure 4, implausible and plausible participants differently weighted the frequency data from the cells [*F*(3, 1742) = 8.19, *p* < .001 for plausibility by cell interaction]. However, unlike Experiment 1, implausible participants gave significantly less weight to all cells than did plausible participants [*p* < .001, *d* = 0.48 for Cell A; *p* = .032, *d* = 0.21 for Cell B; *p* < .001, *d* = 0.45 for Cell C; and *p* = .021, *d* = 0.21 for Cell D]. The interaction obtained because while implausible participants gave similar weight to all four cells, *F*(3,1741) = 1.49, *p* = .215, cell weights for plausible participants varied, *F*(3, 1742) = 19.04, *p* < .001. They gave more weight to Cell A than B, *p* < .001, *d* = 0.56, and more weight to C than D, *p* < .001, *d* = 0.44, while the weights for A and C and that for B and D did not differ (*p* = .117, *d* = 0.13 and *p* = .975, *d* = 0.03, respectively).

As seen in Table 4, the slopes for predicting cell weights from the number of listed alternatives varied across cells [*F*(3, 1742) = 2.66, *p* = 0.047, for the cell by listed alternatives interaction]. Collapsed across plausibility conditions, as participants listed more alternatives, they placed greater negative weight on disconfirming evidence – i.e., frequency information from Cells B and C (Figure 5). This effect reached significance for Cell B, *p* = .032, but not for Cell C, *p* = .061.

We also observed significant main effects of plausibility, *F*(1, 1742) = 14.22, *p* < .001 and listed alternatives, *F*(1, 1742) = 3.85, *p* = .050, whose interpretations are tempered by the interactions described above. No other effects approached significance, *p*s > .11.

Discussion

In Experiment 2, implausible participants gave less weight to the data than did plausible participants. This result is consistent with the predictions of Fugelsang and Thompson (2003) and of the modified causal support model (Appendix A and Figure 2b). Additionally, we observed that all participants placed greater weight on disconfirming evidence as they listed more alternative causes. This result is consistent with studies finding that individuals generating more alternative causes judge a focal cause to be less likely compared to individuals generating fewer alternatives (Dougherty et al., 1997; Hirt & Markman, 1995). It is also consistent with a positive test strategy, broadly construed. Our version of the positive test account posited that participants facing implausible causes – or who think of many alternatives – may not only fail to adopt the experimentally-presented putative cause as their focal hypothesis, but may instead adopt an alternative cause as their focal hypothesis. The results of Experiments 1 and 2 do not support the latter prediction. However, we did observe support for a broadly construed version of the positive test account in which implausibility – or thinking of many alternatives – reduces the positive test for the experimentally-presented cause. In both experiments there was a reduction in the weighting of cause-present information for implausible causes, and in Experiment 2 participants thinking of more alternatives gave more weight to evidence disconfirming the experimentally-presented focal cause.

Experiments 1 and 2 differed in how implausible participants weighted Cells C and D (relative to plausible participants). This difference cannot be attributed to the new cover stories, because an analysis of only the two outcomes used in Experiment 1 left the results of Experiment 2 qualitatively unchanged. While we do not have a definitive account for why these experiments differed, the common finding across both experiments – a reduction in the weighting of data from Cells A and B – is predicted by both the positive test account and Fugelsang and Thompson's (2003) dual process model. We next tested whether this effect generalized to an information search paradigm.

Overview of Experiments 3 and 4

Experiments 3 and 4 assessed whether the effects of plausibility extended to participants' choices about what information they would like to see. While in Experiments 1 and 2 participants received complete contingency information on every trial, in Experiments 3 and 4 they saw a limited number of cases. On each trial, participants elected to see a case where the cause was either present or absent, after which they learned that case’s outcome. Participants chose five and nine cases to observe in Experiments 3 and 4, respectively. We expected a positive test bias (more “cause-present” cases chosen overall), but also an effect of plausibility such that participants given implausible causes would select fewer cause-present cases. We also expected that participants’ choice of cause-present cases may vary with the number of listed alternatives.

Experiment 3

Method

Participants. Seton Hall University undergraduates (N = 109; 74 female) participated in partial fulfillment of a course requirement (*n* = 55 plausible; *n* = 54 implausible). They ranged in age from 18 to 41 years old (*M* = 19.70, *SD* = 3.08).

Procedure. Participants learned of the causes of car accidents and skin rashes, with plausibility of the causes manipulated between-groups (as in Experiment 1). In contrast to previous experiments, there were only four trials, over which participants learned about four different doctor's offices (or county garages). For each office, participants read that there were 12 patients, six representing cause-present cases and six cause-absent cases. However, participants could view the files of only five of these 12 patients. Each sub-trial consisted of participants making a single choice to either view a file in which the cause was present or one in which it was absent. Figure 6A depicts a choice screen representing a sub-trial for participants in the plausible skin rash condition.

The presentation order of the cause-present and cause-absent choices was counter-balanced between participants: Half pressed *1* to see a cause present case (as depicted in 6A) and half pressed *2*, in which case the cause-present option appeared in the second position. After making their choice, participants saw a screen indicating the choice they made and the outcome (see Figure 6B). This screen remained visible until participants pressed the space bar, ending the sub-trial. After making five selections, participants made causal judgments on the same scale as in Experiment 1. Prior to beginning the experiment, participants received booklets to record both their choices and the outcome, so that they would not have to rely upon their memory when making causal judgments. After the choice and causal judgment task, participants listed all possible causes they could think of for car accidents and skin rashes (order counterbalanced across participants).

The objective contingency across all 12 files (of which participants only saw five) was zero. Each trial was associated with separate matrices for the cause-present and cause-absent choices. Within each matrix, three of the files indicated a presence of the outcome and three did not. For any given sub-trial, an outcome was chosen from the appropriate matrix (depending on the participant's choice) at random, without replacement. Thus, while the objective contingency was zero, each participant observed a different objective contingency based on that participant's choices and the random selection of the outcome.

Design and Data Analysis. We cleaned the data by eliminating individual sub-trials for which the participant’s response time was less than 250ms (5% of sub-trials). We then obtained the percent of cause present choices for each trial and took the average of that across each participant's four trials. The design of the experiment was a 2 (outcome: skin rashes, accidents) x 2 (plausibility: implausible, plausible) x 2 (option order: cause present first, cause present second) mixed design with outcome manipulated within-groups and plausibility and option order between-groups. We performed 2-level MLM analyses as in Experiments 1 and 2.

Results and Discussion

We excluded two participants because they failed to make any causal judgments. An additional three participants failed to list causes at the end of the experiment, leaving *N* = 104 (*n* = 50 plausible; *n* = 54 implausible).

Number of Causes Listed. Consistent with Experiment 2, participants in the implausible (*M* = 7.62, *SD* = 4.32) and plausible conditions (*M* = 7.00, *SD* = 2.88) listed a similar number of causes [*F*(1,102) = 2.90, *p* = .091, *d* = 0.17], but they listed more causes of car accidents (*M* = 8.84, *SD* = 4.05) than of skin rashes [*M* = 5.79, *SD* = 2.54; *F*(1, 206) = 41.23, *p* < .001, *d* = 0.82]. The plausibility by outcome interaction did not reach significance, *F* < 1.

Information Choice. Because we observed different ranges for the number of listed alternatives in the plausible (2, 14) and implausible (0, 26) conditions, we restricted the analysis to cases falling in the smaller range. No cases were identified as multivariate outliers.

Overall, we observed a small positive test bias: On average, participants chose the cause-present option more frequently than expected by chance (*M* = .528, SD = 0.10; one-sample *t* versus .50: *t*(103) = 2.04, *p* = .044, *d* = 0.24). Consistent with our expectation, this positive test bias was smaller among implausible participants: They made fewer cause-present choices (*M* = .517, *SD* = .10) than did plausible participants [*M* = .539, *SD* = .11; *F*(1, 100) = 5.16, *p* = .025, *d* = 0.15, for the main effect of plausibility]. We also observed an interaction between plausibility and the number of listed alternatives, *F*(1, 91) = 6.70, *p* = .011, which is depicted in Figure 7. Relative to the plausible condition, as participants in the implausible condition listed more causes, they chose the cause-present option less often (comparing slopes to zero: *b* = -0.007, *SE* = .005, *β* = -0.17, *p* = .162 for implausible; *b* = 0.004, *SE* = .005, *β* = 0.07, *p* = .424 for plausible).

While no other effects reached significance, *p*s ≥.06, there was a tendency for participants to choose the cause present option more often when that option appeared first (*M* = .57, *SD* = .11) versus second (*M* = .47 , *SD* = .14), *F*(1,100) = 3.68, *p* = .058.

Discussion

Relative to the plausible group, implausible participants chose the cause-present option less often and their cause-present choices decreased as they listed more causes of the outcomes. Although both of these effects were small (*d* = 0.15 for the effect of plausibility; *β* = -0.17 for the implausible group's slope), they are uniquely predicted by a positive test account.

Experiment 4

The goal of Experiment 4 was to address limitations of Experiment 3, some of which may have led to its small effects. First, because the potential effect of option order may have obscured the strength of the plausibility effect, we modified the way in which participants made their choices. Instead of pressing number keys mapped to the top and bottom choices, they used the mouse to click a box on the left or right of the computer screen (see Figure 8). Second, because Experiment 3 participants observed only five cases before making a causal judgment, it is likely that they did not see all four cells of the contingency table on many trials. Experiment 4 participants made nine choices and so observed nine cases on every trial. This adjustment not only gave them more information, it allowed us to calculate cell weights. Third, Experiment 4 tested the four outcomes used in Experiment 2.

A final change was to the data from which participants selected cases. Experiment 3's cases were drawn from matrices with an equal number of cause-present and cause-absent cases and objective contingencies of zero (if participants had seen complete information). Experiment 4's cases were drawn from matrices based on the cell frequencies of a subset of the trials used in Experiment 2 (Table 3).

Method

Participants. Seton Hall University undergraduates (*N* = 105; 60 female) participated in partial fulfillment of a course requirement (*n* = 52 implausible; *n* = 53 plausible). They ranged in age from 18 to 26 years old (*M* = 18.96, *SD* = 1.25).

Procedure. The procedure was very similar to that of Experiment 3 but employed the four outcomes used in Experiment 2 (car accidents, skin rash, stress and plant growth). Like Experiment 3, over four trials participants learned about four different doctor's offices (or county garages, or greenhouses, or counseling offices). Each of these four "offices" corresponded to one of the bottom four rows of Table 3. Participants were told the total number of cause-present and cause-absent cases and were allowed to make nine choices on each trial. Participants made their choice by clicking a box to the left or right on the computer screen (Figure 8). The order of the cause-present and cause-absent choices was counter-balanced between participants: Half clicked the box on the left to make their cause-present choice and half the one on the right. Participants received feedback regarding the outcome based on their choice (as in Figure 6b). Feedback was determined by randomly drawing without replacement from matrices constructed to match the bottom four rows of Table 3. The feedback screen remained visible until participants pressed the space bar, ending the sub-trial. After making nine selections, participants made causal judgments as in previous experiments. Like Experiment 3, participants received booklets in which they recorded their choices and the outcomes. After completing the choice task, they listed all the possible causes they could think of for all the outcomes (order counterbalanced across participants).

Design and Data Analysis. We cleaned the data by eliminating individual sub-trials for which a participant's response time was less than 250ms (4.9% of sub-trials). We then computed the percent of cause-present choices for each trial and took the average of the participant's four trials as the primary dependent measure. The design of the experiment was a 4 (outcome: skin rashes, accidents, plant growth, stress) x 2 (plausibility: implausible, plausible) x 2 (option order: cause present left, cause present right) mixed design with outcome manipulated within-groups and plausibility and option order between-groups. We again performed 2-level MLM analyses.

Although our primary dependent measure for Experiment 4 was the proportion of cause present choices, because participants saw nine cases for each trial, we also calculated cell weights as in Experiments 1 and 2. Note that because the exact cell information participants received depended both on their choices and on the random draw from the frequency table, each participant observed different frequencies in each of the cells (and hence different objective contingencies).

Results

We excluded two participants because they gave causal judgments outside the range of the scale, leaving *N* = 103 (*n* = 52 implausible; *n* = 51 plausible).

Number of Causes Listed. As in the previous experiments, participants in the plausible (*M* = 5.68, *SD* = 3.14) and implausible (*M* = 6.21, *SD* = 3.29) conditions listed similar numbers of causes, *F* < 1, but the number of causes they listed varied with the outcome, *F*(3, 297) = 30.01, *p* < .001. While participants listed a similar number for car accidents (*M* = 7.26, *SD* = 3.45) and stress (*M* = 7.02, *SD* = 3.79), *p* = 1.00, and for plant growth (*M* = 4.94, *SD* = 2.05) and skin rashes (*M* = 4.55, *SD* = 2.36), *p* = .171, all other pairwise comparisons among the outcomes reached significance, *p*s < .001. The plausibility by outcome interaction did not reach significance, *F*(3, 297) = 1.22, *p* = .303.

Information Choice. We observed different ranges in the listed alternatives for the plausible (0, 27) and implausible (0, 21) groups and thus, restricted the analysis to the smaller range of alternatives. There were 8 multivariate outliers. While the exclusion of individual cases does not necessarily involve excluding whole participants, doing so led to the exclusion of two participants in the plausible condition, leaving *N* = 101 for the choice data (*n* = 52 implausible; *n* = 49 plausible).

Consistent with Experiment 3, we observed an overall positive test bias: Participants chose the cause-present option more often than expected by chance (*M* = .554, *SD* = .13, one-sample *t* versus .50: *t*(100) = 4.11, *p* < .001, *d* = 0.41). The main effect of plausibility was the only effect to approach significance, *F*(1,97) = 3.32, *p* =

.075, *d* = .19, reflecting a tendency for implausible participants to make fewer cause present choices (*M* = .544, SD = .12) than plausible participants (*M* = .569, *SD* = .15). While this effect failed to reach significance, the size of the plausibility effect here (*d* = 0.19) was larger than the significant effect in Experiment 3 (*d* = 0.15). This difference may result from a slightly higher standard error on the effect of plausibility in Experiment 4 (*SE* = .087) relative to Experiment 3 (*SE* = .069); the significant plausibility by number of listed alternatives interaction in Experiment 3 likely reduced the error variance in that experiment. In Experiment 4, we did not observe an interaction between plausibility and the number of listed alternatives, *F* < 1. Nor did the number of listed alternatives independently predict the proportion of cause-present choices, *F* < 1. Finally, none of the effects involving option ordering approached significance (*p*s > .31), which suggests that the new procedure for making selections successfully eliminated the tendency for option ordering to affect the choice data.

Cell Weights. As described for the choice data, we restricted the analysis to the smaller range of alternatives. Four cases were excluded as multivariate outliers. Furthermore, one participant in the implausible condition gave causal judgments of zero on all trials, rendering it impossible to calculate the participant's cell weights. These exclusions left *N* = 98 for the cell weight analysis (*n* = 50 implausible; *n* = 48 plausible).

As seen in Figure 9, we observed differential weighting of the cells as a function of plausibility [*F*(3, 1099) = 4.81, *p* = .002, for the cell by plausibility interaction]. Replicating Experiment 1, implausible participants gave less weight to Cells A and B than did plausible participants [*F*(1, 1099) = 5.42, *p* = .020, *d* = 0.19, collapsing across the absolute value of the means on A and B], but the groups weighted C and D similarly [*F* < 1, collapsing across the absolute value of the means on C and D].

The differential cell weighting among the groups was further modified by the number of listed alternatives [*F*(3, 1099) = 3.81, *p* = .009, for the three-way interaction]. Marginal slopes for this interaction appear in Table 5. Differences between the slopes of the plausible and implausible groups reached significance only for Cell A. Comparing the slopes to zero, implausible participants placed more positive weight on Cell A and more negative weight on Cell B as they listed more alternatives (see Figure 10).

No other effects reached significance [*p* = .071 for the main effect of cell; *p* = .059 for the plausibility by listed alternatives interaction; and all other *p*s ≥ .20].

Discussion

Participants in the implausible condition of Experiment 4 tended to choose the cause-present option less often than those in the plausible condition (*d* = 0.19). They also placed less weight on Cells A and B (*d* = 0.19) and similar weight on C and D. This pattern of weighting and choice is consistent with that observed in the earlier experiments. However, the modifying effect of the number of listed alternatives was not consistent with prior experiments: Participants in the implausible group of Experiment 4 placed more weight on Cells A and B as they listed more alternatives. This effect is also not consistent with predictions of the extant models, nor with a positive test account.

General Discussion

Our experiments addressed two questions: 1) Do participants use data differently when faced with plausible versus implausible causal mechanisms? and 2) Are effects of plausibility moderated by consideration of alternative causes? Our results suggest that the answer to the first question is “yes,” while that for the second is more equivocal.

Does Plausibility Differentially Affect Data Use?

Yes. Across experiments, we found that participants facing implausible causes gave less weight to cells A and B than did those facing plausible causes. This effect of plausibility extended to participants’ choices: Relative to plausible participants, implausible participants more frequently chose cause-absent over cause-present data (Experiments 3 and 4). These results are consistent with the positive test hypothesis.

However, the effect of plausibility on the weighting of cells C and D was less clear. We predicted that participants faced with an implausible cause may adopt an alternative focal hypothesis, leading them to place more weight on Cells C and D (relative to the plausible participants). We did not observe this predicted pattern (but see Appendix C). In contrast to our hypothesis, implausible participants either placed less weight (Experiment 2), or similar weight (Experiments 1 and 4) on cells C and D relative to plausible participants. Thus, while the reduction of the Cell A and B weights for the implausible group was consistent with the positive test account, we did not observe evidence that these participants selected an alternative focal hypothesis, as represented by frequency information in Cells C and D.

Moderating Effect of Considering Alternative Causes?

We observed moderating effects of the number of alternative causes participants listed on both cell weights and on information choice. However, these effects were not consistent across experiments. In Experiment 2, participants in both plausible and implausible conditions placed more weight on disconfirming evidence (Cells B and C) as they listed more alternative causes. In Experiment 3, participants in the implausible condition chose to see more cause-absent information as they listed more alternative causes. Both of these effects may be consistent with a broadly construed positive test account. As individuals think of more alternatives, they place less weight on positive test evidence for the experimentally-introduced cause. However, in Experiment 4, the number of listed alternatives failed to predict the choice data, and in the cell weight data implausible participants placed more weight on Cells A and B as they listed more causes.

Why did we observe these inconsistent results across experiments? It is possible that these inconsistencies resulted from limitations in our method for assessing the consideration of alternative causes: We relied on participants' listing of causes at the end of the experiment to make inferences about what participants were considering during the experiment. Because we collected information on the number of alternative causes after-the-fact, and because this information reflects an individual difference rather than a manipulated difference, we are left with the possibility that the listing of alternative causes and causal judgments are related because of an additional variable that we did not assess. A more stringent test of our hypothesis would involve creating fake causal worlds in which the objective number of alternative causes supported by events is manipulated.[4]

Indeed, we postulated that differences in the ability to think of alternatives may stem not only from individual differences (i.e., overall, some people think of more causes than do others; Dougherty et al., 1997; Sprenger & Dougherty, 2012), but also from differences in the causal scenarios themselves (Cummins, 1995). That is, some outcomes support more causes than others (e.g., the number of causes of a nuclear explosion versus the number of causes of stress). We observed such a difference here: Across experiments, participants consistently listed more causes of car accidents and stress than of skin rashes and plant growth. Why did we fail to observe effects of the outcomes employed in the causal scenarios? One possibility is that because of shared variance it is not possible to observe effects of the number of alternatives outcomes support when the number of alternatives participants can list is already entered in an analysis.[4] An additional possibility is that manipulation of the number of alternatives supported by the outcomes was not robust enough. In Experiments 2 and 4, the outcomes that supported “more” versus “fewer” alternatives, while statistically different from each other, differed by an average of only two to three causes. While our pilot testing also identified two kinds of events for which people could list even fewer alternatives (colon cancer and chemical reactions; see Appendix D), our more pressing interest was in investigating the moderating effects of individual differences in the consideration of causal alternatives on plausibility. Thus, we avoided choosing outcomes that were at the low extreme in terms of the number of causes they supported, for which participants might be at floor when listing alternatives. Future work will need to more systematically address how the ability of outcomes to support more versus fewer causes affects evidence weighting.

In sum, we may have observed inconsistent moderating effects of the consideration of alternative causes because of limitations of the method we employed for its measurement. Future work correcting for some of these limitations is necessary to clarify exactly how the consideration of alternative causes affects the choice and weighting of data.

Implications for Theories of Causal Inference from Data

The effects of plausibility observed in the current set of experiments were clearly inconsistent with our modification of Cheng's (1997) causal power (Figure 2a and Appendix A). That model predicts decreases in plausibility associated with increasing weight on Cells A and D (confirming information) and decreasing weights on Cells B and C (disconfirming information). In contrast, we observed decreases in plausibility to be associated with decreasing weights on Cells A and B, and either similar (Experiments 1 and 4) or decreased weights on Cells C and D (Experiment 2).

Both Fugelsang and Thompson (2003) and the modification of Griffiths and Tenenbaum's (2005) causal support (Figure 2b and Appendix A) predict a reduction in the weighting of the data from all cells. The consistent observation across experiments that implausible participants gave less weight to Cells A and B is partially commensurate with these accounts. However, across experiments we observed differential weighting of the cell frequency data among plausible and implausible participants not predicted by these accounts. In Experiments 1 and 4, implausible participants gave less weight to Cells A and B, and similar weight to cells C and D relative to plausible participants. In Experiment 2, while implausible participants gave less weight to all cells than did plausible participants, they gave equal weight to the cells, and plausible participants weighted A > B and C > D (Figure 4).

Given that Fugelsang and Thompson (2003) and the modified causal support predict that implausibility leads to an across the board reduction in cell weights (with no differential weighting patterns for plausible and implausible causes), our results are inconsistent with those predictions. Rather, our results may be consistent with a more broadly construed positive test strategy account – one in which implausibility functions primarily to reduce the positive test of the experimentally-presented cause (i.e., reduce the weighting of cause-present information).

An additional consideration not explicitly addressed by most extant formal models of causal learning, nor by our experiments, is the relatively rarity of the events involved (cf. Hattori & Oaksford, 2007). Heavy weighting of Cell A information depends on the assumption that the presence of events is rare, while their absence is quite common. Given rare events, Cell A better-discriminates between two causal alternatives than does Cell D (Anderson, 1990; McKenzie, 2004; McKenzie & Mikkelsen, 2007). Consistent with this argument, participants place more weight on Cell D information for events whose *absence* is rare (McKenzie & Mikkelsen, 2007). Our modeling exercise (Appendix A) also supports the importance of the rarity assumption. When we gave the models data generated by randomly sampling values for the frequencies in the each of the four cells [$p(\mathrm{C}) \approx p(\mathrm{E}) \approx .50$], the models did not predict the cell-weight inequality typically observed for generative causes. However, when we introduced a rarity bias on the presence of events into the data-generation process the model predictions approximated the cell-weight inequality and when the presence of events was common, the models predicted heavy weighting of Cell D information (Appendix A). Across our Experiments 1 and 2, the causes and outcomes were moderately frequent [$p(C) = p(E) = .48$ in Experiment 1 and $p(C) = p(E) = .50$ in Experiment 2]. Thus, differences in the relative rarity of the causes and outcomes cannot explain any of the data-weighting differences we observed for Cells C and D across those experiments. Nonetheless, a complete theory of evidence weighting will need to account for plausibility, the consideration of alternative causes, and for the effects of the relative rarity of the causes and their outcomes.

As a final point, our results have implications for researchers testing hypotheses about causal reasoning from covariation data. Participants may use this data differently with different causal scenarios affording the consideration of more or fewer alternatives. Additionally, our data suggest that the results of laboratory experiments using fabricated causes and outcomes (of which participants have no prior knowledge), may not generalize well to situations in which individuals make causal inferences regarding events for which they have prior beliefs (see also Cummins, 1995).

Conclusion

Across experiments we observed that participants use and weighting of data varied with the plausibility of a cause’s mechanism in a manner partially consistent with Fugelsang and Thompson’s (2003) dual process model and partially consistent with a broadly-construed positive test account. While these effects were further modified by participants’ ability to consider alternative causes of the outcome, the manner in which they were modified varied across experiments. Additional factors may also influence participants’ cell weights. Determination of precisely how these factors interact with plausibility and consideration of alternatives awaits future systematic investigation.

Footnotes

[1] We refrained from using the phrase *confirmation bias* for two reasons: 1) Confirmation bias does not necessarily result from a positive test strategy. Rather, confirmation bias results from a combination of factors at both test and evaluation stages of hypothesis testing (Klayman, 1995; McKenzie, 2004; Slowiaczek, Klayman, Sherman, & Skov, 1992). 2) The phrase confirmation *bias* implies non-normativity, but the relative non-normativity of seeking confirming evidence is disputed: Under certain conditions seeking confirmation may be normative (e.g., Austerweil & Griffiths, 2011).

[2] In their Appendix C, Lu et al. (2008) introduce a specific prior of zero on the strength of background causes in the blicket detector paradigm, because participants learn that only blickets and no other objects activate the blicket detector. Although they have modeled a specific prior, this prior is on the strength of the background causes rather than on the believability of the focal cause.

[3] Taking a weighted average of the prior does not capture previous findings that prior causal mechanism information interacts with new contingency evidence (Fugelsang & Thompson, 2003). However, the weighted average reflects a Bayesian belief updating.

[4] However, an additional observation suggests that we are both tapping the consideration of alternative causes and that the number of alternatives supported by different outcomes may be important: For all experiments we performed preliminary analyses investigating the effects of plausibility on cells weights and information choice excluding the number of listed alternatives – i.e., without the moderator. In Experiment 2, we observed an effect of outcome type in these analyses: Participants' cell weights varied with the outcome [cell by outcome interaction, $F(3, 842) = 3.93$, $p = .008$].

Overall, participants placed less weight on the data when the outcomes supported more (car accidents and stress) versus fewer alternatives (skin rash and plant growth), with this effect reaching significance for Cells B and D (*p*s < .04). This effect of outcome type disappeared once the number of alternatives listed at the end of the experiment was added to the model, consistent with the assumption that differences between the outcomes were driven by the number of alternative causes those outcomes supported.

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Table 1. Summary of the candidate causes used for the plausible and implausible conditions for each of the four outcomes.

Condition	Outcome			
	Skin Rash	Car Accidents	Plant Growth	Stress
Plausible	Hiking in woods	Severed brake lines	Adding fertilizer	Having lots of deadlines
Implausible	Studying vocabulary	Having leather seats	Being in a blue pot	Eating lots of fruits & veggies

Note. Skin rash and car accident outcomes appeared in Experiments 1 and 3. All four outcomes appeared in Experiments 2 and 4.

Table 2. Cell frequencies and objective contingencies for each of the trials in Experiment 1.

Cell A	**Cell B**	**Cell C**	**Cell D**	***phi***
1	2	4	8	0.000
4	1	8	2	0.000
8	2	4	1	0.000
4	8	1	2	0.000
2	1	8	4	0.000
2	8	1	4	0.000
8	4	2	1	0.000
1	4	2	8	0.000
3	7	1	4	0.107
8	1	4	2	0.272
2	4	1	8	0.272
2	7	0	6	0.320
2	0	7	6	0.320
5	0	7	3	0.354
8	2	1	4	0.577
4	1	2	8	0.577

Table 3. Cell frequencies and objective contingencies for each of the trials in Experiment 2.

A	B	C	D	*phi*
2	5	2	1	-0.356
1	2	5	2	-0.356
2	2	5	1	-0.356
2	5	1	2	-0.048
1	2	2	5	0.048
5	2	1	2	0.356
5	1	2	2	0.356
2	1	2	5	0.356
1	2	20	15	-0.238
15	20	2	1	-0.238
20	15	1	2	0.238
2	1	15	20	0.238
1	15	20	2	-0.847
2	20	15	1	-0.847
15	1	2	20	0.847
20	2	1	15	0.847

Table 4. Marginal slopes for predicting cell weights from number of alternatives listed in Experiment 2.

Cell	***b***	***β***	***p***
A	0.0040	0.06	.566
B	-0.0122*	-0.25*	.032
C	-0.0121˜	-0.21˜	.061
D	0.0045	0.09	.421

Note: SE on *b* was 0.01. *p* indicates test of slope against zero. * denotes significantly from zero at *p* < .05; ˜ at *p* < .10. Recall that cells B and C are normatively negatively weighted. Thus, negative slopes on these cells indicate an increasing cell-weight as the number of alternatives increases.

Table 5. Marginal slopes for predicting cell weight from number of listed alternatives as a function of cell and plausibility of the causal mechanism in Experiment 4.

	Implausible Causal Mechanism		Plausible Causal Mechanism		Simple Effects: Implausible Vs. Plausible
Cell	*b*	*β*	*b*	*β*	
A	0.082*	0.23*	-0.042	-0.12	*p* = .010
B	-0.069*	-0.20*	-0.004	-0.01	*p* = .178
C	-0.031	-0.09	0.041	0.16	*p* = .112
D	0.028	0.09	0.023	0.06	*p* = .898

Note: *SE* on *b* was 0.04. * denotes significantly from zero at *p* < .05. Recall that cells B and C are normatively negatively weighted. Thus, negative slopes on these cells indicate an increasing cell-weight as the number of alternatives increases.

Figure Captions

Figure 1. 2 x 2 Contingency Table with cell labels representing the number of times the cause and the outcome were jointly present (Cell A), jointly absent (Cell D), or occurred alone (Cells B and C, respectively).

Figure 2. Absolute value of cell weights based on posterior causal power resulting from a weighted average of (A) causal power (Cheng, 1997) or (B) causal support (Griffiths & Tenenbaum, 2005) and priors on the relation between the cause and outcome with moderately common causes and outcomes [p(C) = p(E) = .50]. A prior of .9 indicated high plausibility, .5 moderate plausibility, and .1 low plausibility.

Figure 3. Absolute value of the mean cell weights of Experiment 1**.** Error bars are ±1 SE.

Figure 4. Absolute value of mean cell weights in Experiment 2. Error bars are ±1 SE.

Figure 5. Scatterplot with regression slopes for predicting Cell B and C weights from the number of alternatives listed in Experiment 2 (averaged over outcome and plausibility). Note that because Cells B and C are normatively negatively weighted, increasing negative values indicates an increase in the cell weight.

Figure 6. (A) Choice and (B) feedback screens in Experiment 3.

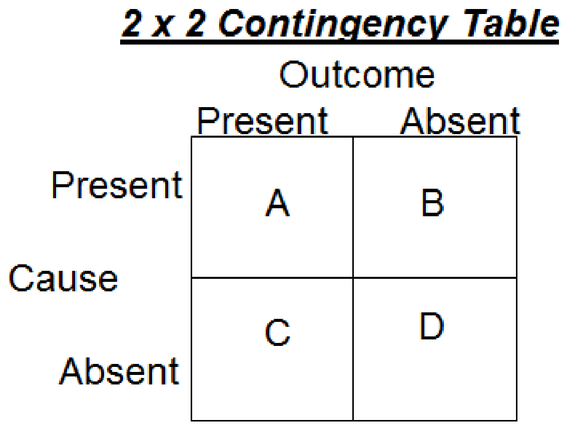
Figure 7. Scatterplot with regression slopes for predicting proportion of cause present choices from the number of causes listed (averaged over outcome) for the (a) implausible and (b) plausible conditions of Experiment 3.

Figure 8. Choice screen in Experiment 4.

Figure 9. Absolute value of mean cell weights of Experiment 4. Error bars are ±1 SE.

Figure 10. Regression slopes for predicting the mean cell weight of implausible participants for Cells A and B in Experiment 4. Note, increasing negative values on Cell B indicate an increase in weight on that cell.

Figure 1. 2 x 2 Contingency Table with cell labels representing the number of times the cause and the outcome were jointly present (Cell A), jointly absent (Cell D), or occurred alone (Cells B and C, respectively).



2 x 2 Contingency Table

		Outcome	
		Present	Absent
Cause	Present	A	B
	Absent	C	D

Figure 2.

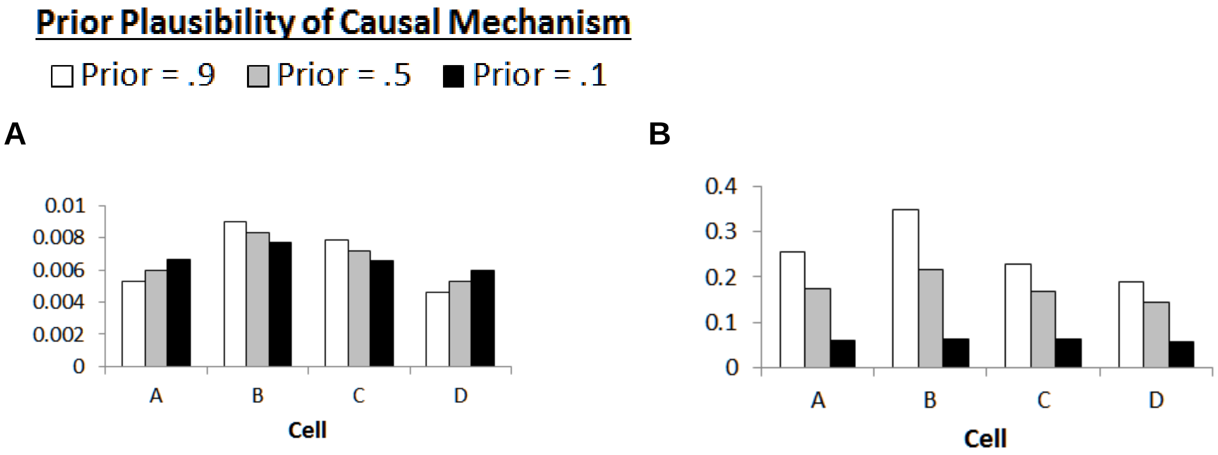

Prior Plausibility of Causal Mechanism
□ Prior = .9
▨ Prior = .5
■ Prior = .1
A
0.01
0.008
0.006
0.004
0.002
0
A
B
C
D
Cell
B
0.4
0.3
0.2
0.1
0
A
B
C
D
Cell

Figure 3.

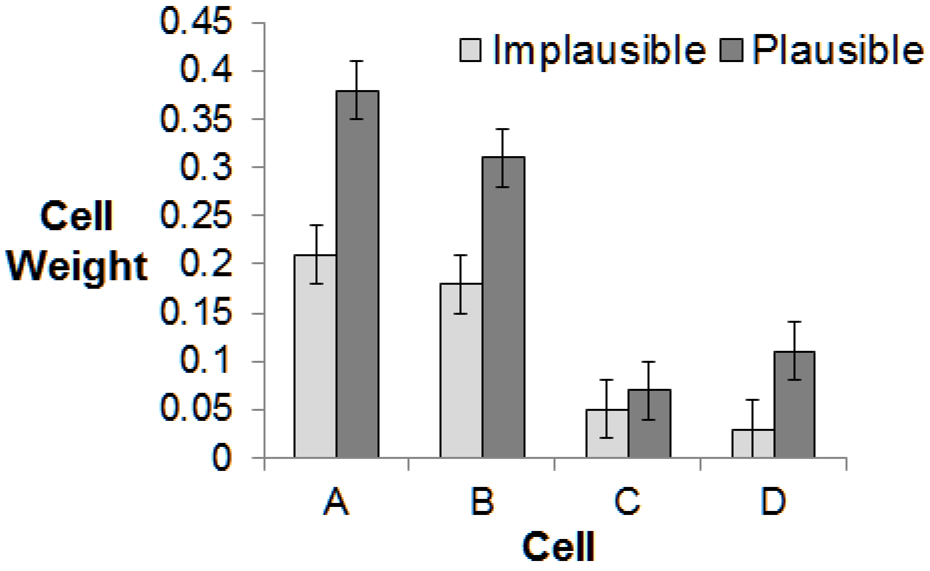

0.45
0.4
0.35
0.3
0.25
0.2
0.15
0.1
0.05
0
Cell Weight
□ Implausible ■ Plausible
A
B
C
D
Cell

Figure 4.

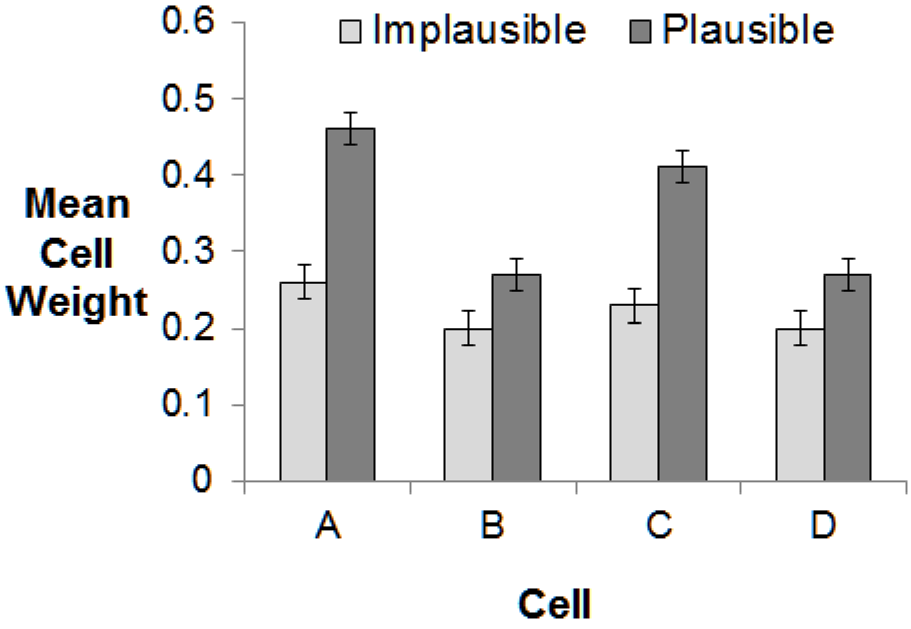

Implausible
Plausible
0.6
0.5
0.4
0.3
0.2
0.1
0
Mean Cell Weight
A
B
C
D
Cell

Figure 5.

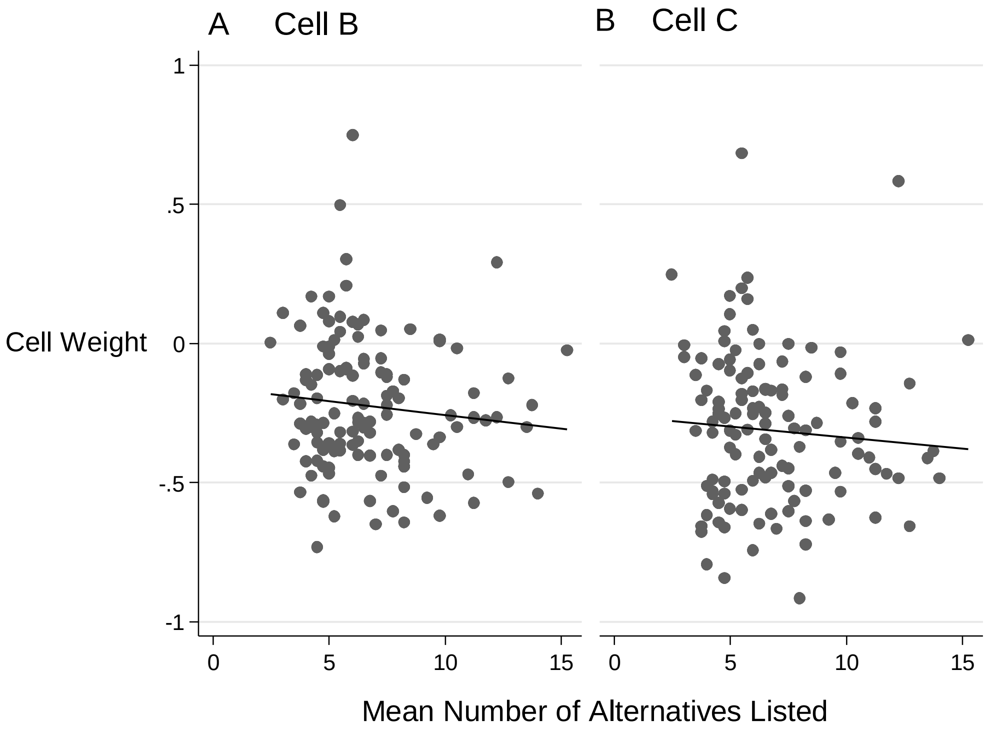
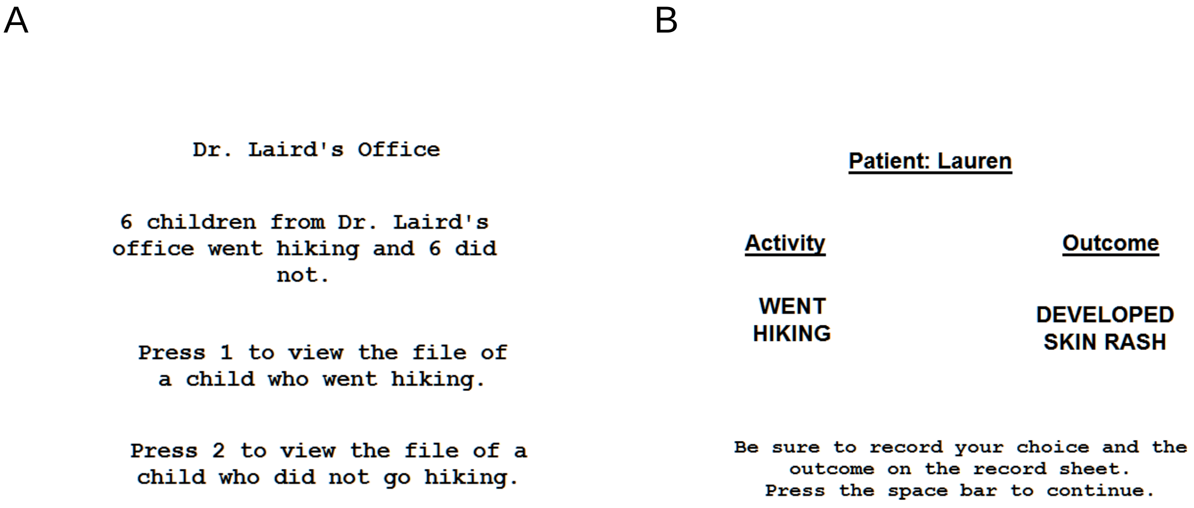

A
Cell B
B
Cell C
Cell Weight
1
.5
0
-.5
-1
0
5
10
15
0
5
10
15
Mean Number of Alternatives Listed

Figure 6.



Dr. Laird's Office

6 children from Dr. Laird's office went hiking and 6 did not.

Press 1 to view the file of a child who went hiking.

Press 2 to view the file of a child who did not go hiking.

Patient: Lauren

Activity	**Outcome**
WENT HIKING	DEVELOPED SKIN RASH

Be sure to record your choice and the outcome on the record sheet.
Press the space bar to continue.

Figure 7.

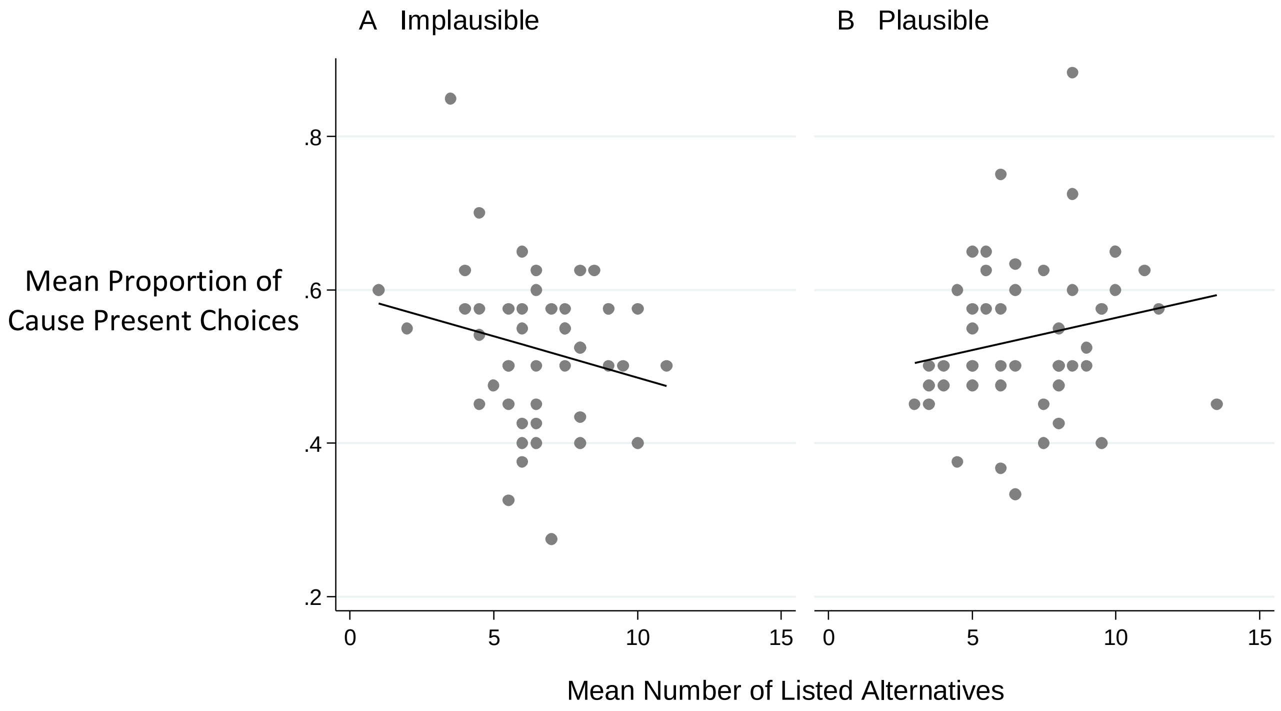
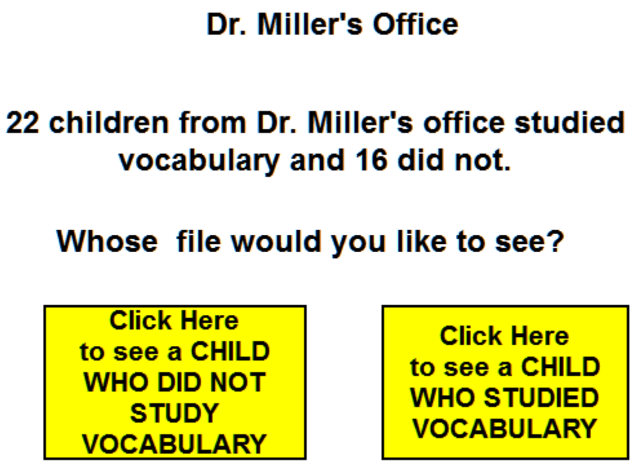

A Implausible
B Plausible
Mean Proportion of Cause Present Choices
.8
.6
.4
.2
0
5
10
15
0
5
10
15
Mean Number of Listed Alternatives

Figure 8



Dr. Miller's Office

22 children from Dr. Miller's office studied vocabulary and 16 did not.

Whose file would you like to see?

Figure 9.

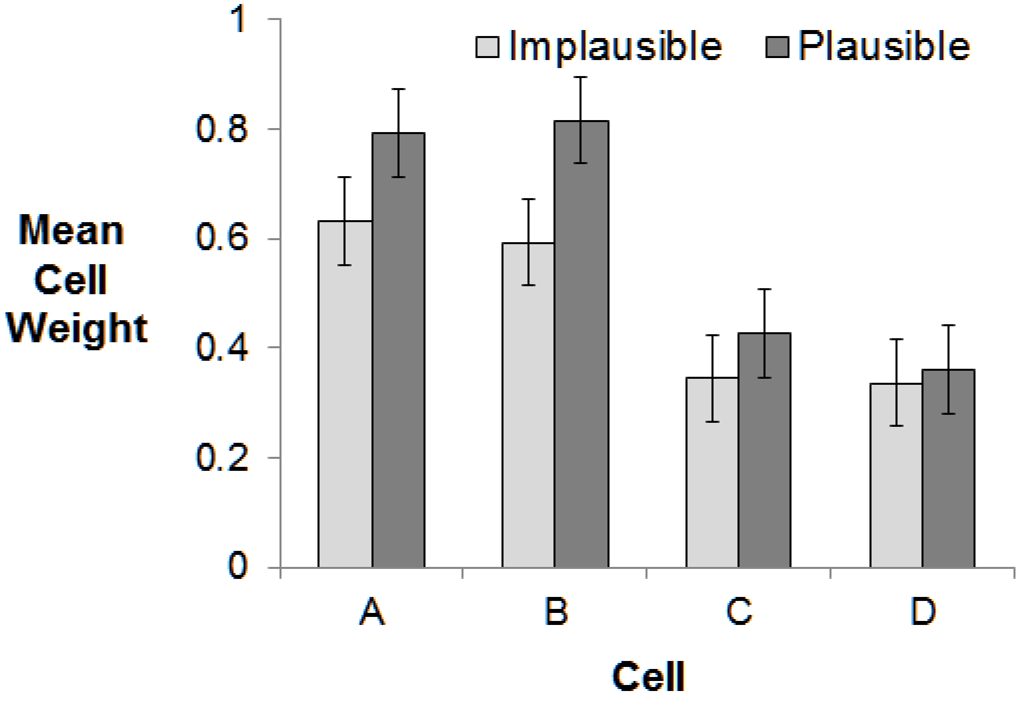
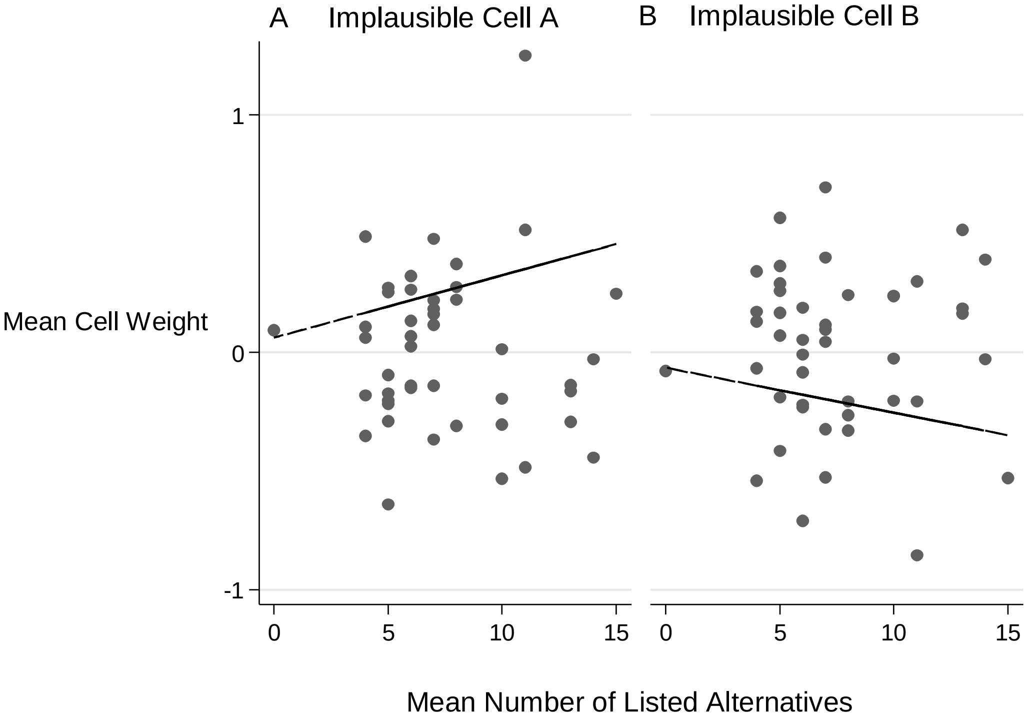

1
0.8
0.6
0.4
0.2
0
□ Implausible
■ Plausible
Mean Cell Weight
A
B
C
D
Cell

Figure 10.


A
Implausible Cell A
B
Implausible Cell B
Mean Cell Weight
1
0
-1
0
5
10
15
0
5
10
15
Mean Number of Listed Alternatives

Appendix A

Modeling Method and Results

Three models enhanced to represent plausible versus implausible causal mechanisms are presented: power-PC (Cheng, 1997), causal support (Griffiths & Tenenbaum, 2005), and sparse and strong (SS) priors (Lu et al., 2008). Each model's predictions were assessed by computing its measure of causal strength for 1000 randomly generated 2 x 2 contingency tables and then regressing that measure on the tables' cell frequencies. For each contingency table, the sample size *N* was first drawn from a uniform distribution bounded by [100, 200]. Two draws of size *N* from a binomial distribution with parameter *p*, representing the presence/absence of the cause and effect, respectively, were then crossed to form a contingency table. A table yielding a cause/effect correlation ≤ 0 was discarded and this process repeated until 1000 tables were accumulated. Simulation results are reported for values of p = .1, .5, and .9, corresponding to cases in which the cause and effect are both rare, occur with probability ≈.5, or are both common.

Power-PC With Priors

The plausibility of the causal mechanism was rendered as a prior distribution over causal power. Because power is a probability in the range [0-1], the prior was represented as a beta distribution. The shape of a beta distribution is controlled by two parameters, α and β, constrained to be positive real numbers. The mean of the prior distribution, $Beta\ (\alpha_{prior}, \beta_{prior})$, is $\alpha_{prior} /(\alpha_{prior} + \beta_{prior})$, was set to either .1 (a prior belief that causal power is low, corresponding to a causal mechanism of low plausibility), .5

(moderate plausibility), or .9 (high plausibility); the prior confidence in that estimate (i.e., how peaked the distribution is), represented by $\alpha_{prior} + \beta_{prior}$, was held constant at 100. (100 can be interpreted as the number of "prior observations.") For each of the 1000 samples, the empirical causal power was calculated in the usual way: $p_i = \Delta P_i/(1 - P(e|{\sim}i))$ where $\Delta P_i = P(e|i) - P(e|{\sim}i)$ (Cheng, 1997). The parameters of a beta distribution that characterizes the information about causal power inherent in the sample were then derived: $\alpha_{empirical} = p_i N$ and $\beta_{empirical} = (1 – p_i)N$, where N is the size of the sample. The posterior distribution is given by *Beta* ($\alpha_{prior} + \alpha_{empirical}$, $\beta_{prior} + \beta_{empirical}$); the posterior estimate of causal power was defined as the mean of the posterior. The results of predicting the posterior causal power of the 1000 samples from the four cell frequencies are shown in Figures 2 and A1B for the case in which the cause and effect are both moderately common, that is $p(C) \approx p(E) \approx .5$.

As mentioned two additional runs of our simulation were conducted where *C* and *E* were both rare ($p = .1$) or both common (.9). Although the weights of the cells relative to each other changed (Cell A was most important when *C* and *E* were rare, Cell D was most important when they were common), the effect of the prior was the same as in Figure 2: As prior causal strength increased, Cells A and D decreased in importance and B and C increased (compare Figure A1A – A1C).

Causal Support

Griffiths and Tenenbaum (2005) assume that causal judgments reflect learners' beliefs in the (log of the) relative probability of two hypotheses, namely, that there is a causal relationship between the two variables ("Graph 1") and that there isn't ("Graph 0"). This measure, *causal support*, is defined as,

$$support \ = \ log \frac{p(Graph\ 1|D)}{p(Graph\ 0|D)} = log \frac{p(D|Graph\ 1)}{p(D|Graph\ 0)} + log \frac{p(Graph\ 1)}{p(Graph\ 0)} \tag{1}$$

where *D* is the observed data. When the prior probabilities of the two hypotheses are equal, Eq. 1 becomes,

$$support \ = log \frac{p(D|Graph\ 1)}{p(D|Graph\ 0)} \tag{2}$$

where

$$p(D|Graph\ 1) \ = \int_0^1 \int_0^1 p(D|w_0, w_1, Graph\ 1) p(w_0, w_1|Graph\ 1) \ dw_0 dw_1 \tag{3}$$

$$p(D|Graph\ 0) \ = \int_0^1 p(D|w_0, Graph\ 0) p(w_0|Graph\ 0) \ dw_0 \tag{4}$$

w_1 and w_0 represent the strength of the candidate and alternative cause, respectively. Griffiths and Tenenbaum make no assumptions about the causal powers in either graph. Rather, they evaluate Eq. 3 by uniformly sampling over all possible values of w_1 and w_0. (Eq. 4 has an analytic solution.)

We elaborate causal support in two ways to represent plausibility. First, rather than assuming a uniform prior on the causal power between the cause and effect variable in Graph 1 (i.e., on w_1), we make the same assumptions as we did in the previous section, namely that the w_1 prior is represented as a beta distribution with a mean of .1, .5, or .9 (the number of “prior observations,” α_{prior} + β_{prior}, was to set to 10). That is, for Graph 1 the prior over parameters becomes

$$p(w_0, w_1|Graph\ 1) \ = p(w_0|Graph\ 1)p(w_1|Graph\ 1) \tag{5}$$

where $p(w_0|Graph\ 1)$ is uniformly distributed and $p(w_1|Graph\ 1)$ is beta distributed with parameters α_{prior} and β_{prior}.

For each of the 1000 data samples, Eq. 3 was evaluated by uniformly sampling w_1 and w_0 a million times. For each of those samples, the prior (Eq. 5) was multiplied by the likelihood of the data (see Griffiths & Tenenbaum, Eq. 7), to compute the posterior. The mean of the posterior distribution that resulted was taken as the value of $p(D|Graph\ 1)$ and causal support was then computed according to Eq. 2. The 1000 values of support computed in this manner were regressed on the cell frequencies.

The results of this simulation revealed a pattern of cell weights qualitatively similar to the prediction of Fugelsang and Thompson (2003): with decreases in plausibility there is a decrease in cell weights for all cells (see Figure 2 and Figure A1D to A1F). Although the same pattern of results obtained when *C* and *E* were both rare (.1) or both common (.9), we further observed that like causal power, cell D was most heavily weighted when C and E were common.

The second elaboration involves changing the prior probabilities of Graph 1 and Graph 0 in Eq. 1 rather than placing a prior on w_1 in Graph 1. Note that this elaboration is based on a different sense of plausibility, one in which an “implausible” causal mechanism is not necessarily weak but rather unlikely to exist and a “plausible” one is not necessarily strong but rather likely to exist. In fact, this elaboration predicts no effect of changing the prior probabilities of Graph 1 and Graph 0 on cell weights. Why it does so is transparent from Eq. 1. Because any change to Eq. 1's second term merely adds a constant to causal support, no change to the weights is observed when it is regressed onto the cell frequencies.

Strong and Sparse Priors (SS)

Lu et al. (2008) assume that causal strength judgments are influenced by a "sparse and strong" (SS) prior on causal power that reflects learners' biases that causes are both strong and few in number. Applied to standard causal learning situations such as those studied here, this assumption entails a prior in which *C* is either a strong cause of *E* (and alternatives causes of *E* are weak or nonexistent) or *C* is a weak or nonexistent cause of *E* (which is thus explained by strong alternative causes instead). Although Lu et al. emphasize learning situations in which prior knowledge is absent and so these *generic priors* dominate, they allow for the potential influence of prior knowledge via *specific priors*. We model plausibility in this framework in the same way as for causal power, namely, with (specific) priors in the form of a beta distribution that reflect the prior expectation that power is weak (.1), moderate (.5), or strong (.9). (α_{prior} + β_{prior} was held constant at 10.) The generic and specific priors were multiplied yielding,

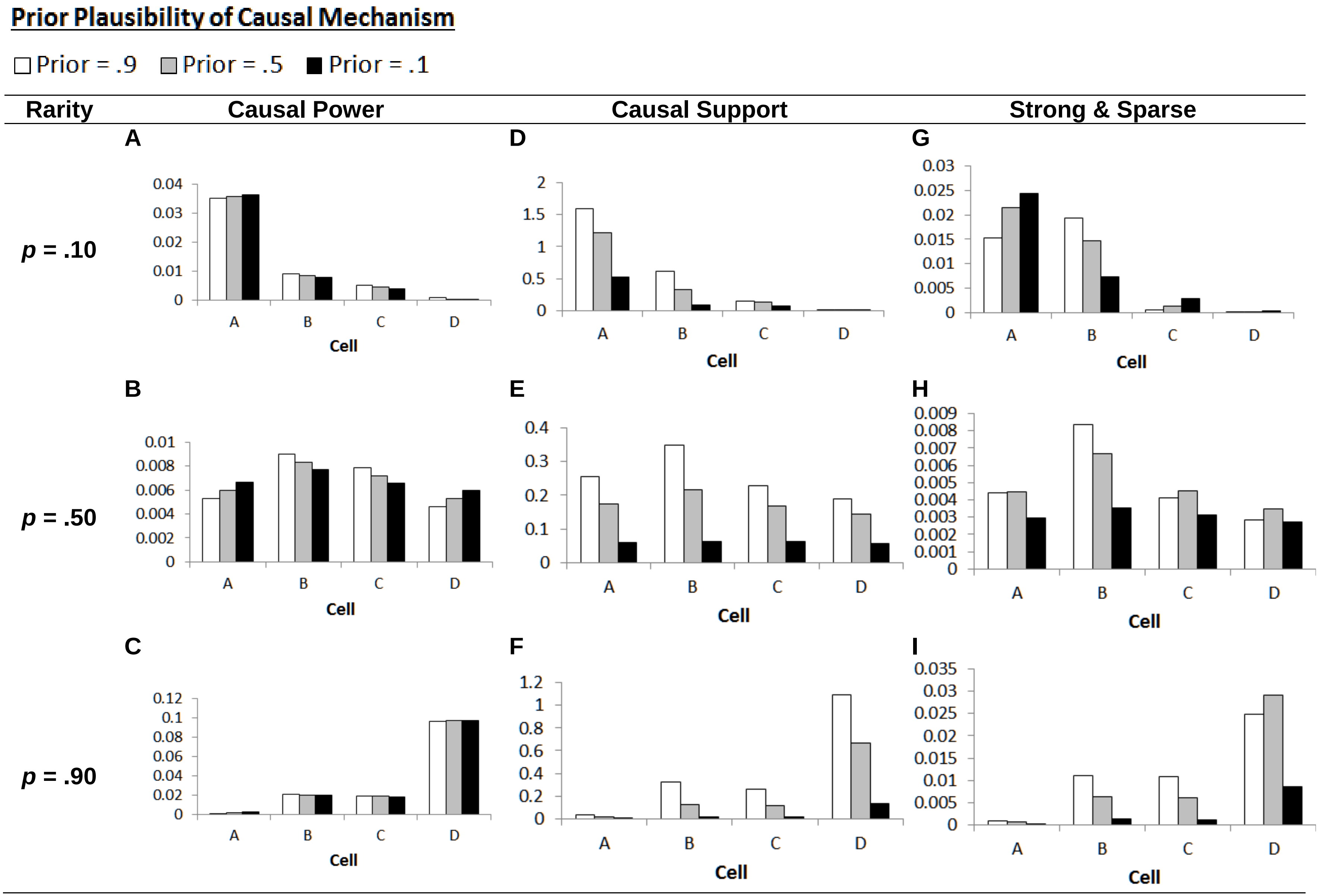
$$p(w_0, w_1|Graph1) \infty (e^{-\gamma w_0 - \gamma(1-w_1)} + e^{-\gamma(1-w_0)-\gamma w_1}) dbeta(w_1|\alpha_{prior} + 1, \beta_{prior} + 1) \tag{6}$$

where w_1 and w_0 again represent the strength of the candidate and alternative cause, respectively, and *dbeta* is the Beta density function. Following Lu et al., the γ parameter, which controls the strength of the SS priors, was set to 5. For each of the 1000 data samples, the above prior was uniformly sampled over w_1 and w_0 a million times; for each of those the posterior was computed by multiplying it with the likelihood of the data. The estimate of causal strength was taken to be the mean value of w_1 in the resulting posterior distribution.

Unlike causal power and causal support, this model predicted different effects of plausibility depending on whether C and E were rare or common. For rare events (Figure A1G), the effects of plausibility on Cells A and B look like those of causal power (Figure A1A): decreases in plausibility are associated with an increase in Cell A weight and decrease in Cell B weight. When the events are common (Figure A1I), plausibility results in decreases in weighting on most cells (more like causal support).

Figure Caption

Figure A1. Cell weight predictions with high (prior = .9), medium (prior = .5), and low (prior = .1) prior plausibility of the causal relations for causal power (graphs A through C), causal support (graphs D through F) and strong and sparse power (graphs G through I) for rare (*p* = .1), moderately common (*p* = .5), and highly common (*p* = .9) events.


Prior Plausibility of Causal Mechanism
□ Prior = .9 ▨ Prior = .5 ■ Prior = .1
Rarity
Causal Power
Causal Support
Strong & Sparse
p = .10
p = .50
p = .90
A
B
C
D
E
F
G
H
I
0.04
0.03
0.02
0.01
0
0.01
0.008
0.006
0.004
0.002
0.12
0.1
0.08
0.06
2
1.5
1
0.5
0.4
0.3
0.2
0.1
1.2
0.8
0.6
0.035
0.025
0.015
0.005
0.009
0.007
0.005
0.003
0.001
A
B
C
D
Cell

Appendix B

Residual Covariance Structures and Random Effects Results

EXPERIMENT 1

Cell Weighting

Covariance Structure. Preliminary likelihood ratio tests suggested the need for modeling different variances for the plausible and implausible conditions [χ^2 *(1)* = 17.40, *p* < .001, compared to homogeneous variance and covariance]. Variance for the implausible and plausible groups was 0.082 and 0.122, respectively.

ICC and Random Effects. The intraclass correlation coefficient (ICC) was 0.00, indicating little within-participant correlation among the cell weights. As to be expected given the ICC of 0, variance attributable to the random effects of participant was σ_0^2 = 0.00, *SE* = 0.002.

EXPERIMENT 2

Number of Causes Listed

Covariance Structure. The covariance structure with homogeneous variances and zero covariance best fit the data (all *p*s > .81 relative to more complex structures).

ICC and Random Effects. The ICC was .46, indicating a moderate within-participant correlation in the number of causes listed. The model yielded significant effects of participants' random intercepts, σ_0^2 = 5.57, *SE* = 0.91, *Z* =6.14, *p* < .001.

Cell Weighting

Covariance Structure. We modeled different variances for each of the four cells in the residual covariance matrix [χ^2 *(3)* = 21.22, *p* < .001, compared to homogeneous variance structure]. Variances for Cells A, B, C, and D were 0.159, 0.108, .140, and 0.105, respectively.

ICC and Random Effects. The ICC was 0.00 and variance attributable to the random effects of participants was σ_0^2 = 0.00, *SE* = 0.00.

EXPERIMENT 3

Number of Causes Listed

Covariance Structure. The likelihood ratio tests suggested the need for modeling different variances for plausibility with covariances equal to zero [χ^2 *(3)* = 30.08, *p* < .001, compared to homogeneous variances]. Variances for the implausible and plausible conditions were 4.50 and 1.62, respectively.

ICC and Random Effects. The ICC was .23, indicating a small to moderate within-participant correlation in the number of causes listed. Consistent with the moderate ICC, participants' random intercepts reached significance, σ_0^2 = 4.61, *SE* = 0.99, *Z* = 4.64, *p* < .001.

Cause Present Choices

Covariance Structure. We modeled different variances for plausibility and presentation order [χ^2 *(3)* = 25.17, *p* < .001, compared to homogeneous variance structure]. Variance for the implausible, cause-present first condition was 0.009; for the implausible, cause-present second it was 0.009; for the plausible, cause-present first it was 0.005; and for the plausible, cause-present second it was 0.023.

ICC and Random Effects. The ICC was .26, indicating a small to moderate within-participant correlation. The random effect of participants' intercepts reached significance, σ_0^2 = 0.0024, *SE* = 0.0011, *z* = 2.71, *p* = .030.

EXPERIMENT 4

Number of Causes Listed

Covariance Structure. We modeled different variances for each outcome with covariances equal to zero [χ^2 *(3)* = 49.37, *p* < .001, compared to homogeneous variance structure]. Variances for the skin rash, car accidents, plant growth, and stress outcomes were 1.99, 5.32, 1.14, and 6.51, respectively.

ICC and Random Effects. The ICC was .48, indicating a moderate within-participant correlation. Consistent with the moderate ICC, participants random intercepts reached significance, σ_0^2 = 3.89, *SE* = 0.64, *Z* = 6.06, *p* < .001.

Cause Present Choices

Covariance Structure. We modeled different variances for plausibility and option order [χ^2 *(3)* = 8.69, *p* = .034, compared to homogeneous variance structure]. Variance for the implausible, cause-present left condition was 0.011; for the implausible, cause-present right it was 0.011; for the plausible, cause-present left it was 0.007; and for the plausible, cause-present right it was 0.015.

ICC and Random Effects. The ICC was .34, indicating a small to moderate within-participant correlation. The random effect of participants' intercepts reached significance, σ_0^2 = 0.005, *SE* = 0.001, *z* = 4.36, *p* < .001.

Cell Weighting

Covariance Structure. We modeled different variances for outcome [χ^2 *(2)* = 18.62, *p* < .001, compared to homogeneous variance structure]. Variance for skin rashes was 0.762; for accidents it was 1.196; for plant growth it was 0.970 and for stress it was 1.144.

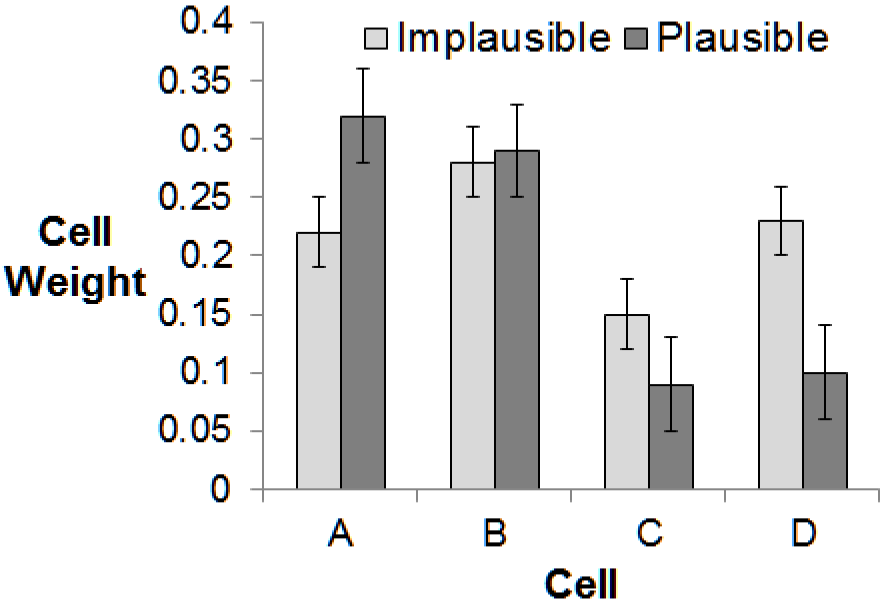
ICC and Random Effects. The ICC was .00, indicating no within-participant correlation. Accordingly, the random effect of participants' intercepts did not reach significance, σ_0^2 = 0.000, *SE* = 0.000, *z* = 0.22, *p* > .05.

Appendix C

Cell Weight Results for Cambridge Participants in Experiment 1

Among the 48 Cambridge participants (*n* = 24 each in the plausible and implausible conditions), none were identified as outliers. As can be seen in Figure C1, there was a tendency for implausible participants to place less weight on Cells A and B, and more weight on Cells C and D, relative to plausible participants [*F*(3, 314) = 4.42, *p* = .005, for the plausibility by cell interaction]. However, the difference between the groups only reached significance for Cells A and D [*p* = .046, *d* = 0.50 for Cell A; *p* = .729, *d* = 0.05 for Cell B; *p* = .236, *d* = 0.26 for Cell C; and *p* = .005, *d* = 0.74 for Cell D]. This pattern of results is qualitatively different than that observed among the US implausible participants, who when compared to the plausible group, weighted Cells A and B less, but Cells C and D about the same. The results of the UK sample are more commensurate with the positive test prediction introduced here. While these results suggest potential cultural differences either in the cell weighting or in the interpretation of the causal cover stories, it was beyond the scope of the current paper to further investigate these.

Figure C1. Absolute value of the mean cell weights for the UK participants in Experiment 1.



Appendix D

Cover Story Piloting

We conducted a pilot study to identify additional outcomes to be used in cover stories for Experiments 2 and 4. Seton Hall University students (n = 102; 70 female) received course credit for completing the study. Participants tested in the laboratory in groups of up to four, completing the study using *Survey Monkey*. They were asked to list as many possible causes as they could think of for these randomly-ordered outcomes: (1) car accident; (2) skin rash; (3) plant growth; (4) colon cancer; (5) chemical reaction; (6) stress; (7) fatigue; and (8) losing one's job. Each outcome was presented one at a time. Participants listed all the causes they could think for each by typing their responses in the response box and then hit "submit" to continue to the next outcome. The outcomes were selected based on conversations with our respective research teams and solicitations on social networking sites. We hoped to identify a set of outcomes that would vary in the number of causes they would elicit. Table D1 depicts the number of causes participants listed for each outcome. Repeated-measures ANOVA indicated a significant effect of outcome type, $F(7,707) = 63.88$, $p < .001$. Results of Tukey's HSD post-hoc are indicated with superscripts in Table D1.

Table D1. Number of causes listed in the pilot study as a function of outcome.

Outcome	**M**	**SD**
Stress[a]	7.07	3.70
Car Accident[a]	7.01	4.06
Lose One's Job[b]	5.76	3.08
Fatigue[b,c]	5.09	3.82
Plant Growing[c,d]	4.33	1.98
Skin Rash[d]	4.04	2.04
Colon Cancer[e]	2.56	1.57
Chemical Reaction[e]	2.30	1.62

Note: Superscripts indicate outcomes not significantly different via Tukey’s HSD.