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"Causal Reasoning" in Rats: A Reappraisal

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It has recently been argued that rats engage in causal reasoning and they do so in a way that is consistent with Bayes net theories (Blaisdell, Sawa, Leising & Waldmann, 2006). This argument was based upon the finding that the tendency of cues to elicit approach to a food-well was reduced when their presentation was contingent on lever pressing. There is, however, an alternative interpretation of the critical experimental findings that is based on the simple principle of response competition: wherein lever pressing interferes with the tendency to approach the food well. Here the authors replicated Experiments 1 and 2a of Blaisdell et al. (2006) and found reciprocal patterns of lever pressing and food well approach during the critical cues. These results lend direct support for an interpretation in terms of response competition while providing evidence contrary to Bayes net theories, and are readily interpreted within the theoretical framework provided by traditional associative learning theory.

Keywords: Bayes casual nets, response competition, Lloyd Morgan

The enduring debate within the field of comparative psychology concerns the degree to which the behavior of human and nonhuman animals is underpinned by the same cognitive mechanisms. Recent interest has focused on whether seemingly complex human behaviors can be understood in terms of simple (e.g., associative) processes that are ubiquitous in the animal kingdom, and whether nonhuman animals exhibit evidence of mechanisms considered to be uniquely human (e.g., causal reasoning). Recently, Blaisdell, Sawa, Leising, and Waldmann (2006a) have claimed that rats engage in causal reasoning—reasoning that is underpinned by the use of causal Bayes net models (see also Blaisdell, Sawa, Leising, & Waldmann, 2006b, 2007; Leising, Wong, Waldmann, & Blaisdell, 2008). Evidence that is consistent with the suggestion that causal reasoning in humans is underpinned by causal Bayes nets comes from some simple observations that are analogous to those upon which Blaisdell et al. (2006a) made their claim.

In humans who are aware that a single cause (e.g., raindrops) can have two separate effects (water droplets on window panes and dry clothes becoming damp on a washing line), the observation of one effect (wet windows) can lead to the inference that the other effect (damp clothes) has also occurred (this example is taken from Clayton & Dickinson, 2006). This inference presumably reflects the assumption that the observed effect (wet windows) must be the result of its usual cause (rain), which should also result in its second effect (damp clothes). However, this type of reasoning does not hold if there is an alternative cause (e.g., turning on a water sprinkler near to the window, but far away from the washing line) for the observed, first effect. The intervention, of turning on the sprinkler, provides an alternative cause for the first effect and the presence of the second effect is no longer inferred; and the response (e.g., removing the clothes from the line) is not required. The different patterns of inferences that obtain when an effect is simply observed rather than being produced by an alternative cause is predicted by Bayes causal model theory (for an alternative causal reasoning explanation of the same data, see Waldmann, Cheng, Hagmayer, & Blaisdell, 2008). It is this sensitivity to the difference between simply observing an effect and that effect having an alternative cause (the intervention) which Blaisdell et al. (2006a) have recently studied in rats. They submit that this sensitivity is inconsistent with simple associative accounts of animal behavior and consistent with an analysis in terms of Bayes net theories.

Blaisdell et al. (2006a) first gave rats training that should produce an analogous situation for rats to the "rainy day" scenario described above (see Table 1). In their study, rats received training in which a visual stimulus (A: analogous to raindrops) served as the "common cause" for two separate effects: an auditory stimulus, B (wet windows), and the delivery of sucrose into a food well (damp clothes). This common cause treatment was arranged by giving rats separate A→B and A→sucrose trials. During subsequent testing, the tendency of rats to approach the food well (indexed by "nosepoking" in the food well; analogous to removing clothes from the line) was examined as a function of whether the presentation of B was contingent upon rats pressing a lever (intervene condition) or simply occurred without apparent cause (observe condition). The rats were less likely to nosepoke if their lever presses resulted in the presentation of B than if B arrived unannounced. On further control trials, the rats received another auditory stimulus C (the "direct-cause") that had been presented simultaneously with sucrose (i.e., C+sucrose trials) during the first stage of training. There was no influence of the intervene/observe manipulation on nosepoking during C.

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Table 1
Design of Experiments 1 and 2

Training	Test condition
Experiment 1: Within-subjects design	
Common-cause training:	
A→B	Intervene Common-cause:
A→Sucrose	**Lever Press→B**
C+ Sucrose	Observe Common-cause: **B**
	Intervene Direct-cause:
	Lever Press→C
	Observe Direct-cause: **C**
Experiment 2: Between-subjects design	
Common-cause groups:	
A→B, A→Sucrose & C	Intervene Common-cause:
	Lever Press→B
	or Observe Common-cause: **B**
Chain groups:	
B→A, A→Sucrose & C	Intervene Chain:
	Lever Press→B
	or Observe Chain: **B**

On the basis of causal Bayes net theory, Blaisdell et al. (2006a) argued that whether or not rats approached the food well in the common-cause condition (i.e., during stimulus B) should be influenced by whether or not their action generated B: If B was simply presented then the rats should infer that stimulus A had occurred and its second effect (i.e., sucrose) would be delivered to the food well; and they should approach the food well. However, if B was generated by their action (i.e., lever pressing) an alternative explanation for B's occurrence is readily available and they should not approach the food well. In contrast, Blaisdell et al. (2006a) argued that, according to causal Bayes net theory, when the test stimulus was presented that was a direct cause of sucrose (i.e., C), rats should approach the food well irrespective of whether or not the presentation of C was contingent upon their action. The results, as described earlier, were consistent with these predictions: while the intervention treatment had no effect on nosepoke responding to the direct cause, C, nosepoking during B was lower in the intervene condition than the observe condition. A second experiment in Blaisdell et al. (2006a) demonstrated that the fact that the intervene/observe manipulation did not influence responding to C was not simply a product of it being paired simultaneously with sucrose during training.

The results described by Blaisdell et al. (2006a) are undoubtedly thought provoking: the possibility that rats are able to control their behavior on the basis of a representation of causal relationships in their world, combined with a sensitivity to the outcomes of their own actions, is potentially very important. It is important because it challenges the prevailing associative interpretation of animal behavior (for a review, see Dickinson, 1980; Rescorla, 1991). If the results reported by Blaisdell et al. (2006a) prove to be reliable under a range of experimental conditions, then it would certainly suggest that a wholesale change in our appreciation of animal learning is required (see Clayton & Dickinson, 2006). In the original experiments, lever presses *during* the critical test stimuli were not recorded (A. P. Blaisdell, personal communication, March 20, 2006); such lever presses might contribute (through a process of response competition) to the modulation of nosepoking that was associated with intervention. Here, we report replications of two experiments reported by Blaisdell et al. (2006a), but we also recorded lever press responses during the test stimuli.

Experiment 1

Experiment 1 is a systematic replication of Blaisdell et al. (Experiment 1, 2006a) in which, as far as possible, their procedures were followed. However, we replaced one of their stimuli (the white noise) with a buzzer, because white noise occasionally elicits convulsive fitting in our rats. In addition, we employed a conventional within-subjects design, which subsumed the design used by Blaisdell et al. (2006a). As noted earlier, we also recorded lever presses during the critical test stimuli, to assess the possibility that the patterns of lever presses during these stimuli differ between the various conditions. For example, if there was greater evidence of lever pressing during the stimuli in one or more of the intervene conditions, then this might provide a simple explanation for there being a lower level of nosepoking.

Method

Subjects and apparatus. Thirty-two experimentally naïve male Lister hooded rats (*Rattus norvegicus*) obtained from OLAC, Bicester, United Kingdom, were maintained at 85% to 90% of their free feeding weights (range = 312–388*g*) by giving them a restricted quantity of food at the end of each day. All were housed in pairs under a 12 hr/12 hr light/dark cycle. Experiment 1 used four standard operant chambers (L × W×H: 30 × 25 × 20 cm), each housed in a separate sound- and light-attenuating box (supplied by Med Associates Inc., St Albans, Vermont). The sidewalls and ceiling of the chambers were clear Perspex, and the front and rear walls were aluminum. The floor was stainless-steel rods each 0.5 cm in diameter, spaced 1.5 cm center-to- center. The enclosure was dimly illuminated by a 28-V, 100-mA shielded incandescent house light mounted on the top of the rear wall of the conditioning chamber, 2 cm below the ceiling. All experimental procedures were conducted with the house light on, except where noted. A diffuse light was located 5 cm above the ceiling of the chamber. A flashing light (.25 s on /.25 s off) could be presented by turning off the house light and flashing the diffuse light. A speaker on the rear wall of the chamber could deliver a high-frequency tone (3000 Hz) and a buzzer, each at 80 dB. A separate mechanical clicker could deliver a click train at 80 dB. Each chamber was equipped with a dipper that could deliver sucrose solution (20%). When raised, a small well (0.05 cc) at the end of the dipper arm was accessible from the food well. An infrared photo-detector was positioned across the entrance to the food well. This detector was interrogated at intervals of 0.1s and each time this interrogation revealed that the photo-detector was interrupted it was recorded as a nosepoke. Although this recording method produces a measure that conflates number of entries into the food well with entry durations, we have retained this measure and the terminology (nosepoking) used by Blaisdell et al. (2006a) for consistency. A retractable lever was located 4 cm to the left of the food well, 6 cm above the chamber floor, and could be extended 2 cm into the chamber. Ventilation fans in each enclosure provided a low level of constant background noise.

Procedure

Magazine training. The levers were retracted during all phases of the experiment except during testing. On Day 1, rats were trained to approach the food well in a single 60-min session during which a dipper filled with sucrose solution was raised into the food well with an intertrial interval (ITI) of between 5 and 35 seconds. Sucrose was available for 10 s on each trial.

Stimulus training. On Days 2–7, rats received four of each of the following trial types that were pseudorandomly presented within each session, with a variable ITI (range = 2–8 min): Stimulus A (light or click; counterbalanced within group; cf. Blaisdell et al., 2006a) immediately followed by Stimulus B (the stimulus in this common-cause condition was a tone or buzzer, counterbalanced within group; A→B trials); A immediately followed by sucrose (A→sucrose trials); and Stimulus C (the direct-cause stimulus which was a buzzer or tone, counterbalanced within group) presented simultaneously with sucrose (C+ sucrose trials). The durations of the three stimuli (A, B, and C) and sucrose were 10 s.

Testing. Levers were extended into the chambers on Days 8–11 and all rats were tested in each of four conditions across four consecutive days of training: Intervene common-cause, observe common-cause, intervene direct-cause, and observe direct-cause. In the intervene conditions, each lever press caused the presentation of the relevant stimulus (B or C) for 10 seconds (with the exception that lever presses during stimulus presentations had no programmed effect). Each rat in an observe condition was yoked to a rat in the corresponding Intervene condition, so that it received stimulus B or C at the same time as its partner rat. Lever presses had no programmed consequences in the observe conditions. Test sessions were 60 min and no sucrose was presented (see Blaisdell et al., 2007). Lever presses and nosepokes were recorded separately as a function of whether they occurred during stimulus presentations or not. The order in which the conditions were presented across the four test days was counterbalanced in the following manner: Eight rats received the order: Intervene common-cause, observe direct-cause, observe common-cause, and intervene direct-cause. Each rat in these groups was yoked to a rat that was tested in the following order: Observe common-cause, intervene direct-cause, intervene common-cause, and observe direct-cause. A further group of eight rats was tested in the sequence: Intervene direct-cause, observe common-cause, observe direct-cause, and intervene common-cause. Each rat in this group was yoked to a rat that received the sequence: Observe direct-cause, intervene common-cause, intervene direct-cause, and observe common-cause. This order of testing meant that, in addition to providing a full within-subject test of the effects of intervene versus observe and common- versus direct-cause, the first two test days were an exact replication of the design used by Blaisdell et al. (2006a). We report the data from the full within-subjects design and continue by presenting an analysis of the first two days of testing which constitute a direct replication of the procedure used by Blaisdell et al. (2006a).

Rats that did not perform nosepoke responses during either of the common- or direct-cause stimuli during the last session of acquisition were excluded from analysis. Data from five rats were excluded by this criterion. One rat failed to press the lever at any point during the intervene common-cause test so the data for this rat (and its yoked partner) were also excluded as they did not receive exposure to the stimulus in one test condition. Blaisdell et al. (2006a) excluded rats for similar reasons.

Results and Discussion

Training and baseline data: Full within-subjects design. The data from the final training session are shown in the top row of Table 2. Nosepoke responses for stimulus A, as well as in the common-cause and direct-cause conditions, were all above background, prestimulus (Pre-S), levels during training. Analysis of variance (ANOVA) confirmed that there was a main effect of training condition, Pre-S, A, common-cause, direct-cause: $F(3, 72) = 21.96$, $p < .001$, and simple main effect analyses revealed that there were more nosepokes during each of the stimuli than during the prestimulus periods, smallest $F(1, 24) = 35.77$, $p < .001$. Table 2 also shows, for the test sessions, the total number of stimuli presented, and number of lever presses and nosepokes when the stimuli were not present (i.e., the baseline levels of lever pressing and nosepoking). Although the presentation of stimuli in the observe conditions was yoked to the presentation of stimuli in the intervene conditions, because there is variability in the number of stimuli presented across rats, and some rats are excluded from the analysis (see above) the mean numbers of stimuli presented in the different conditions were not identical. Inspection of Table 2 indicates that the number of stimuli produced in the direct-cause conditions was greater than in the common-cause condition and ANOVA confirmed that there was an effect of training condition, common- vs. direct-cause: $F(1, 23) = 4.61$, $p = .042$, but no effect of test condition (intervene vs. observe) and no interaction between these factors (Fs < 1). Inspection of Table 2 indicates that background lever press rates (which included the lever presses that produced the stimuli in the intervene conditions) were higher in the direct-cause conditions than in the common-cause conditions, and higher in the observe that intervene conditions. ANOVA confirmed that there was an effect of training condition, $F(1, 23) = 5.43$, $p = .028$, and an effect of test condition, $F(1, 23) = 7.39$, $p = .012$, but no interaction between these factors ($F < 1$). The fact that the number of stimuli presented and background rates of lever presses were higher in the direct-cause conditions presumably reflects the fact that stimulus C had greater conditioned reinforcement value (because C unlike B had been directly paired with sucrose). This effect of test condition might have been influenced by the fact that rats in the intervene conditions could only produce one lever press outside the stimulus per stimulus presented (these lever presses turned on the stimulus) while rats in the observe conditions could make multiple lever presses outside the stimulus (these were without scheduled consequence). Finally, ANOVA conducted on the baseline nosepoke responses confirmed that there was no effect of training or test condition, and no interaction between these factors (all Fs < 1).

Test data: Full within-subjects design. The critical test data from Experiment 1 are nosepokes (panel A of Figure 1) and lever pressing (panel B of Figure 1) in the common- and direct-cause conditions. Inspection of this figure reveals that there was a reciprocal relationship between nosepoking and lever pressing: There was less nosepoking during B and C in the intervene than the observe conditions, and there was more lever pressing in the intervene than the observe conditions. ANOVA confirmed that

Table 2
Baseline Results From Experiments 1 and 2

Experiment 1				
Training	Pre-S	A	B	C
	6.39 (0.86)	19.10 (2.33)	17.06 (2.04)	21.88 (1.66)
Full within-subjects test	Number of Ss	LPs outside S	Baseline NPs	
Common-cause intervene	11.36 (1.70)	11.36 (1.70)	3.10 (0.75)	
Common-cause observe	10.60 (1.64)	13.76 (1.71)	2.82 (0.42)	
Direct-cause intervene	14.96 (1.99)	14.96 (1.99)	2.48 (0.29)	
Direct-cause observe	12.60 (1.95)	17.16 (1.77)	2.90 (0.37)	
Test days 1 and 2 only	Number of Ss	LPs outside S	Baseline NPs	
Common-cause intervene	15.09 (1.89)	15.09 (1.89)	4.99 (1.65)	
Common-cause observe	12.36 (2.47)	14.43 (2.32)	3.50 (0.62)	
Direct-cause intervene	16.86 (2.30)	16.86 (2.30)	3.32 (0.66)	
Direct-cause observe	15.18 (2.09)	17.81 (3.21)	3.51 (0.55)	

Experiment 2			
Training	Pre-S	A	
Common-cause intervene	18.86 (2.41)	26.53 (4.23)	
Common-cause observe	20.70 (3.97)	35.78 (4.97)	
Chain intervene	23.17 (2.84)	30.25 (5.50)	
Chain observe	19.41 (5.01)	31.75 (6.51)	
Test	Number of Ss	LPs outside S	Baseline NPs
Common-cause intervene	13.67 (1.56)	13.67 (1.56)	3.33 (0.50)
Common-cause observe	14.36 (1.54)	38.45 (5.02)	3.62 (0.45)
Chain intervene	10.42 (1.28)	10.42 (1.28)	4.32 (0.47)
Chain observe	10.42 (1.28)	36.25 (4.91)	4.83 (0.79)

Note. Training data are nosepokes (NPs; maximum = 100) during the stimuli (A, B = common-cause, C = direct-cause) and the prestimulus periods (Pre-S). Test data are total number of stimuli (Ss) presented, total number of lever presses (LPs) outside stimulus presentations (and the 20s poststimulus period in Experiment 2) and nosepokes (NPs) outside the stimulus (and the 20s poststimulus period in Experiment 2) adjusted to the same scale as stimulus NPs (maximum = 100). Note that for Experiment 1, this data is reported twice, once for the full within-subject design and below that for the data from test days 1 and 2 only, that replicates the confounded factorial 2 × 2 design used by Blaisdell et al. (2006a).

there was more nosepoking in the observe than intervene conditions, $F(1, 23) = 5.34$, $p = .030$ and in the direct- than common-cause conditions, $F(1, 23) = 15.07$, $p = .001$; but critically there was no interaction between these factors, $F(1, 23) = 1.58$, $p = .222$. In addition, nosepoke responding was higher during each of the individual test stimuli than it was during background, smallest $t(24) = 3.06$, $p = .005$. The finding that there was more nosepoking in the direct-cause conditions than the common-cause conditions is consistent with the fact that stimulus C was directly paired with sucrose whereas stimulus B was not. Finally, ANOVA confirmed that rats made more lever presses during the stimuli in the intervene than observe conditions, $F(1, 23) = 53.45$, $p < .001$, there was no difference between common- and direct-cause conditions, $F(1, 23) = 1.77$, $p = .197$, and no interaction between these factors ($F < 1$).

In addition to the group-level data, the individual relationship between nosepoke and lever press responding is illustrated in Figure 2 (A). An account of the group-level data in terms of response competition would be difficult to reconcile with large numbers of rats showing high levels of both nosepoke and lever-press responses. No such pattern of behavior was seen in any animal. Considered irrespective of test condition there was a significant negative correlation between nosepoke and lever press ($r = -0.204$, $p = .042$) although this correlation was not significant within any individual test condition (smallest $p = .239$). Thus, the analysis of individual responding confirms the existence of a reciprocal relationship between nosepoke and lever press responses as is predicted by a response competition account of the intervene/observe manipulation.

Baseline and test data: Blaisdell et al. (2006a) design. Across Days 1 and 2 of testing, half of the rats were tested in the Intervene Noise and Observe Tone conditions and the remainder in the Intervene Tone and Observe Noise conditions. This is a Randomized Block Confounded Factorial 2 × 2 design (see Kirk, 1995, p. 588) with within-subject factors of test condition (intervene vs. observe) and training condition (common vs. direct). The interaction between these factors is confounded with the between-subjects factor of Group (i.e., test combination, intervene common and observe direct vs. intervene direct and observe common). Therefore a between-subjects error term, rather than a within-subjects error term, must be used to calculate the F ratio for the interaction. The baseline data from the two test days is shown in Table 2. When analyzed using the method described above, there were no significant effects of test condition, training condition and no interaction between these factors on the number of stimuli presented on test (all Fs < 1). Similarly, there were no significant effects of these factors and no interaction between them on lever pressing outside of the critical test stimuli, largest $F(1, 23) = 1.548$, $p = .226$; a difference from the full within-subjects design, or on baseline nosepoking, largest $F(1, 23) = 1.273$, $p = .271$. Panels C and D of Figure 1 show, respectively, the nosepoke and lever press data during the critical stimuli from the two test days. Inspection of these panels reveals a similar pattern of results to that shown in panels A and B of Figure 1: less nosepoking during the

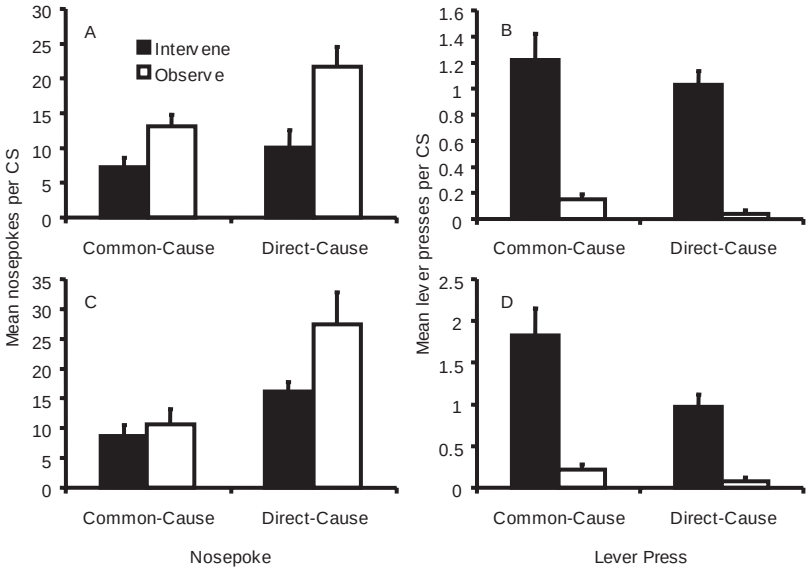

Figure 1. Test results from Experiment 1. Panels A and B show the results from the full within-subjects design; mean (with *SEM*) nosepoke responding (maximum = 100; panel A) and mean (with *SEM*) number of lever presses during common-cause (B) and direct-cause (C) conditioned stimulus (CS) presentations (panel B). Panels C and D show the corresponding data for Test Days 1 and 2 that constitute a direct replication of the design used by Blaisdell et al. (2006a).

critical stimuli in the intervene than observe conditions (panel C) and more lever pressing in the intervene than in the observe conditions (panel D). Analysis of nosepoking during the critical stimuli, confirmed that there were significant effects of test condition, $F(1, 23) = 5.867$, $p = .024$, that there was less nosepoking in the common-cause than the direct-cause conditions, $F(1, 23) = 19.829$, $p < .001$, but that there was no interaction between these factors, $F(1, 23) = 1.927$, $p = .178$. In addition, nosepoke responding was higher in the observe common-cause, intervene direct-cause, and observe direct-cause conditions than it was during background, smallest $t(13) = 3.33$, $p = .005$, but responding during the intervene common-cause condition was not higher than background, $t(10) = 2.03$, $p = .070$. Analysis of lever pressing during the critical stimuli confirmed that there were significant effects of test condition, $F(1, 23) = 50.781$, $p < .001$, training condition, $F(1, 23) = 8.101$, $p = .009$, and an interaction between theses factors, $F(1, 23) = 4.763$, $p = .040$. Simple main effects analyses, however, revealed that the difference between intervene and observe conditions was significant for both common-cause and direct-cause conditions, smallest $F(1, 23) = 28.385$, $p < .001$. As with the full within-subject analysis, the relationship between nosepoke and lever press responding is illustrated in Figure 2 (B). Considered irrespective of test there was a, one-tailed, significant negative correlation between nosepoke and lever press ($r = -0.264$, $p =$

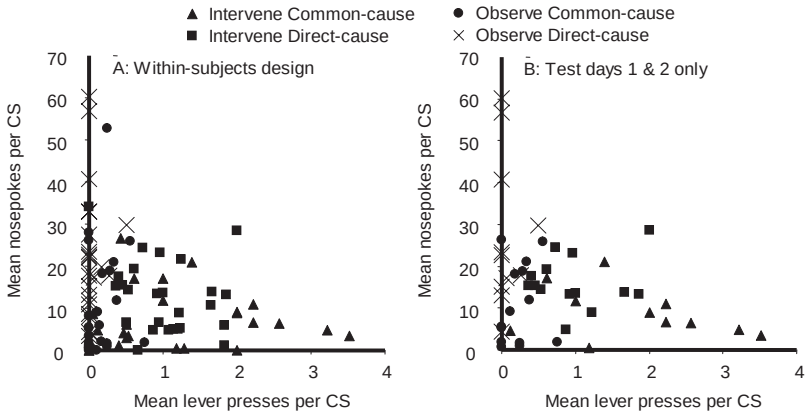

Figure 2. Test results from Experiment 1 showing the relationship between nosepoke and lever press responding for all test conditions. Panel A shows the results from the full within-subjects design while Panel B shows the corresponding data for Test Days 1 and 2 that constitute a direct replication of the design used by Blaisdell et al. (2006a).

.032), although this correlation was not significant within any individual test condition (smallest $p = .312$).

Thus the analysis of the test results from the confounded factorial 2 × 2 design used by Blaisdell et al. (2006a) confirms the pattern of results observed in the full within-subject design: There was a difference between intervene and observe conditions in the rates of nosepoking during the stimuli that did not interact with training condition; and there was a complementary pattern of differences in lever pressing during the critical stimuli. In fact, the absolute size of the intervene/observe difference in the common cause condition is smaller in this subset of the test data than it was in the full within-subject data set and in the data reported by Blaisdell et al., (2006a). The intervene/observe effect might have been partially obscured as nosepoke responding in the intervene common-cause condition was not significantly above baseline levels. However, there is no doubt that overall we replicated the intervene/observe effect reported by Blaisdell et al. (2006a); but this effect was at least as marked in the direct-cause condition as it was in the common-cause condition.

Experiment 2

The fact that stimulus C provoked more nosepoking than stimulus B, irrespective of test condition, is uncontroversial from an associative perspective: C was directly paired with sucrose and B was not. However, this raises the possibility that there was no interaction between the effects of test condition (intervene vs. observe) and stimulus (B and C; cf. Blaisdell et al., 2006a) because B and C provoked different levels of nosepoking. Blaisdell et al. (2006a) considered this form of explanation for their (albeit different) results, and in their Experiment 2a the effect of intervention was assessed after common-cause training (A→B and A→sucrose) and "chain" training (B→A and A→sucrose, which generates a B→A→sucrose chain; see Table 1). In both types of training, the critical test stimulus (B) was indirectly paired with sucrose and should therefore provoke similar levels of nosepoking. Blaisdell et al. (2006a) argued that causal model theory predicts that the effect of intervention should be restricted to rats given common-cause training and the results that they observed were consistent with this prediction. Experiment 2 was a replication of Experiment 2a from Blaisdell et al. (2006a), but we again directly assessed the possibility of response competition by measuring lever pressing during the test stimulus, B, on a single test day (rather than the two test days used by Blaisdell et al., 2006a).

Method

Subjects and apparatus. Forty-eight experimentally naïve male Lister hooded rats (free-feeding weight range = 361–462*g*) were obtained from the same source, housed in the same fashion, and maintained in the same manner as in Experiment 1. Experiment 2 used eight operant chambers of the same specification as those described previously.

Procedure

Stimulus training. Magazine training was the same as in Experiment 1. On each day of stage 1 (Days 2–5), rats in groups common-cause intervene and common-cause observe received six trials where Stimulus A (click) was followed by Stimulus B (tone or buzzer, counterbalanced within group; i.e., A→B trials) with the offset of A coinciding with the onset of B, and six presentations of stimulus C (buzzer or tone, counterbalanced within group). Rats in groups chain intervene and chain observe were treated in the same fashion except the order in which stimuli A and B were presented was reversed (i.e., they received B→A trials). In all groups, the two trial types were pseudo-randomly presented within each session with an intertrial interval of between 2 and 8 min. On both days of stage 2 (Days 6 and 7), all rats received 12 trials where stimulus A (click) was followed immediately by 10-s access to sucrose. Each of these sessions was 60 min and the mean intertrial interval was 5 min (± 3 min). One rat in group common-cause observe was removed from the experiment because it failed to respond during stimulus A on either Day 6 or 7.

Testing. Levers were extended into the chambers for testing on Day 8. In the intervene groups each lever press resulted in the presentation of stimulus B for 10 seconds (with the exception that lever presses during B and for 20 seconds after its offset had no programmed effect). As in Experiment 1, each rat in an intervene group was yoked to a rat in the corresponding observe group so that it received the presentations of B at the same time as its partner rat. Lever presses had no programmed consequences in the observe groups. Test sessions were 60 min. Lever presses and nosepokes were recorded separately as a function of when they occurred relative to stimulus presentations.

Results and Discussion

Training and baseline data. The data from the final acquisition session are shown in Table 2. ANOVA confirmed the impression gained from Table 2 that there was more nosepoking during stimulus A than during the baseline, prestimulus periods, $F(1, 43) = 33.07$. $p < .001$, and that there was no effect of test condition or training type on responding during the prestimulus or stimulus A, and no interactions between these factors (Fs < 1). Table 2 also shows, for the test sessions, the total number of stimuli presented, total number of lever presses and nosepokes outside the stimulus presentations. Because the number of stimuli that were presented was not independent between intervene and observe conditions, only the numbers of stimuli presented in the intervene conditions were analyzed. This analysis revealed there was no difference between common-cause and chain groups, $t(22) = 1.61$, $p = .122$. Analysis of lever pressing outside the stimulus presentations and the 20-s period following each stimulus revealed that there was no effect of stimulus ($F < 1$), an effect of test condition, $F(1, 43) = 49.80$, $p < .001$, and no interaction between the two factors ($F < 1$). As in the analysis of the full within-subjects design used in Experiment 1, the effect of test condition on lever pressing outside of the stimulus might reflect the fact that rats in the intervene condition could only produce one lever press outside of each stimulus. The levels of baseline nosepokes during the test were similar in the four conditions. ANOVA confirmed that there was no significant effect of either training type, common-cause vs. chain, $F(1, 43) = 3.68$, $p = .061$, or test condition (intervene vs. observe, $F < 1$), and that there was no interaction between these factors ($F < 1$).

Test data. Responding during the test presentations of stimulus B is shown in the upper panels of Figure 3. Inspection of panel

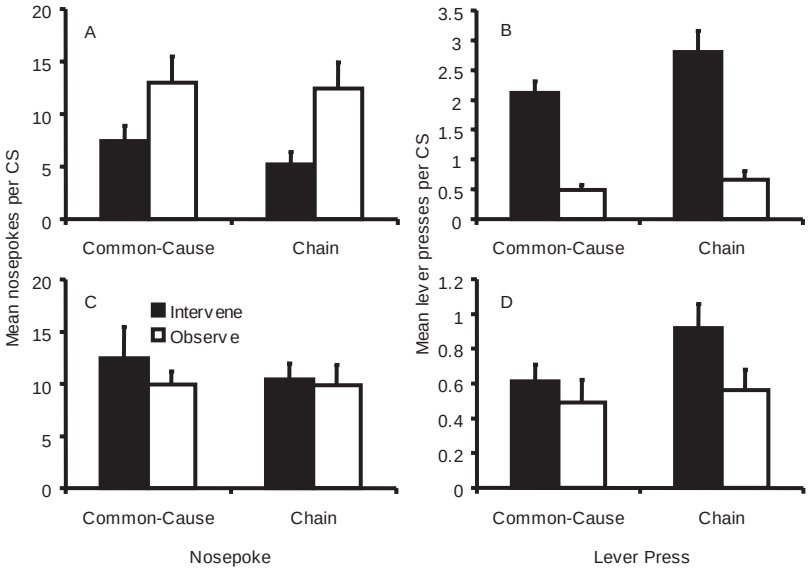

Figure 3. Test results from Experiment 2. Mean (with *SEM*) nosepoke responding (maximum = 100; panel A) and mean (with *SEM*) number of lever presses during the test stimulus for the common-cause and chain groups (panel B). Panels C and D show the corresponding data during the period 10 to 20 s after stimulus offset.

A reveals more nosepoking in the observe than intervene groups, $F(1, 43) = 10.58$, $p = .002$, but no effect of training condition (common-cause vs. chain) and no interaction between these factors (Fs < 1). In addition, nosepoke responding was higher in the observe common-cause, intervene common-cause, and observe chain conditions than it was during background, smallest $t(11) = 2.94$, $p = .014$, but responding during the intervene chain condition was not higher than background ($t < 1$). Rats made more lever presses in the intervene than observe groups, $F(1, 43) = 72.47$, $p < .001$, there was no significant effect of training condition, $F(1, 43) = 3.70$, $p = .061$, and no interaction between these factors, $F(1, 43) = 1.41$, $p = .242$. Like Blaisdell et al. (2006a), we examined responding 10 to 20 s after stimulus offset, when causal model theory would predict that rats in the chain conditions should expect sucrose to occur (see Figure 3C and 3D). During this period, there was no effect on nosepoking of intervention (see Figure 3C) or training type, and no interaction between these factors (all Fs < 1). In this post-CS period, nosepoke responding in each test condition was higher than it was during background, smallest $t(11) = 3.34$, $p = .011$. Parallel analysis of lever pressing (see Figure 2D) revealed that there were no significant effects of either factor and no interaction between them, largest $F(1, 43) = 3.80$, $p = .058$, which was the effect of intervention.

As for Experiment 1, the individual relationship between nosepoke and lever press responding is illustrated in Figure 4: Panel A shows the data for responding during the CS while Panel B shows the data in the period 10 to 20 s after CS offset. As would be expected from the group level data, a negative relationship be-

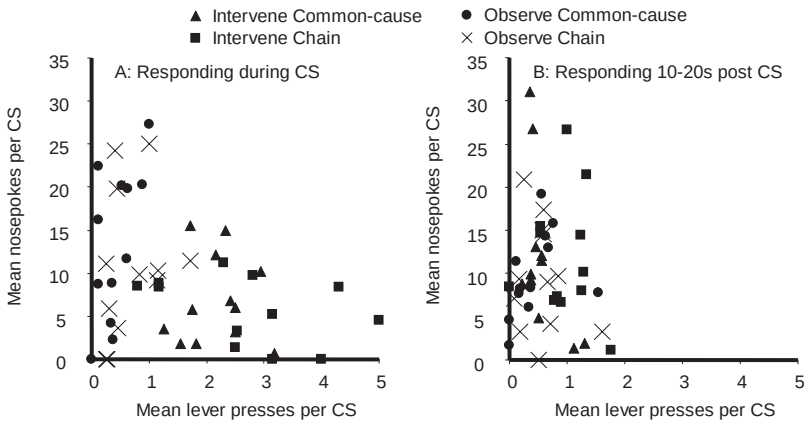

Figure 4. Test results from Experiment 2 showing the relationship between nosepoke and lever press responding for all test conditions. Panel A shows the results during the test stimulus while Panel B shows the corresponding data during the period 10 to 20 s after stimulus offset.

tween nosepoke and lever press responding was apparent during the CS but not in the post-CS period (largely because of the very small number of lever-press responses). Considered irrespective of test condition there was a significant negative correlation between nosepoke and lever press during the CS ($r = -0.364$, $p = .012$). There were no significant negative correlations in any individual test condition but in the Observe common-cause condition the correlation was actually significantly positive ($r = .6.49$, $p = .031$; see General Discussion). There was no significant correlation in the post-CS data ($r = -0.071$, $p = .638$).

To summarize: As in Experiment 1 and Blaisdell et al. (2006a), we observed an effect of the intervention manipulation; and as in Experiment 1 the effect of intervention was also evident in the control condition. Moreover, in Experiment 2, like Experiment 1, there was (overall) a reciprocal relationship between nosepoking and lever pressing during the stimuli. Unlike Blaisdell et al. (2006a), we found no evidence for differential nosepoking (or lever pressing) between conditions in the poststimulus period. Although there was some indication that lever pressing was greater in the intervene conditions during the poststimulus period, this difference was not significant, and as is immediately apparent in Figure 4, the absolute rates of responding in this period were very much lower than during the stimulus.

As in Experiment 1, the fact that the rats' lever press intervention influenced their nosepoking in Experiment 2 is explicable in terms of response competition. However, when consideration is given to the size of the effects of intervention on lever pressing and nosepoking during the critical stimuli, this account might appear implausible: with numerically small differences in lever pressing during the critical stimuli having to produce seemingly marked differences in nosepoking during those stimuli. However, the differences in nosepoke responses between the intervene and observe conditions across experiments are only of the order of 1 s per stimulus which is in fact consistent with a difference of about one lever press per stimulus. Moreover, it should also be remembered that lever presses are the discrete endpoints of approach and contact with the lever, whereas nosepoking is a relatively arbitrary measure that reflects the amount of time the rat spends with its snout in the food well.

General Discussion

Experiments 1 and 2 represent direct replications of the experimental protocols used by Blaisdell et al. (2006a) to investigate casual learning in rats. Like Blaisdell et al. (2006a), we observed that the presentation of stimuli that were contingent on the rats' own actions elicited less nosepoke behavior than did stimuli that were not produced by their actions. Unlike Blaisdell et al. (2006a), in both Experiments 1 and 2 we were not able to replicate the observation that this effect of intervention interacted with the nature of prior training—an interaction predicted by Bayes net theories of causal reasoning. That is, we also found an effect of the manipulation of interest (i.e., intervention) in the control conditions as well as in the common-cause conditions, which implies that there might be some more general account of the effects of intervention than that given detailed consideration by Blaisdell et al. (2006a). There is internal evidence from Experiments 1 and 2 that is consistent with one general interpretation of the effect of intervention. We observed that the number of lever press responses that occurred during a stimulus was higher when its presentation was response contingent than when it was not. That is, there was an inverse relationship between levels of lever pressing and levels of nosepokes, suggesting that the reduction in nosepokes in the intervene conditions was because of competition from lever pressing.

We supplemented the observation that there was inverse pattern of lever pressing and nosepokes at the group level, with further analyses that examined the correlations between these two types of response. At an overall level, and as would be expected on the basis of the group level effects, there was a negative correlation between lever pressing and nosepokes. However, within individual groups there were no such correlations. That is, the group level intervention had a powerful effect on the relative amounts of the two types of responses, but within groups the rats' tendency to lever press was relatively independent of their tendency to nosepoke. Of course, one might expect individual differences in general levels of activity to result in a positive correlation between different responses; and this is precisely what was observed in at least one condition. It is worth repeating the point that the response competition analysis of the effects of intervention would have been undermined by finding many rats that produced high levels of both lever presses and nosepokes. Inspection of Figures 2 and 4 confirms that there were no such rats in Experiments 1 and 2.

Given the fact that Experiments 1 and 2 and those reported by Blaisdell et al. (2006a; see also, Experiment 3 in Leising et al., 2008) employed essentially the same stimulus parameters and equipment, and similar strains of rats (Hooded Lister vs. Long Evans, respectively), what should be made of the fact that they produced different patterns of results? It should be acknowledged that exact replication in another laboratory is virtually impossible. For example, although the complete design of Experiment 1 was not a direct replication of Blaisdell et al. (2006a), it is at least as powerful a design and should have given results that are consistent with an analysis in terms of Bayes causal nets if such an analysis is a good account of rat behavior.

The pattern of test results that we have reported is consistent with a simple analysis in terms of competition between lever pressing and nosepoking; with the lever press intervention simply resulting in further lever presses that compete with nosepoking during the critical stimuli. If such response competition does indeed explain our results, then why should it not have exerted a similar influence in the experiments by Blaisdell et al. (2006a) and Experiment 3 of Leising et al. (2008)? One plausible possibility is that apparently minor differences between the procedures employed by the two laboratories might have resulted in differences in the degree to which lever pressing and nosepoking competed with each other in the conditions under investigation. This possibility could have been directly assessed had Blaisdell et al. (2006a) recorded lever pressing during the stimuli, but they did not. Alternatively, it is possible that there was something in the procedures used in the two sets of studies that influenced the likelihood that rats would exhibit their capacity for causal reasoning. This possibility is less parsimonious.

Where does all this leave the question of whether rats engage in causal reasoning? In two experiments, we were unable to replicate the key interaction between training type and test condition that was critical to central claim in Blaisdell et al. (2006a, p. 1020); the claim that rats "made causal inferences . . . . . consistent with

causal Bayes net theories." This failure to replicate is not merely a failure to find an effect: We observed an effect of intervention following all of the training conditions, not just a subset of them. Although we do not want to claim on the basis of our results that rats cannot reason, we do have direct evidence that is entirely consistent with a simple (associative) account of "causal reasoning" in rats. This account is grounded on one of the guiding general principles of comparative psychology and it seems especially apt to conclude this article with Lloyd Morgan's canon: "In no case may we interpret an action as the outcome of the exercise of a higher psychical faculty, if it can be interpreted as the outcome of one which stands lower in the psychological scale" (1894, p. 53).

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