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Causal Reasoning in Rats

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Empirical research with nonhuman primates appears to support the view that causal reasoning is a key cognitive faculty that divides humans from animals. The claim is that animals approximate causal learning using associative processes. The present results cast doubt on that conclusion. Rats made causal inferences in a basic task that taps into core features of causal reasoning without requiring complex physical knowledge. They derived predictions of the outcomes of interventions after passive observational learning of different kinds of causal models. These competencies cannot be explained by current associative theories but are consistent with causal Bayes net theories.

The ability to acquire and reason with causal knowledge is among our most central human cognitive competences (*1*). Causal knowledge serves two important functions: It allows us to predict outcomes on the basis of observations, and it underlies our ability to control events in the world. We investigated whether animals understand the relation between observations and interventions, which some philosophers regard as a core feature of causal reasoning (*2–4*).

Although a number of psychologists have claimed that both humans and animals use basic associative mechanisms to learn about causal relations (*5*), human studies have demonstrated a deeper understanding of causal relations that cannot be reduced to associative learning (*6–8*). In contrast, research on the cognitive competencies of nonhuman primates concludes that they demonstrate a superficial understanding of the association between tool use and its effects but fail to comprehend the unobservable physical mechanisms underlying these relations [(*9–11*), but see (*12*, *13*)]. It may well be, however, that nonhuman animals lack knowledge about physical mechanisms but still are capable of basic causal reasoning. The capacity to derive predictions for interventions after purely observational learning is a core competency that is not reducible to associative learning (*14*).

Humans and animals can learn associations between passively observed events (Pavlovian conditioning) as well as between interventions and outcomes (instrumental conditioning). Moreover, these two learning modes may interact (*15*). An understanding of the interrelations between observations ("seeing") and interventions ("doing"), however, requires more sophisticated representations. Simple transfer from observational learning can lead to inadequate predictions for interventions. For example, barometer readings statistically predict the weather, but at the same time, setting the barometer to an arbitrary reading does not influence the weather. Both relations could be learned with associative mechanisms in separate observational and instrumental learning trials, but associative theories are incapable of deriving correct predictions for interventions after observational learning when no prior instrumental learning is available.

The causal model in Fig. 1A shows how predictions for interventions can be derived from observations. Imagine that an animal learns in an observational Pavlovian learning phase that a light cue (L) temporally precedes both a tone stimulus (T) and food (F), thus learning a common-cause model with two effects (top panel). After learning this model, observing T should, via L, lead to the predictive inference that F should also be present. However, if the animal learns in the test phase that a newly introduced lever turns on T, it should be more

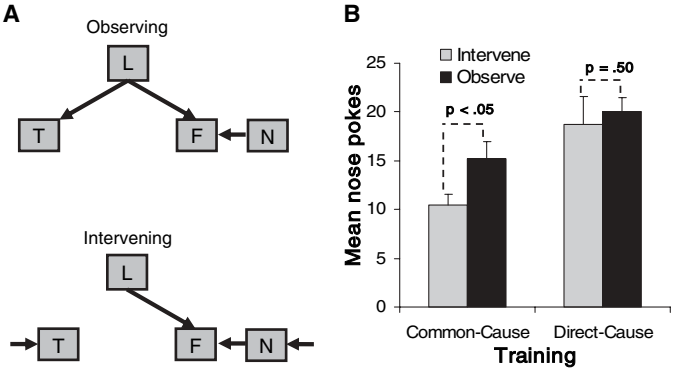

Fig. 1. (**A**) Causal model used in experiment 1. L (light) is the common cause of T (tone) and F (food); N (noise) is the direct cause of F. (Top panel) Observed causal relations. (Bottom panel) Model modified under the assumption of an intervention in T and N. (**B**) Experiment 1: Mean nose pokes in response to test stimulus T ($P < 0.05$) in the common-cause condition and to N ($P > 0.50$) in the direct-cause condition after a lever press (intervene) or no lever press (observe). Bars indicate SEM. Planned comparisons from a two-way mixed analysis of variance (ANOVA) are shown. There was a main effect of causal model (common or direct), $F(1, 21) = 6.01$, $P < 0.05$, and an interaction between causal model and test condition (intervene or observe), $F(1, 21) = 4.31$, $P = 0.05$.

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reluctant to predict F (bottom panel). Generating T by means of an alternative cause—the lever—does not predict F because the manipulation of an effect does not influence its cause (L). A dissociation between seeing and doing would be remarkable, because in the observational learning phase T is positively correlated with L.

The only theoretical model that derives correct predictions for interventions from observational learning data is causal Bayes nets (*2*–*4*). Predictions for observations make use of the full causal model acquired during observational learning (top panel). Predictions for interventions, however, are based on a modified graph (bottom panel); the insight that generating T in the common-cause model happens independently of its usual cause L is modeled by removing the causal arrow that leads into the manipulated effect: a manipulation called graph surgery (*3*). Because the manipulated T is unrelated to L, the likelihood of L's other effect F should not be altered by T's presence.

A possible alternative associationist explanation of the failure to expect F after an intervention in T may be that the animal does not expect F because it lacks prior instrumental learning experiences relating lever presses to F. This alternative theory, however, erroneously also predicts a failure to expect F in the presence of noise (N), after these events had been paired during observational learning (Fig. 1A). Because of the direct causal link between N and F, causal Bayes nets predict that animals should equally expect F, regardless of whether N is observed or generated by an intervention. Recent research with similar tasks has shown (*14*) that human participants are capable of deriving correct predictions for interventions on the basis of observational data (*16*).

In experiment 1, 32 rats were trained on the causal model shown in Fig. 1A, using an observational Pavlovian procedure (*17*). Training consisted of three types of trials interspersed within each session. The first type of trial was presentations of stimulus L (a 10-s flashing light or click train) forward-paired with stimulus T (a 10-s tone or noise); the second was presentations of stimulus L forward-paired with stimulus F (a 10-s delivery of sucrose solution); the third was simultaneous presentations of stimulus N (a 10-s noise or tone) and 10 s of F. We trained each causal link in the common-cause model separately to make it more likely that subjects did not induce a direct link between effects T and F.

Why did the rats not induce that the alternative effect is always absent when the cause and one effect are present (that is, conditioned inhibition)? With few learning trials, rats tend to integrate individual learning relations into a coherent integrated model. Only after many trials do rats encode the explicit absence of the nonpresented cues (*18*). Supporting these findings, the results of all our experiments show that rats induced second-order excitatory rather than inhibitory relations (*19*). Apparently, in the initial phases of learning, rats tend to conservatively treat the absent but expected events as possibly present but missed. A similar ability to combine individually learned causal links into complex causal models has been demonstrated in humans (*20*).

Do rats treat L as a common cause of both T and F, and do they correctly differentiate between seeing and doing with respect to T and N? Rats were allocated to one of four test conditions and were placed in the conditioning chamber with a lever present. This lever had not been present in the observational learning phase, so that no prior instrumental knowledge was available. Rats in condition intervene-T received a 10-s presentation of T each time they pressed the lever. Rats in condition observe-T merely observed presentations of T independently of any emitted lever presses. Conditions intervene-N and observe-N were conducted in an identical fashion, except that N was either the product of an intervention by lever pressing or was observed. We recorded the number of nose pokes into the magazine where F had been delivered during the training phase, to assess the rats' expectation of F.

Causal Bayes nets predict that observing T in condition observe-T should lead the rats to reason that the temporally prior cause L was probably present (but missed), and to consequently expect that F should also be present; therefore, they should emit many nose pokes. In contrast, rats in condition intervene-T should attribute T to their intervention and therefore expect L and consequently its effect, F, to occur with the probability corresponding to the base rate of its cause L. Consequently, we should observe a lower rate of nose poking in condition intervene-T than in condition observe-T. There should not be any difference in rates of nose poking, however, between conditions intervene-N and observe-N. The direct causal relationship should lead the rats to expect F regardless of whether N was observed or intervened on at test. Unlike causal Bayes nets, associationist theories predict equivalent nose poking in the presence of T in both the observe and intervene conditions.

Figure 1B shows the mean rate of nose poking per 10-s presentation of stimuli T and N as a function of test condition (with a maximum rate of 100 nose pokes per presentation). As predicted by causal Bayes nets, rats that produced T through a lever-press intervention (condition intervene-T) made fewer nose pokes than rats that merely observed T (condition observe-T). However, rats that intervened in N (condition Intervene-N), which was trained as a direct predictor of F, did not nose poke less than rats that merely observed N (condition observe-N). [An analysis of the lever press data ruled out selective interference between lever pressing and nose poking (*17*).]

In experiment 1, we observed a dissociation between seeing and doing within the common-cause model, whereas both tasks led to identical expectations with the direct causal link, which is consistent with causal Bayes nets. A critic might point out that we found a dissociation within a complex causal model with two separately learned links (the common-cause model), whereas we found similar responses to the less complex direct link. To rule out complexity or second-order learning as the basis of our dissociation, we compared a common-cause condition with an equally complex causal chain in which the individual causal links were also presented separately (that is, second-order conditioning) (Fig. 2). Whereas causal Bayes nets predict a dissociation between seeing and doing in the common-cause model, no such dissociation is expected for the causal chain. Regardless of whether the initial cause (T) of the chain is observed or generated by means of an intervention, the intermediate (L) and final effect (F) should equally be expected.

In experiment 2a, rats received either common-cause training, as in experiment 1, or causal-chain training, which was identical except that T preceded L during observational learning (*17*). In the test phase, groups

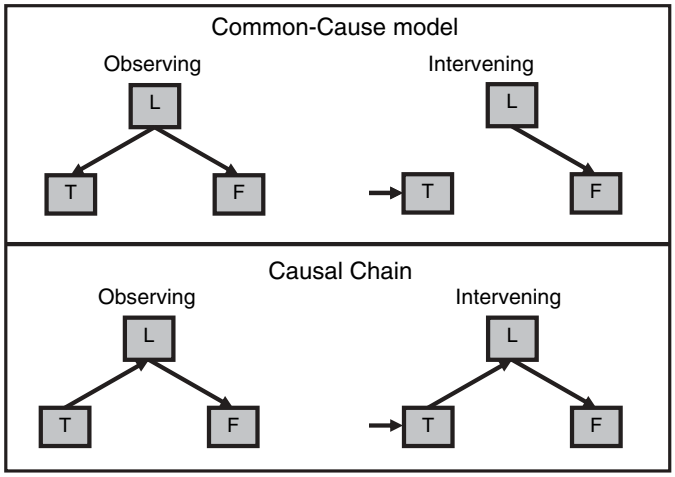

Fig. 2. Common-cause and causal chain models from experiment 2. (**Left**) Observed causal relations. (**Right**) Model modified under the assumption of an intervention in T.

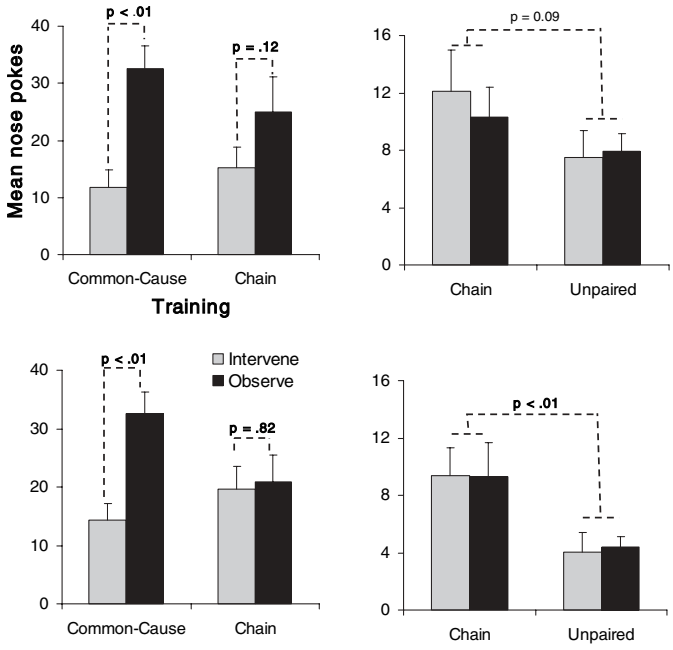

Fig. 3. Experiment 2a (left panels): Mean nose pokes during test stimulus T (top panel) or 10 s after the termination of T (bottom panel) after a lever press (intervene; $P = 0.01$ in both panels) or no lever press (observe; $P = 0.12$ and 0.82 in top and bottom panels, respectively). Common-cause and chain indicate the type of causal model training. Bars indicate SEM. Planned comparisons from two-way ANOVAs are shown. Experiment 2b (right panels): Mean nose pokes during test stimulus T (top panel) or 10 s after the termination of T (bottom panel) after a lever press (intervene) or no lever press (observe). Chain and unpaired indicate the type of causal model training. Bars indicate SEM. $P = 0.09$ and 0.01 in top and bottom panels, respectively, for the main effect of training.

common-cause–intervene and chain-intervene received presentations of T each time the lever was pressed. Groups common-cause–observe and chain-observe merely observed T. We report the number of nose pokes during the 10-s presentation of T and during the 10-s period beginning 10 s after the termination of T (post-T interval 2) for all subjects. In the chain condition, F should rationally be expected between 10 and 20 s after delivery of T (*19*). In contrast, the expected time of delivery of F for rats that received common-cause training is during T itself.

Figure 3 shows the mean rate of nose poking on test trials with T. Group common-cause–intervene nose poked less than group common-cause–observe, which replicates the pattern of experiment 1. In contrast, no difference was found between groups chain-intervene and chain-observe, as predicted by causal Bayes nets.

Rats in group chain-intervene did not nose poke more than did rats in group common-cause–intervene. This low level of responding does not reflect a failure to learn a causal chain, however. Experiment 2b replicated the chain condition and added groups for which T and L were unpaired during observational learning (*17*). Figure 3 reveals no difference between seeing and doing, as predicted by causal Bayes nets. Moreover, responding in the causal-chain groups was higher than in the unpaired groups, which signifies that the rats had indeed learned the second-order chain relations.

A number of researchers have recently concluded that causal reasoning is a faculty that divides humans from animals (*7*, *9*–*11*). The present results cast doubt on that conclusion. With tasks that did not require complex physical knowledge, the experiments have shown that rats grasp the relationship between seeing and doing. Rats made correct inferences for instrumental actions on the basis of purely observational learning, and they correctly differentiated between common-cause models, causal chains, and direct causal links. These results contradict the view that causal learning in rats is solely driven by associative learning mechanisms, but they are consistent with causal Bayes net theories. The core competency of reasoning with causal models seems to be already in place in animals, even when elaborate physical knowledge may not yet be available.

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Causal Reasoning in Rats

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Experiment 1

Subjects. Thirty-two experimentally-naïve, male Long-Evens rats (*Rattus norvegicus*) were pair-housed in plastic tubs with wood shaving substrate in a vivarium maintained on a 12-hr dark/light cycle. Experimental manipulations occurred during the dark portion of the cycle. Rats were maintained at 85% of free-feeding weights and were handled prior to the study. Rats were randomly assigned to two of four conditions (*n*s = 16 per condition), either Intervene-T and Observe-N, or Observe-T and Intervene-N.

Apparatus. Each of eight identical experimental chambers measuring 30 L x 25 W x 20 H cm was housed in a separate sound- and light-attenuating chest. The walls and ceiling of the chamber were constructed of clear Plexiglas and the floors were constructed of stainless-steel rods measuring 0.5 cm in diameter, spaced 1.5 cm center-to-center. The enclosure also contained a 28-V, 100 mA shielded incandescent house light and a diffuse light mounted on the left-side wall of the conditioning chamber.

Each chamber was equipped with a liquid dipper that could deliver .05 cc sucrose solution (20%). Three speakers on the outside walls of the chamber could deliver a high-frequency tone stimulus (3000 Hz) 8 dB(A) above a background noise of 62 dB(A), a white noise stimulus 8 dB(A), and a click train stimulus (6/s) 8 dB(A). A flashing light (2/s) could be produced by turning off the house light and flashing the diffuse light. All stimuli, including sucrose delivery, were 10s in duration. Levers could be inserted into the cage 4 cm to the left of the food niche, 6.5 cm above the floor.

Procedure.

Magazine training. Day 1: The levers were retracted and 10s access to sucrose was delivered every 20 ± 15s to train rats to approach the feeding niche.

Causal model training. Days 2-7: All rats received four of each of the following daily trials pseudorandomly interspersed within each session with an interval of 5 ± 3 min: Light or click (Stimulus L; counterbalanced within group) followed by Stimulus T (tone or noise, counterbalanced within group) (onset of T coincided with the termination of L); L followed by F (onset of F coincident with the termination of L); and Stimulus N (noise or tone, counterbalanced within group) and F simultaneously presented (with co-onsets and co-terminations). (We presented N and F simultaneously because, based on a common-cause model assumption, it would be rational to expect T and F to occur simultaneously.)

Testing. Levers were extended into the chambers for testing. Day 8: Rats in Condition Intervene-T received a 10s presentation of T each time they pressed the lever (except that lever presses during the presentation of T had no nominal consequence). Each lever press by a rat in Condition Intervene-T also caused the presentation of T for a rat in Condition Observe-T in an adjacent chamber (i.e., a yoking procedure). Rats in Condition Intervene-N received a presentation of N for each lever press, and caused the presentation of N for a rat in Condition Observe-N in an adjacent chamber. Day 9: Rats that received Condition Intervene-T on Day 8 received Condition Observe-N on Day 9, rats that received Condition Observe-T on Day 8 received Condition Intervene-N on the Day 9, rats that received Condition Intervene-N on Day 8 received Condition Observe-T on Day 9, and finally, rats that received Condition Observe-N on Day 8 received

Condition Intervene-T on Day 9. This counterbalancing scheme ensured that rats tested for lever pressing for one type of stimulus (e.g., T) on the first test session received yoked presentations of the other stimulus (e.g., N) on the second test session, and vice versa. Test sessions were 30 min. Lever pressing in the Observe conditions had no nominal consequence. All lever presses and nose pokes were recorded. In all experiments reported here, subjects nose poking more than two standard deviations below their group mean during the last session of acquisition were excluded from analysis for failing to acquire nose poke responding to Stimulus L. Data from five subjects (three from conditions Intervene-X/Observe-Y, and two from conditions Observe-X/Intervene-Y) that met this elimination criterion were discarded. Furthermore, data from four subjects (two from condition Intervene-X/Observe-Y, and two from condition Observe-X/Intervene-Y) were lost due to equipment failure, leaving a final $n = 23$ for data analyses.

Results

No differences among the test conditions were found in mean number of nose pokes during the background—in the absence of the test stimulus—indicating no contribution of baseline levels of responding to the test stimulus (Means ± SEM = 279 ± 57, 305 ± 99, 288 ± 82, and 284 ± 49 for Conditions Intervene-T, Observe-T, Intervene-N, and Observe-N, respectively). A two-way mixed ANOVA on training model (Common-Cause vs. Direct-Cause) and testing condition (Intervene vs. Observe) found neither main effects nor an interaction, Fs$(1, 21) < 1.0$.

A two-way mixed ANOVA on mean number of nose pokes (max = 100) to T or N at test found a main effect of causal model (Common-Cause vs. Direct-Cause), $F(1, 21) =$

6.01, $P < .05$, and an interaction between causal model and testing condition (Intervene vs. Observe), $F(1, 21) = 4.31$, $P = .05$. Planned comparisons revealed less nose poking in Condition Intervene-T than in Condition Observe-T, $F(1, 21) = 5.12$, $P < 0.05$, while nose poking did not differ in Conditions Intervene-N and Observe-N, $F(1, 21) < 1.0$. Rats in Condition Intervene-T nose poked less than rats in Condition Intervene-N, $F(1, 21) = 10.02$, $P < .01$, but there were no differences in mean nose pokes between Conditions Observe-T and Observe-N, $F(1, 21) < 1.0$. Thus, only interventions in effect T of a common cause attenuated nose poking at test.

Perhaps nose poking was lower in Condition Intervene-T than in Condition Observe-T simply because nose poking and lever pressing were incompatible responses. If a rat in Condition Intervene-T was lever pressing at the onset of T, it could not simultaneously place its nose in the feeder. There are two arguments against this possibility. First, nose-poke scores were high in Condition Intervene-N, which did not differ from Condition Observe-N. Thus, it was physically possible to first lever press and subsequently nose poke at a high rate. Second, no difference was found in the rate of lever pressing between Condition Intervene-T (M ± SEM = 21 ± 6) and Observe-T (M ± SEM = 19 ± 6), $t(17) < 1$. No difference in the rate of lever pressing was found for Conditions Intervene-N (M ± SEM = 18 ± 5) and Observe-N (M ± SEM = 11 ± 4), $t(17) = 1.46$, $P > .10$, further denouncing the response interference explanation.

One might expect N to act as a conditioned reinforcer (S1), and thereby support a higher rate of lever pressing in Condition Intervene-N than in Observe-N. Although there was a non-significant tendency in this direction, our procedure was not designed to assess

conditioned reinforcement, which is parameter dependent and typically a relatively weak effect compared to primary reinforcement. A large number of first-order pairings (e.g., 120 (S2) or 600 (S3)) are typically required to produce conditioned reinforcement (our procedure had only 24 N-F pairings), and testing conventionally involves two levers (we only had one), one producing the conditioned reinforcer, the other producing a novel stimulus (S2-S3).

Experiment 2a

Subjects and Apparatus. Forty rats participated as in Experiment 1. Apparatus and stimuli as in Experiment 1, except that only the flashing light served as L and the tone and noise served as T and N, counterbalanced within group.

Magazine training. As in Experiment 1.

Phase 1: Sensory preconditioning. Days 2-5: Rats in Groups Common-Cause-Intervene and Common-Cause-Observe each received 6 daily trials of L→T (i.e., L followed by T) and N pseudorandomly interspersed. (N trials were included to reduce generalization). Rats in Groups Chain-Intervene and Chain-Observe each received 6 daily trials of T→L and N. Trials occurred with a mean interval of 5 ± 3 min during each daily 60-min session.

Phase 2: First-order conditioning. Days 6-7: All rats received 12 trials of L→F in each daily 60-min session with a mean interval of 5 ± 3 min.

Testing. Levers were extended into the chambers for testing and the bulb on which L had been presented during training was removed from the experimental chamber. (A pilot experiment found it necessary to remove the light bulb on which L was

presented during training to produce reliable responding to T after causal-chain training (S4). Thus, the bulb was removed from the apparatus prior to testing for all subjects in Experiments 2a and 2b to equate conditions among all groups).

Days 8-9: In each 30-min session, rats in Groups Common-Cause-Intervene and Chain-Intervene received presentations of T each time the lever was pressed. Rats in Groups Common-Cause-Observe and Chain-Observe received presentations of T yoked to the number of presentations of T by rats in Groups Common-Cause-Intervene and Chain-Intervene, respectively, in the same manner as described in Experiment 1. We recorded the number of nose pokes during T, during the 10s after T (Post-T Interval 1), and during the 10s after Post-T Interval 1 (Post-T Interval 2). We analyzed nose pokes during T and during Post-T Interval 2. Due to a programming error, lever press data were lost for all subjects in Experiments 2a and 2b preventing us from analyzing lever press data in these experiments. One subject from Group Chain-Observe was eliminated for meeting the elimination criterion on acquisition of responding to L during acquisition.

Results

No group differences in mean number of nose pokes during the background were found, thus, baseline responding did not contribute to responding to the test stimulus (Means ± SEM = 11895 ± 1664, 11081 ± 1206, 13096 ± 1256, and 11642 ± 2756 for Conditions Common-Cause-Intervene, Common-Cause-Observe, Chain-Intervene, and Chain-Observe, respectively). An ANOVA on Training Model (Common-Cause vs. Chain) and Testing Condition (Intervene vs. Observe) found neither main effects nor an interaction, Fs(1, 35) < 1.0.

A two-way ANOVA conducted on mean nose pokes during T revealed a main effect of Testing, $F(1, 35) = 12.65$, $P < 0.01$, but no interaction between Training Model and Testing. Although subjects nose poked numerically less in Group Chain-Observe than in Group Common-Cause-Observe, this difference was not significant, $F(1, 35) = 1.54$, $P > .22$. However, responding in Group Chain-Intervene also did not differ from Group Common-Cause-Intervene, $F < 1.0$.

A similar ANOVA conducted on mean nose pokes during Post-T Interval 2 revealed a main effect of Testing, $F(1, 35) = 6.43$, $P < 0.05$, and a Training Model X Test interaction, $F(1, 35) = 4.86$, $P < 0.05$. Planned comparisons revealed that rats in Group Common-Cause-Intervene nose poked less during Post-T Interval 2 than did rats in Group Common-Cause-Observe, $F(1, 35) = 11.55$, $P < 0.01$. No difference was found between Groups Chain-Intervene and Chain-Observe, $F < 1.0$. Subjects in Group Chain-Observe nose poked less than those in Group Common-Cause-Observe, $F(1, 35) = 4.42$, $P < .05$. No difference in number of nose pokes was found between Groups Chain-Intervene and Common-Cause-Intervene, $F = 1.0$. It is not clear why the Chain groups should make fewer nose pokes than Group Common-Cause-Observe. Nevertheless, it is important to document learning of the chain. Thus, Experiment 2b compared responding in the Chain groups to that of an unpaired cue.

Experiment 2b

Subjects, Apparatus, and Magazine training. As in Experiment 2a.

Phase 1: Sensory preconditioning. As in Experiment 2a for Groups Chain-Intervene and Chain-Observe. Rats in Groups Unpaired-Intervene and Unpaired-Observe

received 6 trials each of T, L, and N separately with a mean interval of 5 ± 3 min during each daily 60-min session.

Phase 2: First-order conditioning. As in Experiment 2a.

Testing. Days 8-11: T was presented in the following manner in each 30-min session. Groups Chain-Intervene and Chain-Observe received treatment identical to Experiment 2a. Rats in Groups Unpaired-Intervene and Unpaired-Observe received presentations of T when rats in Group Unpaired-Intervene lever pressed. Data recorded as in Experiment 2a. One subject from Group Unpaired-Observe was removed for meeting the elimination criterion. Data from one rat from Group Chain-Intervene and two from Group Unpaired-Intervene were lost due to equipment failure.

Results

Mean number of nose pokes during the background did not differ among groups—indicating no contribution of baseline levels of responding to the test stimulus (Means = 920 ± 233, 1211 ± 224, 1173 ± 258, and 1598 ± 286 for Conditions Chain-Intervene, Chain-Observe, Unpaired-Intervene, and Unpaired-Observe, respectively). A two-way ANOVA on Training Model (Chain vs. Unpaired) and Testing Condition (Intervene vs. Observe) found neither main effects nor an interaction, Fs(1, 33) < 1.53.

A two-way ANOVA conducted on nose poking during T revealed a marginal effect of Training Model, $F(1, 33) = 2.96$, $P = 0.09$, but neither a main effect of Testing nor an interaction between Training Model and Testing. A similar ANOVA conducted on mean nose pokes during the Post-T Interval 2 revealed a main effect of Training Model,

$F(1, 33) = 8.32$, $P < 0.01$, but neither a main effect of Testing nor a Training Model X Testing interaction.

The main effect of training model during Post-T Interval 2 demonstrates significant nose poking in the Chain groups relative to the Unpaired groups, establishing the effectiveness of causal-chain training to establish a causal chain representation. The findings also demonstrate that rats exhibit a sensitivity to the T→L and L→F temporal relationships underlying the causal-chain (S5). Thus, rats in the Chain groups expected F during Post-T Interval 2, but not before.

Supporting References

S1. B. F. Skinner, *The behavior of organisms: an experimental analysis,* (Appleton-Century Company, Inc., New York, 1938).

S2. N. Winterbauer, B. Balleine, *unpublished*.

S3. W.L. Inglis, M. C. Olmstead, T. W. Robbins, *Behav. Neuro.* **114**, 285 (2000)

S4. We thank K. Holyoak for suggesting this procedure.

S5. H. I. Savastano, R. R. Miller, *Behavioural Processes* **44**, 147 (1998).

S6. All procedures involved in this research met the approval of UCLA's animal care and followed institutional guidelines.