Question Framing Modulates the Cause Density and Effect Density Biases in Causal Illusions

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Abstract

A causal illusion occurs when people perceive a causal relationship between two events that are not contingent on each other. This experiment explored how this illusion varies when people reason diagnostically (i.e., in an effect-to-cause direction). Participants learnt about an illusory cause-effect relationship in which the probability of the cause and the probability of the effect were orthogonally manipulated to be either high or low. Participants learnt either predictively (i.e., cause-to-effect) or diagnostically, and at test had to make two causal judgements that encouraged either predictive (cause-to-effect) or diagnostic (effect-to-cause) reasoning. Diagnostic reasoning at test increased the strength of the cause density bias and decreased the strength of the effect density bias. It also decreased causal ratings, but only after predictive learning. Explaining these results requires an understanding of how the process of causal learning can impact later reasoning; something the current literature is yet to provide.

Keywords: diagnostic reasoning; causal reasoning; causal illusion; cause density; effect density

Introduction

The contingency between two events is crucial for assessing causality. For instance, if a drug makes patient recovery more likely than when no treatment is administered, we have grounds to believe the drug causes recovery. Learners are usually good at detecting contingencies for causal judgements (Allan & Jenkins, 1983; Dickinson et al., 1984; Wasserman et al., 1993), and peoples' opinions about common health beliefs correlate with their subjective perceptions about cause-effect contingencies (Chow et al., 2021). However, people often report causal relationships when there is no contingency between a putative cause and its presumed effect (Alloy & Abramson, 1979; Jenkins & Ward, 1965); these are called causal illusions (Matute et al., 2015). Causal illusions are linked to potentially harmful beliefs in pseudoscience and pseudomedicine (MacFarlane et al., 2020; Torres et al., 2020); thus, it is important to understand the different situations in which they emerge.

Although causes must precede their effects, sometimes our reasoning about causal relationships occurs in the opposite direction; we first consider an outcome and then speculate what the responsible causal entity was. That is, rather than reasoning predictively – in a cause-to-effect direction – we reason diagnostically. For example, we may ponder the reason behind our spouse's bad mood, or we may feel sick after a meal and consider which of the ingredients is most likely at fault. Although there is evidence that diagnostic reasoning changes our perception of causal relationships (Fernbach et al., 2011; Meder et al., 2014; Tversky & Kahneman, 1982), how it affects causal illusions has not been extensively explored.

The goal of this study was to determine how diagnostic reasoning affects causal illusions. We first detail how causal illusions are assessed, and what causes them. We then explore how causal illusions may be affected by diagnostic reasoning, before introducing the current experiment.

The Assessment and Causes of Causal Illusions

In contingency learning experiments, participants observe a series of learning trials in which a potential cause and effect are either present or absent (although see Chow et al., 2019, for a causal illusion demonstration with continuous outcomes). This gives rise to four different trial types, depicted in table 1.

Table 1: Contingency Table as a Function of Whether the Cause and Effect are Present or Absent

	Effect present	Effect absent
Cause present	a	b
Cause absent	c	d

The frequencies of each trial type can be used to estimate contingency via the ΔP metric (Allan, 1980; see Cheng, 1997; Griffiths & Tenenbaum, 2005 for alternative normative standards). ΔP indexes the change in probability of the effect when the cause is present, relative to when the cause is absent:

Equation 1.

$$\Delta P = P(E|C) - P(E|\sim C) = [a/(a+b)] - [c/(c+d)]$$

Here, *a*, *b*, *c* and *d* refer to the frequencies of the four trial types in table 1. A positive contingency ($\Delta P > 0$) indicates that the cause increases the probability of the effect. A negative ΔP indicates the cause makes the effect less likely. $\Delta P = 0$ indicates no relation between the two events. Thus, a causal illusion occurs when someone makes a non-zero causal judgement (usually positive) even though $\Delta P = 0$.

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Participants' causal judgements are prone to deviating from ΔP under certain conditions. Causal judgements tend be higher when the effect is present on most trials (i.e., *a* and *c* trials; Allan & Jenkins, 1983; Alloy & Abramson, 1979). Likewise, causal judgements tend to be higher when the cause is present on most trials (i.e., *a* and *b* trials Matute et al., 2011; Torres et al., 2020). Termed the effect density bias and the cause density bias, respectively (Perales et al., 2017), these effects suggest that *a* trials are important for driving causal illusions. When two independent events occur frequently, their inevitable co-occurrence leads to causal illusions (Matute et al., 2015).

Blanco et al. (2013) compared the cause and effect density biases. In experiment 1, while holding ΔP = 0, they orthogonally manipulated the probability of the cause and the probability of the effect to be either high or low (see table 2). These factors interacted; when the density of one was already high, there was a heightened effect of increasing the other.

Table 2: Experimental design and trial type frequencies used in Blanco et al. (2013, Experiment 1)

Group	Cause Density	Effect Density	Trial Type Frequencies
HighC-HighE	High (.80)	High (.80)	64 *a*, 16 *b*, 16 *c*, 4 *d*
HighC-LowE	High (.80)	Low (.20)	16 *a*, 64 *b*, 4 *c*, 16 *d*
LowC-HighE	Low (.20)	High (.80)	16 *a*, 4 *b*, 64 *c*, 16 *d*
LowC-LowE	Low (.20)	Low (.20)	4 *a*, 16 *b*, 16 *c*, 64 *d*

Note. Group names refer to cause density (C) and effect density (E), whose values are shown in the second and third columns, respectively.

Blanco et al. found that the probability of the effect was a stronger determinant of causal illusions; comparing HighC-LowE and LowC-HighE groups, the LowC-HighE group gave higher causal judgements. These groups had the same number of *a* trials (see table 2), but the LowC-HighE group had more *c* trials, whereas the HighC-LowE group had more *b* trials. These findings indicate *b* trials influence judgements more heavily than *c* trials, suggesting people ascribe relatively little weight to trials where the cause is absent. The findings concur with broader literature suggesting that judgements follow the inequality $a > b > c > d$ (Perales et al., 2017; Perales & Shanks, 2007; Wasserman et al., 1993).

Theoretical Accounts of Causal Illusions and Implications for Diagnostic Reasoning

Theoretical accounts of causal illusions can be divided into associative accounts of causal learning, and statistical/inferential accounts of causal reasoning (Pineño & Miller, 2007; Vadillo & Matute, 2007).

Associative accounts explain casual learning via the automatic development of an associative link between mental representations of the cause and effect, which strengthens each time the organism experiences a co-occurrence of the two events (Chapman & Robbins, 1990; Wasserman et al., 1993). These accounts generally explain causal illusions as the result of a transient association that develops due to cause-effect co-incidences, and which disappears given enough experience.

Statistical and inferential reasoning accounts explain causal reasoning in terms of statistical rules which describe the mental processes people use to form causal judgements (Perales et al., 2017). These accounts explain causal illusions either as the result of a rational or quasi-rational reasoning process when viewed within an alternative normative framework to ΔP (Cheng, 1997; Griffiths & Tenenbaum, 2005; Hattori & Oaksford, 2007), or due to heuristics used to form causal judgements that lead to the $a > b > c > d$ cell weighting bias (Mandel & Vartanian, 2009; White, 2003).

Diagnostic reasoning presents a significant challenge to both approaches because they make no explicit attempt to explain why diagnostic causal judgements should differ from predictive causal judgements. Associative accounts are largely concerned with how covariation information concerning two events is acquired by the organism via associative link formation; less attention is paid to how these associations may be transformed into causal judgements (Shanks, 2007; although see Vadillo & Barberia, 2018; also see Vadillo & Matute, 2007), whether these judgements are diagnostic or otherwise. By appealing to higher-order reasoning processes used to form causal judgements, statistical and inferential reasoning accounts appear better poised to explain how diagnostic reasoning may affect causal illusions; yet there seems to be no account that explicitly addresses how diagnostic reasoning may alter the statistical rules used to form causal judgements.

Nonetheless, literature comparing predictive inferences (i.e., estimating the likelihood an outcome has occurred, given a potential cause has occurred) with diagnostic inferences (i.e., estimating the likelihood a that a specific cause is present, given its effect has occurred) suggests that diagnostic reasoning may decrease causal illusions. Diagnostic inferences are thought to be less intuitive because mental models of causation run forward in time (Tversky & Kahneman, 1982). This disfluency could encourage people to think more analytically and deliberatively, such that they produce less biased judgements (Alter et al., 2007; Evans, 2008; Sloman, 1996; Tversky & Kahneman, 1982). Additionally, diagnostic inferences are more sensitive to alternative causes than predictive inferences, such that the former often produce more normative causal inferences (Fernbach et al., 2010, 2011).

Consequently, diagnostic reasoning could decrease causal illusions by encouraging participants to reason more deliberatively and attend to alternative causes (i.e., cause-absent trials). However, empirical evidence for this is unclear. Moreno-Fernández and Matute (2020) did not find

any differences between a predictive and diagnostic causal judgement question when assessing causal illusions. Interestingly, White (2003) found that a diagnostic causal judgement (which he refers to as a 'passive-wording' causal question) decreased the weighting of *b* trials, and increased the weighting of *d* trials, such that the cell weighting was $a > b \approx c \approx d$. The equal weighting of *b* and *c* trials suggests that diagnostic reasoning may result in comparable cause density and effect density biases. Whether this would also decrease causal illusions, however, is unclear; although there were no differences in causal ratings on average across the questions, they were tested across a range of contingencies rather than focusing on $\Delta P = 0$ specifically.

Overall, previous research on causal inferences suggests that diagnostic reasoning may reduce causal illusions. However, the little research that has examined how diagnostic reasoning affects causal illusions and contingency judgements more broadly does not support this. Instead, it suggests that the weighting of the cause density and effect density bias may be modulated by diagnostic reasoning. Both issues are explored in the current study.

Aims and Hypotheses

The aim of the current study was to determine how diagnostic reasoning affects causal illusions in contingency learning. Participants determined if a fictitious drug caused patient recovery from an unspecified disease. We orthogonally manipulated the cause density and the effect density to either be high (.75) or low (.25) while holding $\Delta P = 0$ across all groups (see table 3).

Table 3: Experimental design and trial type frequencies in the current study

Group	Cause Density	Effect Density	Trial Type Frequencies
HighC-HighE	High (.75)	High (.80)	27 *a*, 9 *b*, 9 *c*, 3 *d*
HighC-LowE	High (.75)	Low (.20)	9 *a*, 27 *b*, 3 *c*, 9 *d*
LowC-HighE	Low (.25)	High (.80)	9 *a*, 3 *b*, 27 *c*, 9 *d*
LowC-LowE	Low (.25)	Low (.20)	3 *a*, 9 *b*, 9 *c*, 27 *d*

Note. Group names refer to cause density (C) and effect density (E), whose values are shown in the second and third columns, respectively. Learning direction is omitted as a grouping factor as it did not change the trial type frequencies.

At test, participants answered a standard causal judgement question used in many causal illusion experiments (Barberia et al., 2021; Matute et al., 2015). Specifically, participants were asked: "On a scale from -100 to 100, to what extent do you think the drug was effective for patient recovery?". Above the text was the stimulus image used during training for drug administration; below the question was a visual analogue scale ranging from -100 (effectively worsens recovery) to +100 (effectively improves recovery). Since the drug was the focus of this question, we will refer to this as the *cause-framed* question. Importantly, participants answered a second question: "on a scale from -100 to 100, to what extent do you think patient recovery was due to the effectiveness of the drug?". Above the question was the stimulus image for patient recovery used during training; below was a visual analogue scale ranging from -100 (recovery much worse with drug) to 100 (recovery much better with drug). This was intended to encourage diagnostic reasoning (i.e., from effect-to-cause) by making recovery the focus of the question, which appeared underneath the cue for recovery; we refer to this as the *effect-framed* question.

We expected lower causal ratings for the effect-framed question. Additionally, White's (2003) findings would suggest that the effect-framed question might decrease the strength of the effect density bias relative to the cause density bias. This could manifest as 1) an interaction of question framing with cause density, such that the cause density bias is more pronounced with the effect-framed question relative to the cause-framed question, and/or 2) an interaction with effect density, such that the effect density bias is less pronounced with the effect-framed question relative to the cause framed question.

Additionally, the order in which participants learnt about the putative cause-effect relationship was varied between-subjects. Half learnt predictively: On each trial, they first observed whether the patient received the drug and then predicted whether the patient recovered. Half learnt diagnostically: They predicted whether a patient had been administered the drug based on recovery information. This was done to determine whether diagnostic learning affected causal judgements; however, the focus of this paper is on the effect of diagnostic reasoning at test. Although we report all analyses for completeness, the effects of learning direction on causal judgements will not be interpreted unless such effects involve an interaction with reasoning direction.

Method

Participants

The sample was 270 (M_{age} = 19.69, *SD* = 3.47) undergraduate psychology students from the University of Sydney. They were randomly allocated to one of eight conditions according to time of arrival (n = 33-35 each group).

Stimuli and Apparatus

The experiment was programmed using the Psychophysics Toolbox extension for MATLAB (Brainard, 1997; Pelli, 1997). On cause-present trials, drug administration was represented as an orange pill bottle with the drug name 'Serizone' underneath; on cause-absent trials this image was greyed out, and 'no Serizone' was written underneath. On effect-present trials, patient recovery was represented as a man standing next to a wheelchair with their arms extended

triumphantly with 'patient recovered' written underneath; on effect-absent trials, a greyed-out image of the same man in a wheelchair was presented with 'patient did not recover' written underneath.

Design

The experiment was a 2 (cause density) x 2 (effect density) x 2 (learning direction) design. Cause density and effect density were orthogonally manipulated to be either high (.75) or low (.25) by varying the number of *a*, *b*, *c* and *d* trials, while holding $\Delta P = 0$ across all groups (see table 3). The two dependent variables were the ratings for the cause-framed and effect-framed questions, which we used to investigate the influence of reasoning direction at test. These formed a within-subjects variable that we will refer to as *framing*.

Procedure

Participants were asked to imagine they were a medical researcher investigating the use of a fictitious drug Serizone for treating a new, unspecified disease caused by a virus. Their job was to determine the effectiveness of Serizone by viewing medical records of patients suffering the disease, some of which were treated with Serizone. They completed 48 learning trials, each representing a medical record from a different patient. On each trial they were either presented with information about Serizone administration and asked to predict whether the patient recovered (predictive learning); or presented with information about patient recovery and asked to predict whether the patient was administered Serizone (diagnostic learning). They then answered the cause-framed and effect-framed questions in randomized order.

After causal judgements, participants estimated the frequency of *a*, *b*, *c* and *d* trials and rated how important each trial type was for causal judgements. These were included to assess the impact of learning direction and were unrelated to our hypotheses about question framing; consequently, they are not discussed further in this proceeding.

Results

Figure 1A and 1B present causal ratings for the predictive and diagnostic learning groups, respectively. From these panels the density biases and their interaction are apparent: causal judgements are highest when both cause density and effect density is high and are much lower when even one of these densities is low. Panels 1C-E present the difference in causal ratings between the cause-frame and effect-framed question as a function of either learning direction (1C), cause density (1D), or effect density (1E). Positive scores in these panels indicate lower causal ratings for the effect-framed question relative to the cause-framed question. From these panels, it appears that the effect-framed question elicits lower causal ratings after predictive learning, when cause density is low, and when effect density is high.

A four-way mixed model ANOVA was run using cause density, effect density, learning direction and framing as factors. There was a significant effect of cause density, $F(1,262) = 26.94$, $p < .001$, $\eta_p^2 = .093$, and a significant effect

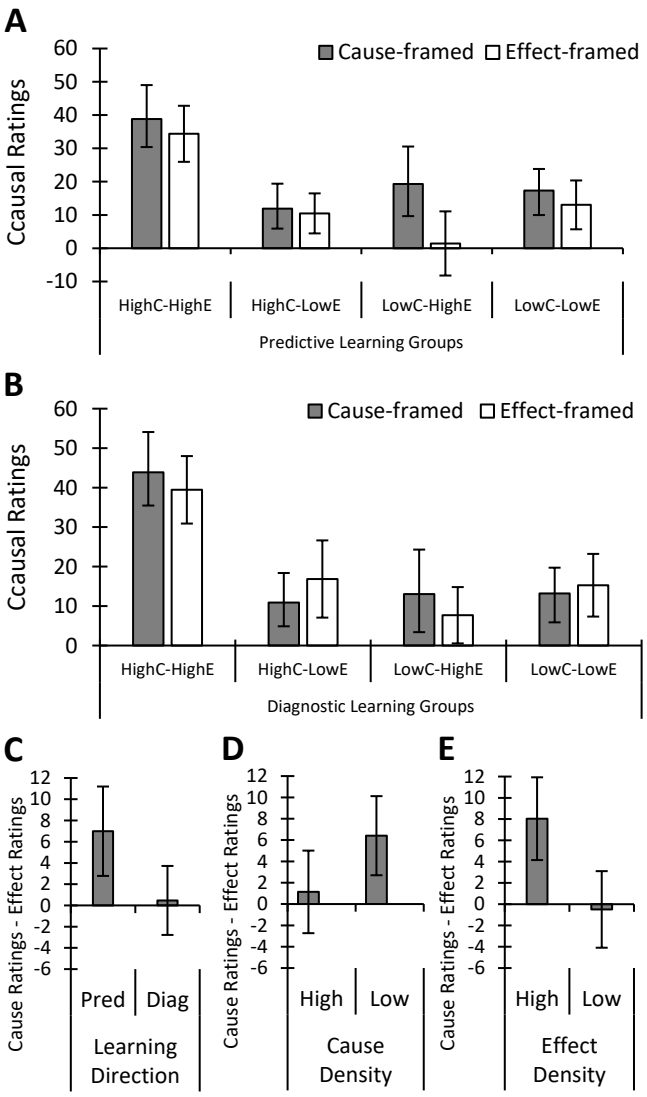

Figure 1: Mean Causal Ratings. Panel A shows ratings for the cause-framed and effect-framed question as a function of each predictive learning group. Panel B shows the same information for the diagnostic learning groups. Group names refer to cause density (C) and effect density (E), and whether it was set to high (.75) or low (.25). The bottom three panels plot the decrease in causal ratings elicited by the effect-framed question as a function of C) learning direction, D) cause density, and E) effect density. Cause ratings and effect ratings refer to ratings for the cause-framed and effect-framed question, respectively. 'Pred' and 'Diag' in panel C refer to predictive and diagnostic learning, respectively.

of effect density, $F(1,262) = 18.87$, $p < .001$, $\eta_p^2 = .067$; in both instances, ratings were higher when the density was high rather than low. These factors interacted, $F(1,262) = 36.49$, $p < .001$, $\eta_p^2 = .122$. Simple effects analysis showed that when cause density was low, mean ratings did not significantly differ with effect density, $F(1,262) = 1.44$, $p = .231$, $\eta_p^2 = .005$; but when cause density was high, mean causal

judgements were higher when effect density was high rather than low, eliciting a simple main effect of effect density $F(1262) = 53.93$, $p < .001$, $\eta_p^2 = .171$.

As expected, the main effect of framing was significant, $F(1,262) = 7.96$, $p = .005$, $\eta_p^2 = .029$, such that average ratings were lower for the effect-framed question than the cause-framed question; however, this was qualified by an interaction with learning direction, $F(1,262) = 6.17$, $p = .014$, $\eta_p^2 = .023$. Simple effects analysis revealed a significant simple main effect of framing for the predictive learning group, $F(1,262) = 14.29$, $p < .001$, $\eta_p^2 = .052$, such that ratings were lower for the effect-framed question relative to the cause-framed question (see figure 1C); but ratings did not significantly differ after diagnostic learning, $F(1,262) = .06$, $p = .814$, $\eta_p^2 = .000$. Framing also significantly interacted with effect density, $F(1,262) = 10.61$, $p = .002$, $\eta_p^2 = .039$, such that the effect density bias was less pronounced with the effect-framed question relative to the cause-framed question; and framing significantly interacted with cause density, $F(1,262) = 3.985$, $p = .047$, $\eta_p^2 = .015$, such that the cause-density bias was more pronounced with the effect-framed question relative to the cause-framed question. Notably, the larger cause density bias appears to be due to a difference in ratings for the two questions in the low cause density groups (see figure 1D); whereas the smaller effect density bias appears to be driven by a difference in ratings for the high effect density groups (see figure 1E).

There were no other significant effects: the main effect of learning direction was not significant, and did not significantly interact with cause or effect density; there were no significant three-way interactions; and the four-way interaction was not significant (largest $F = 1.65$, $p = .201$, $\eta_p^2 = .006$).

Discussion

The aim of this study was to investigate the effect of diagnostic reasoning on causal illusions. As expected, encouraging diagnostic reasoning at test via an effect-framed question led to a decrease in causal ratings; however, this only occurred after predictive learning. Relative to the cause-framed question, the effect-framed question increased the magnitude of the cause density bias and decreased the strength of the effect density bias. We will now explore the implications of each of these findings.

Diagnostic Reasoning and the Density Biases

Although our results indicate that the effect-framed question increased the cause density bias and weakened the effect density bias, Moreno-Fernández and Matute (2020) found no influence of reasoning direction on the density biases. In their experiment, participants could choose which data they sampled based on whether a drug was administered (cause group) or whether an allergic reaction occurred (effect group). As such, participants controlled the frequency of one event; the other was set to 75%. Since participants in both groups chose positive instances of the event (i.e., cause present or effect present) more than 50% of the time on average, participants in both groups were exposed to relatively high densities of both events. Such high densities could have obscured variation in these biases with diagnostic reasoning. Alternatively, when orthogonally manipulating each trial type, White (2003) found the cell weight bias $a > b \approx c \approx d$ for diagnostic causal judgements. The relative equality of b and c trial weighting is reflective of the current study, as it indicates an increase in the weighting of the cause density bias relative to the effect density bias. This suggests that modulation of the density biases can be observed when there is sufficient variation in cause and effect density.

Given the distinct pattern of causal ratings elicited by the effect-framed question, it is an interesting issue as to whether current theory on causal reasoning can account for these findings. As mentioned earlier, associative accounts do not speak directly to our results since they do not focus on how reasoning demands at test affect causal judgements (Pineño & Miller, 2007). Instead, we consider how statistical and inferential reasoning approaches may accommodate our results. We consider two approaches; one which assumes normative causal reasoning, and one which assumes that heuristics are used to generate causal judgements.

From a normative perspective, one possibility is that the effect-framed question requires participants to make a causal attribution judgement (Cheng & Novick, 2005, equation 4). Specifically, the question asks how responsible the cause was for the observed probability of the effect. The prevalence of the cause and effect normatively influence judgements according to the following formula:

Equation 2.

$$\mathrm{P}(\mathrm{C}-\mathrm{alone} \rightarrow \mathrm{E}|\mathrm{E}) = \frac{\mathrm{P}(\mathrm{C}) * \Delta\mathrm{P}}{\mathrm{P}(\mathrm{E})}$$

Where P(C-alone→E|E) is the probability that the effect is due to the focal cause alone, given the effect's presence. If we assume that participants have a biased method for estimating contingency in place of ΔP, then this equation may be compatible with the current study's findings: for a given estimate of contingency, ratings for the effect-framed question should be higher when cause density is high and effect density is low. Nonetheless, one would still need an account as to why the participants failed to accurately estimate ΔP. A variation of this measure uses the causal power metric (Cheng, 1997) instead of ΔP (Cheng and Novick 2005, equation 3). Since a discussion of causal power is beyond the scope of this study, we have used this formula instead.

A heuristic reasoning approach to causal illusions can also accommodate the modulation of the density biases by the effect-framed question. Specifically, the weighted positive test strategy (WPS; Mandel & Vartanian, 2009) model could provide an explanation. It assumes that people have a positive-test bias – that is, a preference to test hypotheses by searching for data consistent with it (Leventhal et al., 1994) – in relation to two tests for causality. The first is that the cause brings about the effect – a hypothesis test – which leads

to weighing *a* and *b* trials and requires predictive reasoning. The second test is that the effect is due to the cause – a target test – which leads to weighing *a* and *c* trials and requires diagnostic reasoning. The combination of these tests leads to $a > b = c > d$ trial weighting. Crucially, the $b > c$ inequality is obtained by assuming the hypothesis test is weighted more due to people's preference for predictive reasoning (Tversky & Kahneman, 1982). Thus, this model could be extended to account for our current results by assuming that the weighting of these tests varies with the demands imposed by the wording of causal judgement questions. Specifically, the shift towards a $c > b$ trial weighting for the effect-framed question could be because people perceive the target test as more relevant when answering it. An interesting avenue for future research would be to determine whether the modified WPS model or the causal attribution equation better account for causal ratings elicited by the effect-framed question.

Diagnostic Reasoning and Learning Direction

The effect-framed question was hypothesised to decrease causal ratings on the basis that diagnostic reasoning is considered less intuitive (Fenker et al., 2005; Tversky & Kahneman, 1982) and more sensitive to the presence of alternative causes (Fernbach et al., 2011). It seems unlikely that the current results are due to the effect-framed question encouraging the consideration of alternative causes. This is because the effect-framed question only elicited lower causal ratings after predictive learning. If anything, a diagnostic learning phase would make alternative causes of recovery more salient; yet cause-framed and effect-framed ratings did not significantly differ for this group.

Rather, we believe our results are reflective of the fact that diagnostic reasoning is less intuitive, with the proviso that learning experience may change whether diagnostic causal judgements are considered unintuitive. Specifically, having half the participants learn the putative cause-effect relationship diagnostically may have familiarised them with diagnostic reasoning about the relationship, such that they did not find the effect-framed question unintuitive. This would explain why diagnostic learners did not give decreased ratings for the effect-framed question, while the predictive learners did. As such, these results may reflect a disfluency effect, whereby participants give lower causal ratings when reasoning in an unfamiliar direction.

Although a disfluency effect in causal reasoning is generally consistent with the notion that causal illusions are due to higher-order reasoning processes, there appears to be no statistical or inferential reasoning account that explicitly captures this phenomenon. The WPS model, for example, cannot account for this finding, even if we assume that the weighting of the target and hypothesis test change based on the direction of learning and/or reasoning. Since event densities were counter-balanced across conditions, weighing one test over another should not change mean causal ratings when averaged across cause and effect density; any increase in one condition would be cancelled out by a decrease in the other. Likewise, assuming that participants responded to the effect-framed question by providing a causal attribution judgement also does not explain this interaction. Producing a causal judgement according to equation 2 would yield the same output irrespective of learning direction. More broadly, statistical and inferential reasoning accounts do not address how covariation information is acquired by organisms; rather, these accounts simply specify how such information is transformed into causal judgements (Perales et al., 2017). Consequently, these accounts struggle to explain why the direction of learning would impact the reasoning process that transforms the acquired information into causal judgements.

Considerations for Future Research

The current study manipulated question framing within-subjects. Similar within-subject manipulations have been used to explore the effect of test question phrasing since having participants answer both questions encourages them to interpret the meaning of each question in its intended manner (Matute et al., 1996). Nonetheless, it would be interesting to determine if the effects of question framing persist when manipulated between-subjects.

Disfluency manipulations are thought to debias reasoners by triggering analytic reasoning (Alter et al., 2007; Gervais & Norenzayan, 2012). Nonetheless, it is possible that predictive learners gave lower ratings for the effect-framed questions due to uncertainty associated with the disfluency of the question, rather than due to more normative reasoning. Future research could resolve this issue by including a condition in which there is a contingency between the putative cause and outcome. Contingency overestimations are most pronounced at null contingency (Blanco et al., 2015); otherwise, causal judgements tend to approximate ΔP reasonably well (Dickinson et al., 1984; Wasserman et al., 1993). Therefore, if the decrease in causal ratings is due to a debiasing effect, then this decrease should be less pronounced at positive contingencies since causal ratings for the cause-framed question should already approximate the actual contingency. Indeed, this manipulation has been used to explore other debiasing measures for causal illusions (Díaz-Lago & Matute, 2019a, 2019b).

Conclusion

Causal illusions have been linked to potentially harmful beliefs, such as belief in pseudoscience and pseudomedicine (MacFarlane et al., 2020; Torres et al., 2020). Consequently, this study fills an important gap by investigating how the illusion varies when people are required to reason diagnostically. Our results reveal that the impact of diagnostic reasoning on causal judgements varies depending on how frequently the cause and effect occur, and the temporal order in which the putative cause-effect relationship was learnt. Although these effects are broadly consistent with statistical and inferential reasoning approaches to causal judgements, explaining the full pattern of results requires an understanding of how the process of causal learning can later impact reasoning processes; something which the current literature is yet to provide.

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