DISCUSSION 3

Prior to beginning this discussion, please read and view the following required sources:

- "Flexible Retrieval: When True Inferences Produce False Memories"
- "Memory Suppression in Alzheimer's Disease"
- "Mnemonic Instruction in Science and Social Studies for Students with Learning Problems: A Review"
- "Short-Term Memory and Long-Term Memory Are Still Different"
- "The Development of Real-Time Stability Supports Visual Working Memory Performance: Young Children's Feature Binding Can Be Improved Through Perceptual Structure"
- *Memory*

In your initial post, you will apply what you learned from each of the five articles, but you will discuss the findings and implications for just one of these articles. The articles are assigned based on the first letter of your last name. Please see the list below to determine which of the articles you will focus on for your initial post based on the first letter of your last name:

- A through E: "Flexible Retrieval: When True Inferences Produce False Memories"

In your initial post,

- Explain the empirical research presented in your assigned article, applying appropriate citations and references.
- Describe, in your own words, how the research relates to your own experiences as well as how this area of psychology may have affected your past or current beliefs about memory development. Do the research findings refute or support your current beliefs, and in what ways? Are there variables about memory of which you were unaware based on your article?
- Apply skeptical inquiry to the potential problems that might arise from research in the area of memory, and relate it to the APA's Ethical Principles of Psychologists and Code of Conduct.
- Provide a rationale for whether or not this premise is valid and/or appropriate based on the findings reported by the assigned articles.

It is recommended that you research articles in the Ashford University Library to support your assertions if the required articles do not provide sufficient information. Your initial post should be at least 500 words in length.

Required Source For Discussion

Journal of Experimental Psychology:
Learning, Memory, and Cognition
2017, Vol. 43, No. 3, 335–349

0278-7393/17/$12.00 http://dx.doi.org/10.1037/xlm0000340

Flexible Retrieval: When True Inferences Produce False Memories

Alexis C. Carpenter and Daniel L. Schacter
Harvard University

Episodic memory involves flexible retrieval processes that allow us to link together distinct episodes, make novel inferences across overlapping events, and recombine elements of past experiences when imagining future events. However, the same flexible retrieval and recombination processes that underpin these adaptive functions may also leave memory prone to error or distortion, such as source misattributions in which details of one event are mistakenly attributed to another related event. To determine whether the same recombination-related retrieval mechanism supports both successful inference and source memory errors, we developed a modified version of an associative inference paradigm in which participants encoded everyday scenes comprised of people, objects, and other contextual details. These scenes contained overlapping elements (AB, BC) that could later be linked to support novel inferential retrieval regarding elements that had not appeared together previously (AC). Our critical experimental manipulation concerned whether contextual details were probed *before* or *after* the associative inference test, thereby allowing us to assess whether (a) false memories increased for successful versus unsuccessful inferences, and (b) any such effects were specific to after compared with before participants received the inference test. In each of 4 experiments that used variants of this paradigm, participants were more susceptible to false memories for contextual details after successful than unsuccessful inferential retrieval, but only when contextual details were probed after the associative inference test. These results suggest that the retrieval-mediated recombination mechanism that underlies associative inference also contributes to source misattributions that result from combining elements of distinct episodes.

Keywords: inference, false memory, episodic memory, memory, associative processes

Supplemental materials: http://dx.doi.org/10.1037/xlm0000340.supp

Episodic memory allows individuals to recollect particular past experiences (Tulving, 2002). It has been well established that episodic memories are not literal representations of past experiences, but instead depend on constructive processes that are sometimes prone to error and distortion (cf., Bartlett, 1932; Brainerd & Reyna, 2005; Loftus, Miller, & Burns, 1978; McClelland, 1995; Roediger, 1996; Schacter, 1996). Such memory errors can arise as a consequence of multiple processes, including knowledge- or schema-based inferences made about the meaning of observed actions or events, which are later integrated into memories of presented materials, such as sentences and stories (e.g., Alba & Hasher, 1983; Bransford, Barclay, & Franks, 1972; Bransford & Franks, 1971); activation of associations to semantically related words that may produce subsequent false recognition of a nonpresented word that is strongly associated to the list items that were presented (e.g., Gallo, 2006; Roediger & McDermott, 1995); and a variety of influences that operate during retrieval of past experiences, such as misleading suggestions or instructions to imagine what might have happened earlier (Loftus, 2003, 2005; Shaw & Porter, 2015).

While these and other forms of memory distortion could be viewed as flaws or defects in episodic memory, a number of researchers have built on Bartlett's (1932) seminal insights and suggest instead that such errors can be viewed as byproducts of adaptive constructive processes (Schacter, 2012) that play a functional role in memory but produce errors or distortions as a direct consequence of doing so (cf., Howe, 2011; Howe, Wilkinson, Garner, & Ball, 2016; Newman & Lindsay, 2009; Schacter, 2001; Schacter, Guerin, & St. Jacques, 2011). Bartlett (1932), of course, focused on the functional role of schemata in guiding constructive retrieval, which he maintained "must always be supposed to be operating in any well-adapted organic response" (p. 201) but also contributed to the memory distortions that he documented. Others have argued that such well-established memory errors as the misinformation effect and associative false recognition may reflect, respectively, the operation of adaptive memory updating processes and retention of themes and meanings (for review, see Schacter et al., 2011). More recently, it has become increasingly clear that episodic memory supports a variety of cognitive functions, including imagining future experiences (e.g., Schacter et al., 2012; Szpunar, 2010), inferential processing (e.g., Zeithamova, Dominick, & Preston, 2012; Zeithamova & Preston, 2010), means-end problem solving (e.g., Madore & Schacter, 2014; Sheldon, McAndrews, & Moscovitch, 2011), and divergent creative think-

This article was published Online First December 5, 2016.

Alexis C. Carpenter and Daniel L. Schacter, Department of Psychology and Center for Brain Science, Harvard University.

This research was supported by National Institute of Mental Health Grant MH060941 and National Institute on Aging Grant AG08441 to D.L.S. We thank Karen Campbell, Aleea Devitt, Alison Preston, and Preston Thakral for helpful comments on a draft of the manuscript.

Correspondence concerning this article should be addressed to Alexis C. Carpenter, Department of Psychology, Harvard University, 33 Kirkland Street, Cambridge, MA 02138. E-mail: alexiscarpenter@g.harvard.edu

ing (e.g., Madore, Addis, & Schacter, 2015). An important feature of episodic memory that supports these and other adaptive functions is the capacity to flexibly retrieve and recombine information from distinct past experiences into novel representations. For example, according to the *constructive episodic simulation hypothesis* (Schacter & Addis, 2007a, 2007b), the capacity to flexibly recombine elements of past experiences is crucial for our ability to imagine or simulate new situations that might occur in the future. Similarly, recent evidence suggests that flexible recombination plays a key role in our capacity to make inferences based on distinct past events that share a common feature (Zeithamova, Dominick, & Preston, 2012; Zeithamova & Preston, 2010).

In line with the theoretical perspectives noted earlier that emphasize the close link between adaptive aspects of episodic memory and susceptibility to memory errors, the constructive episodic simulation hypothesis also holds that the functional benefits of flexible retrieval and recombination are accompanied by a cost: vulnerability to memory errors such as source misattribution and false recognition that can result from mistakenly combining elements of distinct past experiences (Schacter & Addis, 2007a, 2007b; for related views, see Dudai & Carruthers, 2005; Suddendorf & Corballis, 2007). There is indeed evidence that memory errors can result from mistakenly combining features of distinct episodic or autobiographical memories (e.g., Burt, Kemp, & Conway, 2004; Devitt, Monk-Fromont, Schacter, & Addis, 2016; Odegard & Lampinen, 2004). However, we are not aware of any study that has directly tested the central idea of the constructive episodic simulation hypothesis that the same flexible recombination process that supports an adaptive cognitive process can also produce memory errors that result from miscombining elements of distinct past experiences.

To test this idea, we required a task that both requires flexible recombination and supports an adaptive cognitive process. The *associative inference task* used by Preston and colleagues fits these requirements (e.g., Preston, Shrager, Dudukovic, & Gabrieli, 2004; Zeithamova, Dominick, & Preston, 2012; Zeithamova & Preston, 2010). Associative inference is an adaptive process that allows people to link together related information acquired in distinct episodes to make novel connections that they have not directly experienced (Zeithamova, Schlichting, & Preston, 2012). For example, if one sees two different individuals entering the same house on different days, retrieving and recombining details of the two episodes allows one to infer that the two individuals are related in some way by their relationship with the house. This kind of flexible recombination is quite similar to the kind of flexible recombination that is required to draw on elements of past experiences to construct simulations of novel future events, as discussed by Schacter and Addis (2007a, 2007b). In previous studies using the associative inference procedure, participants learned direct associations between two items (AB) and then learned direct associations between two items that included one member of the previously studied pair (BC) and also learned indirect associations based on the overlapping pairs (AC). Later, participants received a memory test for both the direct AB and direct BC associations. In addition, participants received an associative inference test for the indirect association (AC). Here, they are told that the link between the two items is mediated by a third item (B) that was previously associated with both the A and C items, and to choose which of two items was linked to A via the shared B association.

There are two ways that participants can perform successfully on the associative inference test. First, during study of BC, participants may bring to mind the related AB pair and encode an integrated representation (ABC) that is later retrieved during the associative inference test (*integrative encoding*; e.g., Shohamy & Wagner, 2008). Second, participants may engage in flexible recombination at the time of retrieval, bringing to mind and combining the previously studied AB and BC pairs during the associative inference task. Neuroimaging evidence suggests that both mechanisms contribute to associative inference (Zeithamova, Dominick, & Preston, 2012; Zeithamova & Preston, 2010). In the present study, we adapted the associative inference paradigm developed by Zeithamova and Preston (2010) to assess whether mechanisms linked with inferential processing (i.e., retrieval-related recombination and encoding-related integration) also contribute to source memory errors. As noted earlier, pioneering studies on memory distortion have already shown that knowledge- or schema-driven inferences about sentences and stories can contribute to memory errors (e.g., Alba & Hasher, 1983; Branford & Franks, 1971), but the kind of inferential processing tapped by Zeithamova and Preston's associative inference task focuses specifically on combining elements from distinct episodes that are not linked by preexisting knowledge or schemas, and thus likely draws on different processes than the meaning-based inferences elicited in classic studies of sentence and story processing. Indeed, it is precisely because the associative inference paradigm developed by Zeithamova and Preston (2010) targets flexible recombination processes that link elements of distinct episodes that their paradigm is well suited for testing the key claim of the constructive episodic simulation hypothesis—that the same flexible retrieval processes that are used to combine elements of distinct episodes into functionally useful, novel representations can also produce memory errors that result from mixing up elements of these episodes. More generally, we attempt to determine whether the domain of adaptive memory distortions, where a memory error results from carrying out a cognitive operation that has demonstrably beneficial consequences on another aspect of performance, extends to associative inference. Although the literature on associative inference has grown considerably during the past decade (for review, see Schlichting & Preston, 2015), we are not aware of any studies using the associative inference paradigm, which requires combining elements of distinct episodes, that have linked successful associative inference with memory errors.

In our version of the associative inference paradigm, during an initial session participants study scenes that include AB items (e.g., a person [A] and a toy [B] in a room with a white couch; Figure 1) and then study scenes comprised of BC items (e.g., the toy [B] and a different person [C] in a room with a brown couch). Participants are instructed to try to learn both the direct association between each person and object (AB and BC) and the indirect association between the two people based on the shared object (AC). After a delay, participants return for a second session in which they are tested for direct associations (AB, BC) and perform an associative inference test for novel combinations that are linked via the B item (AC). To test whether retrieval-related recombination processes underlying successful inference can also contribute to memory errors, memory for contextual details from both the AB and BC scenes is also probed (e.g., What

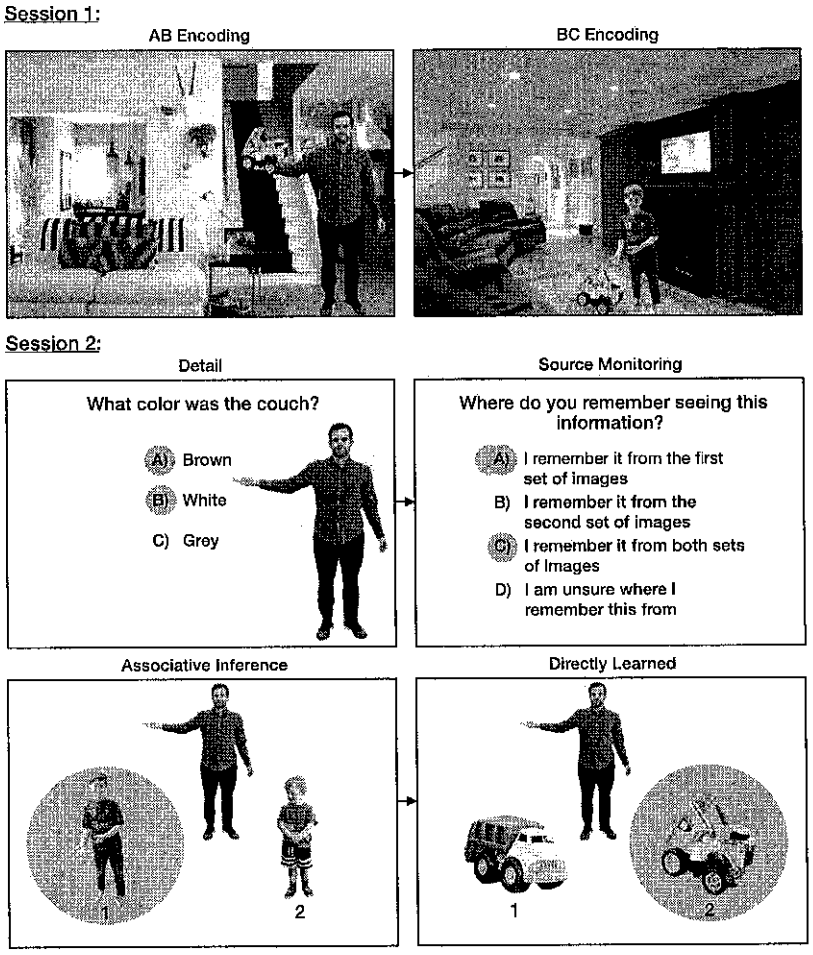

Figure 1. Illustration of materials, stimuli, and test displays from Experiments 1a and 1b. The Session 1 section shows one example of an AB image in which the man is item "A" and the toy truck is item "B" and the corresponding BC image in which the boy is item "C." The Session 2 section shows one example of a detail and source monitoring question linked to the example AB image. For each detail question, participants saw a cutout of the "A" or "C" individual presented to the right of the question in order to indicate to which event the question referred. False memories occurred when participants chose both the misinformation detail (e.g., brown couch) during the detail question and attributed the misinformation detail incorrectly to either the original event or both events—as indicated by the red (dark grey) circles. True memories occurred when participants both chose the correct detail during the detail question (e.g., white couch) and attributed the correct detail correctly to the original event—as indicated by the green (light grey) circles. Other example detail questions for this ABC triad included: Where were the stairs located? What color were the walls in the room? What was this individual sitting/standing on? What was hanging on the wall directly behind this individual? and so forth. It is important that all of these questions relate to two contradictory details from images AB and BC (e.g., stairs directly behind vs. to the far left; yellow vs. white walls; wood floors vs. carpet; potted plants vs. picture frames; etc.). The green (light grey) circles indicate the correct answer for the associative inference and directly learned questions. Participants saw these images without the red (dark grey) and green (light grey) circles. Individuals depicted here, or their guardians, gave signed consent for their likenesses to be published in this article. See the online article for the color version of this figure.

color was the couch?) followed immediately by a source memory test (In which set of images do you remember seeing this information?). For one half of the AB and BC scenes, *detail/source memory tests* were given before the test of direct (AB, BC) and indirect (AC) associations, and for the other half, the detail/source memory tests were given after the tests of direct and indirect associations. For the detail/source test, a *true memory* is defined as a response in which the participants both chose the correct item and attributed the source of their memory correctly (e.g., white couch attributed to AB scene), whereas a *false memory* is defined as a response for which the participant both chose the item from the overlapping image (e.g., BC) and misattributed its source (e.g., brown couch attributed to AB scene; see Methods for further details).

The critical comparison concerns the proportions of false memories on the detail/source tests given before versus after the associative inference test, for correct compared with incorrect associative inference trials (i.e., AC). We distinguish among three competing hypotheses:

(1) If recombination during retrieval both enhances associative inference performance and also increases susceptibility to false memories, then the proportion of false memories should be higher for correct than incorrect inference trials, but only when the detail/source test is given *after* the associative inference test (during which recombination occurs); there should be no difference in the proportion of false memories for correct versus incorrect inference trials when the detail/source test is given before the associative inference test.

(2) If the proportion of false memories is higher for correct than incorrect inference trials both when the detail/source tests are given before and after the associative tests, then these effects would be attributable to integrative encoding processes.

(3) If there is no link at all between source memory misattributions and associative inference, then there should be no difference between the proportion of false memories for correct and incorrect inference trials regardless of when the detail/source test is given.

To test these hypotheses, and determine the reliability of the results across variations in procedure and experimental parameters, we conducted three initial experiments that used the same basic paradigm and differed only in methodological details. Experiment 1 used a 24-hr study-test delay and a two-alternative forced choice on the associative inference test, whereas Experiment 2 used a 48-hr study-test delay and included an additional "neither" option on the forced-choice test (see Experiments 1 and 2 for rationale regarding these changes). In Experiment 3, we increased the delay between the directly learned (AB and BC) and associative inference trials (AC) on the one hand, and the second set of detail and source questions on the other, to assess the durability of the effects observed in Experiments 1 and 2. All three of these experiments provided evidence in favor of the first hypothesis outlined previously: The proportion of false memories was higher for correct than for incorrect inference trials, and only when the detail/source test was given *after* the associative inference test, during which recombination occurs. These findings implicate recombination during retrieval in both associative inference and memory misattribution, in line with the constructive episodic simulation hypothesis. To further test the hypothesis, in Experiment 4 we eliminated tests of directly learned associations (AB and BC), which in theory could have contributed to the effects that we attributed to flexible recombination. However, Experiment 4 again replicated the major findings of Experiments 1–3, providing further evidence that recombination during retrieval is responsible for the observed pattern of false memory effects.

Experiments 1 and 2

Because Experiments 1 and 2 used nearly identical procedures with only minor differences, we report the methods and results for these experiments together. To provide an overview of the basic procedure, participants came to the lab for two sessions, separated by a 24-hr (Experiment 1) or a 48-hr (Experiment 2) delay. The delay in Experiment 2 was extended from 24- to 48-hr to more closely replicate accuracy levels on the directly learned and associative inference test reported in the standard associative inference paradigm designed by Preston and colleagues (Preston et al., 2004; Zeithamova, Dominick, & Preston, 2012; Zeithamova & Preston, 2010; Zeithamova, Schlichting, & Preston, 2012). Participants completed a modified version of an associative inference paradigm based on prior studies by the Preston group (Preston et al., 2004; Zeithamova, Dominick, & Preston, 2012; Zeithamova & Preston, 2010; Zeithamova, Schlichting, & Preston, 2012). In the first session, participants intentionally encoded directly learned associations between individual "A" and object "B" followed by a second set of images with overlapping associations between object "B" and individual "C" (Figure 1); participants were also presented with nonoverlapping X-Y individual-object pairs to reduce performance for directly learned associations below ceiling levels. A total of 24 ABC triads and 24 XY pairs were used in the experiment. In the second session, participants were tested on directly learned associations (i.e., AB, BC, XY) and associative inference trials consisting of novel combinations of person pairings (i.e., AC). In addition, for one half of the ABC triads, participants answered 10 detail and source monitoring questions per triad *before* they were tested on directly learned and associative inference trials. For the alternate half of the triads, participants answered these detail and source monitoring questions *after* the directly learned and associative inference trials for all items. As noted earlier, the contrast between performance on the detail and source memory tests given before compared with after the directly learned/associative inference trials is critical to testing the three key hypotheses we outlined.

Method

Participants. For both experiments, participants were recruited via advertisements at Boston University and Harvard University. All had normal vision and no history of neurological impairment. They gave informed consent, were treated in accordance with guidelines approved by the ethics committee at Harvard University, and received either course credit or pay for completing the study. Experiment 1 included 26 young adults (mean age = 21.20, SD = 2.19; 15 women). Two participants were excluded from the true, false, and foil memory analyses because they were 100% accurate on the associative inference trials; thus, our final sample consisted of 24 participants. Participants who were 100% accurate on the associative inference trials were removed from the true, false, and foil memory analyses because they did not have any trials for which they correctly recalled the directly learned relationships *and* incorrectly inferred the relationship between item A and item C, thereby precluding meaningful comparisons of successful inference to unsuccessful inference both before and after flexible retrieval. Experiment 2 included 25 young adults (mean age = 20, SD = 1.93; 14 women). One participant was excluded from all analyses for having prior experience with several of the task stimuli; thus, our final sample consisted of 24 participants. Prior to the experiment, we decided on a sample size of 24 based on previous work utilizing a similar source monitoring paradigm (Okado & Stark, 2005). We stopped data collection after reaching the target of 24 participants with analyzable data.

AB and BC encoding. All experimental sessions were executed on an Apple desktop computer using PsychoPy2 (v1.80.03).

Stimuli consisted of 72 still color images depicting everyday life events (e.g., walking to work). Color images of common objects (e.g., toy truck) and individuals were superimposed on outdoor and indoor scenes. Scenes were counterbalanced across participants such that each scene was used equally often for both AB and BC pairs. Using Adobe Photoshop CC 2015, 48 overlapping pairs (24 AB pairs, 24 BC pairs—24 total ABC triads) and 24 unique, nonoverlapping pairs (XY) were constructed. Overlapping AB and BC pairs were constructed such that two individuals (A and C) shared an association with an overlapping object (B; i.e., one ABC triad). XY pairs were constructed of unique individual—object pairs that did not share an overlapping association with other pairings.

Participants received one of two versions of the AB encoding task, which consisted of 36 images (i.e., AB and XY) followed by the corresponding BC encoding task, which consisted of 36 images (i.e., BC and XY; Figure 1). Each image was randomly presented for 10 seconds within each encoding block (i.e., AB encoding and BC encoding). Participants were instructed to learn both the direct associations (i.e., AB, BC) and the indirect associations (i.e., AC) along with the contextual information presented. Following each image, participants were asked to provide a judgment of learning on a scale from 1 to 4 (1 = *definitely forget*, 4 = *definitely remember*). These judgments were collected to ensure participants' attention during the encoding phase.

Detail and source monitoring. Ten detail and source monitoring questions were constructed for each of the 24 ABC triads (5 questions related to image AB and 5 questions related to image BC). Detail questions were directly related to background details that were present but contradictory in the AB and BC scenes and did not reference the overlapping "B" object (Figure 1). A cutout of the cue individual (i.e., either "A" or "C") was presented to the right of each detail question to indicate which scene the question was referring to (Figure 1). For each detail question, participants were given three options: the correct item, a misinformation item, and an unrelated foil item. The misinformation item consisted of information from the overlapping image in the triad (e.g., if the detail question were related to the AB image, the misinformation item would be a contradicting detail from the BC image, such as a brown couch when a white couch had appeared in the AB image). Foil items were details that were not presented in either of the overlapping images (e.g., gray couch). Following each detail question, participants indicated where they remember seeing this contextual detail (i.e., the source of the information; Figure 1). Participants were given four possible answer choices: (a) the first set of images—AB, (b) the second set of images—BC, (c) both sets of images, or (d) unsure. Immediately following participants' source monitoring response, they were asked to rate their confidence in their response on a scale from 1 to 4 (1 = *very unsure*, 4 = *very sure*). The presentation order of each set of questions (i.e., detail, source, confidence) was randomized for each participant and the questions were self-paced.

Participants answered the 10 detail and source monitoring questions for one half of the 24 ABC triads before being tested on the directly learned and associative inference trials. After participants were tested on the directly learned and associative inference trials, they completed the 10 detail and source monitoring questions for the alternate half of the 24 ABC triads.

Directly learned and associative inference trials. Following the first half of the detail and source questions, participants were tested on directly learned (AB and BC) and associative inference trials (AC). During each directly learned trial, a single cue individual (e.g., an "A" or "C" individual) was presented at the top of the screen and two choice objects were presented at the bottom of the screen (e.g., two "B" objects from different ABC triads; Figure 1). On the associative inference trials, a cue individual (A) was presented along with two individuals at the bottom of the screen (i.e., the correct "C" individual from the ABC triad and a lure "C" individual from another triad). Participants were instructed on associative inference trials that the association between the cue (A) and the correct choice (C) was indirect, mediated through an object (B) that shared an association with both the cue and the correct choice during encoding. In Experiment 1, participants were required to make a forced-choice decision indicating which of the two choice objects/individuals was associated with the cue individual. In Experiment 2, participants were given a third option to respond "neither" when they believed that the items had not been previously paired, in order to reduce the possible influences of guessing on the associative inference task. If participants could not recall which of the two options given was paired with the cue individual, they were allowed to guess in Experiment 1, which could add noise to the source memory data by including triads for which participants were not actually able to successfully infer the relationship between item A and item C, but appeared to do so because of guessing. Thus, in an attempt to replicate the results of Experiment 1 and also control for the potential effects of guessing, a neither option was included for Experiment 2. It is important that for both directly learned and associative inference trials, the incorrect choice was a familiar item that had been studied in the context of another individual independent from the cue. Thus, correct responses required retrieval of learned associations and could not be made based on the familiarity of the choice. The presentation order of the trials was randomized with the only constraint being that AC associative inference trials were shown before their corresponding AB and BC directly learned trials in order to ensure that participants were not able to form an association between "A" and "C" individuals during test. Following each of the directly learned and associative inference trials, participants rated their confidence in their response on a scale from 1 to 4 (1 = *very unsure*, 4 = *very sure*).

Coding of true and false memories. Consistent with previous work using a similar detail and source monitoring paradigm (Okado & Stark, 2005), true memories were defined as detail questions for which the participant both chose the correct detail *and* attributed the source of their memory correctly to the currently cued image. False memories were defined as detail questions for which the participant both chose the misinformation detail *and* attributed the misinformation detail incorrectly to either the currently cued image or both images in the triad (Figure 1). False, true and foil memories were analyzed for ABC triads for which participants correctly inferred the relationship between "A" and "C" compared with triads for which the inference was not correctly made. In addition, false, true and foil memories were evaluated both before explicit retrieval of the inference (i.e., before AC associative inference trials) and after the retrieval of the inference in order to selectively compare the distinct effects of

integration during encoding and flexible recombination at retrieval on subsequent memory errors.

Results

Directly learned and associative inference trials.

Experiment 1. First, we evaluated overall accuracy on directly learned and associative inference trials. Performance on both directly learned and associative inference trials was generally accurate, and there was no significant difference in the proportion of directly learned (M_{direct} = 0.78, SE = 0.02) compared with associative inference trials ($M_{\text{associative inference}}$ = 0.80, SE = 0.03) that participants answered correctly, $t(25) = -.99$, $p > .250$, mean difference = −0.02, 95% confidence interval (CI) [−0.06, 0.02], d = .19. Consistent with previous research (Zeithamova & Preston, 2010), we found significantly longer reaction times (RTs) on associative inference trials ($M_{\text{associative inference}}$ = 4,425 ms, SE = 341) compared with directly learned trials (M_{direct} = 3,306 ms, SE = 314), suggesting that there may be an additional recombination-related retrieval mechanism necessary for inferential versus direct retrieval, $t(25) = 9.48$, $p < .001$, mean difference = 1.12, 95% CI[0.88, 1.36], d = 1.85. Furthermore, participants assigned significantly higher confidence ratings to their responses on directly learned (M_{direct} = 3.34, SE = 0.07) compared with associative inference trials ($M_{\text{associative inference}}$ = 2.87, SE = 0.09), suggesting that participants were more confident in their memory for events that they had directly experienced compared with those resulting from recombination, $t(25) = 9.38$, $p <$ .001, mean difference = 0.47, 95% CI[0.37, 0.58], d = 1.89.

Experiment 2. Again we evaluated overall accuracy on directly learned and associative inference trials. There was a trend toward a significant difference in the proportion of directly learned (M_{direct} = 0.69, SE = 0.03) compared with associative inference trials ($M_{\text{associative inference}}$ = 0.64, SE = 0.03) that participants answered correctly, t(23) = 2.00, p = .057, mean difference = 0.05, 95% CI[−0.002, 0.10], d = .42. While this trend is slightly different from the results reported in Experiment 1, it does not affect the main hypotheses of interest, which are related to the false memory analyses. Consistent with results from Experiment 1, we found significantly longer RTs on associative inference trials ($M_{\text{associative inference}}$ = 4,401 ms, SE = 185) compared with directly learned trials (M_{direct} = 3,052 ms, SE = 129), suggesting an additional recombination-related retrieval mechanism for inferential versus direct retrieval, $t(23) = 5.66$, $p < .001$, mean difference = 1.35, 95% CI[0.99, 1.71], d = 1.62. Furthermore, results revealed that participants were significantly more confident in their responses on directly learned (M_{direct} = 3.22, SE = 0.09) compared with associative inference trials ($M_{\text{associative inference}}$ = 2.83, SE = 0.08; $t(23) = 5.67$, $p < .001$, mean difference = 0.39, 95% CI [0.25, 0.53], d = 1.18). Thus, results from Experiment 2 replicate those in Experiment 1.

False memory.

Experiment 1. To assess the effects of integrative encoding and recombination mechanisms at retrieval on subsequent memory errors, we examined source memory errors for the detail and source monitoring questions with a 2 (Time: before vs. after inference retrieval) × 2 (Inference: correct vs. incorrect inference) repeated-measures analysis of variance (ANOVA). It is important that only trials for which participants correctly remembered the directly learned association were included in subsequent analyses. See Supplementary Table 1, available online, for the raw number of trials per bin for each experiment. Results revealed a trend toward a main effect of time, $F(1, 23) = 3.04$, p = .095, η_p^2 = .12; no main effect of inference, $F(1, 23) = 2.40$, p = .135, η_p^2 = .10; and a significant time by inference interaction, $F(1, 23) = 7.05$, p = .014, η_p^2 = .24 (Figure 2a). Participants falsely attributed more details to the overlapping event after successful inference retrieval (M_{after} = 0.27, SE = 0.01) than before successful inference retrieval (M_{before} = 0.21, SE = 0.02; $t(23) = 4.05$, $p < .001$, mean difference = 0.06, 95% CI[0.03, 0.08], d = .83). Furthermore, participants did not falsely attribute more details to the overlapping event after unsuccessful inference retrieval (M_{after} = 0.21, SE = 0.02) than before unsuccessful inference retrieval (M_{before} = 0.22, SE = 0.02; $t(23) = .385$, $p > .250$, mean difference = −0.01, 95% CI[−0.05, 0.04], d = .08). Participants did not falsely attribute

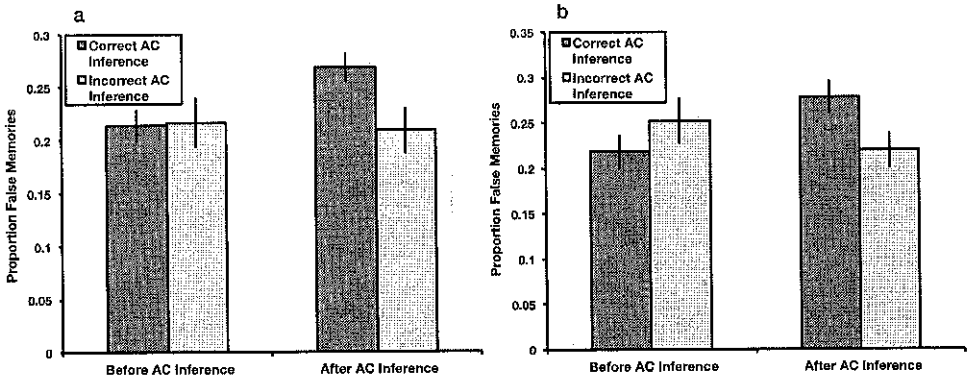

Figure 2. Proportion of false memories in Experiment 1 (a) and Experiment 2 (b). Performance on detail and source monitoring questions was examined both before and after either successful or unsuccessful inference. It is important that only trials for which participants responded correctly to directly learned trials were included in this analysis. Results revealed a significant time by inference interaction in both Experiment 1 and 2. Subsequent t tests confirm that false memories selectively increased only following successful associative inference. Error bars represent ± 1 *SEM*.

more details to the overlapping event before successful inference retrieval ($M_{correct}$ = 0.21, SE = 0.02) than before unsuccessful inference retrieval ($M_{incorrect}$ = 0.22, SE = 0.02; $t(23) = .143$, $p > .250$, mean difference = 0.003, 95% CI[−0.04, 0.05], $d = .03$). Critically, participants falsely attributed more details to the overlapping event after successful inference retrieval ($M_{correct}$ = 0.27, SE = 0.01) than after unsuccessful inference retrieval ($M_{incorrect}$ = 0.21, SE = 0.02; $t(23) = 2.73$, $p = .012$, mean difference = 0.06, 95% CI[0.01, 0.10], $d = .56$), suggesting that recombination processes underlying successful inference at retrieval may also lead to source memory errors.

Experiment 2. Identical to Experiment 1, we examined source memory errors for the detail and source monitoring questions with a 2 (Time: before vs. after inference retrieval) × 2 (Inference: correct vs. incorrect inference) repeated-measures ANOVA. Results revealed no main effect of time, $F(1, 23) = .357$, $p > .250$, $\eta_p^2 = .02$; no main effect of inference, $F(1, 23) = .57$, $p > .250$, $\eta_p^2 = .02$; and a significant time by inference interaction, $F(1, 23) = 7.40$, $p = .012$, $\eta_p^2 = .24$ (Figure 2b). Participants falsely attributed more details to the overlapping event after successful inference retrieval (M_{after} = 0.28, SE = 0.02) than before successful inference retrieval (M_{before} = 0.22, SE = 0.02; $t(23) = 2.48$, $p = .021$, mean difference = 0.06, 95% CI[0.01, 0.11], $d = .51$). Furthermore, participants did not falsely attribute more details to the overlapping event after unsuccessful inference retrieval (M_{after} = 0.22, SE = 0.02) than before unsuccessful inference retrieval (M_{before} = 0.25, SE = 0.03; $t(23) = -1.022$, $p > .250$, mean difference = −0.03, 95% CI[−0.10, 0.03], $d = .21$). Participants did not falsely attribute more details to the overlapping event before successful inference retrieval ($M_{correct}$ = 0.22, SE = 0.02) than before unsuccessful inference retrieval ($M_{incorrect}$ = 0.25, SE = 0.03; $t(23) = 1.40$, $p = .175$, mean difference = 0.03, 95% CI[−0.02, 0.08], $d = .29$). Critically, participants falsely attributed more details to the overlapping event after successful inference retrieval ($M_{correct}$ = 0.28, SE = 0.02) than after unsuccessful inference retrieval ($M_{incorrect}$ = 0.22, SE = 0.02; $t(23) = 2.56$, $p = .018$, mean difference = 0.06, 95% CI[0.01, 0.11], $d = .52$), replicating results from Experiment 1 and suggesting that recombination during retrieval required for successful inference may be linked to source memory errors.

True memory.

Experiment 1. To examine the effects of integrative encoding and recombination mechanisms at retrieval on successful source memory, we examined correct responses on the detail and source monitoring questions with a 2 (Time: before vs. after inference retrieval) × 2 (Inference: correct vs. incorrect inference) repeated-measures ANOVA. Results revealed no main effect of time, $F(1, 23) = 2.33$, $p = .141$, $\eta_p^2 = .09$; no main effect of inference, $F(1, 23) = .10$, $p > .250$, $\eta_p^2 = .02$; but a significant time by inference interaction, $F(1, 23) = 6.83$, $p = .016$, $\eta_p^2 = .23$. Participants attributed more details to the correct source after successful inference retrieval (M_{after} = 0.23, SE = 0.02) than before successful inference retrieval (M_{before} = 0.17 SE = 0.02; $t(23) = 3.82$, $p = .001$, mean difference = 0.06, 95% CI[0.03, 0.09], $d = .82$). By contrast, participants did not attribute more details to the correct source after successful inference retrieval ($M_{correct}$ = 0.23, SE = 0.02) than after unsuccessful inference retrieval ($M_{incorrect}$ = 0.20 SE = 0.03; $t(23) = 1.04$, $p > .250$, mean difference = 0.03, 95% CI[−0.03, 0.08], $d = .21$), and did not attribute more details to the correct source before successful inference retrieval ($M_{correct}$ = 0.17, SE = 0.02) than before unsuccessful inference retrieval ($M_{incorrect}$ = 0.22, SE = 0.03; $t(23) = 1.50$, $p = .146$, mean difference = 0.04, 95% CI[−0.02, 0.10], $d = .31$). Furthermore, participants did not attribute more details to the correct source after unsuccessful inference retrieval (M_{after} = 0.20, SE = 0.03) than before unsuccessful inference retrieval (M_{before} = 0.22, SE = 0.03; $t(23) < 1$, $p > .250$, mean difference = 0.01, 95% CI[−0.03, 0.06], $d = .12$). The increase in true memory from before inference retrieval to after appears to be attributable to changes in "unsure" responses on the source monitoring test following recognition of the correct item: Participants were significantly less likely to respond unsure following successful inference when they correctly recognized the detail (M_{after} = 0.12, SE = 0.05) than before successful inference (M_{before} = 0.26, SE = 0.06; $t(23) = 3.04$, $p = .008$, mean difference = 0.14, 95% CI[0.04, 0.23], $d = .18$).

Experiment 2. A 2 (Time: before vs. after inference retrieval) × 2 (Inference: correct vs. incorrect inference) repeated-measures ANOVA on correct responses to the detail and source monitoring questions revealed no main effect of time, $F(1, 23) = .40$, $p > .250$, $\eta_p^2 = .02$; no main effect of inference, $F(1, 23) = .55$, $p > .250$, $\eta_p^2 = .02$; and no time by inference interaction, $F(1, 23) = 1.34$, $p > .250$, $\eta_p^2 = .06$. Thus, true memory scores were similar both before (M_{before} = 0.18, SE = 0.03) and after successful inference retrieval (M_{after} = 0.21, SE = 0.03). In addition, true memory scores were similar both before (M_{before} = 0.16, SE = 0.03) and after unsuccessful inference retrieval (M_{after} = 0.20, SE = 0.03).

Foil memory.

Experiment 1. To assess whether critical patterns of source misattribution errors are specific to related items from previously studied episodes, we examined foil memories, which were defined as detail questions for which participants chose the unrelated foil option (e.g., gray couch) and attributed the information to either the currently cued image or both images in the triad. We conducted a 2 (Time: before vs. after inference retrieval) × 2 (Inference: correct vs. incorrect inference) repeated-measures ANOVA to evaluate participants' foil memory scores. Results revealed no main effect of time, $F(1, 23) = 1.16$, $p > .250$, $\eta_p^2 = .05$; no main effect of inference, $F(1, 23) = 1.71$, $p = .204$, $\eta_p^2 = .07$; and no time by inference interaction, $F(1, 23) = 1.59$, $p = .220$, $\eta_p^2 = .07$. Thus, foil memory scores were similar both before (M_{before} = 0.18, SE = 0.01) and after successful inference retrieval (M_{after} = 0.18, SE = 0.01). In addition, foil memory scores were similar both before (M_{before} = 0.14, SE = 0.03) and after unsuccessful inference retrieval (M_{after} = 0.17, SE = 0.02).

Experiment 2. We conducted a 2 (Time: before vs. after inference retrieval) × 2 (Inference: correct vs. incorrect inference) repeated-measures ANOVA to evaluate participants' foil memory scores. Results revealed no main effect of time, $F(1, 23) = 1.04$, $p > .250$, $\eta_p^2 = .04$; no main effect of inference, $F(1, 23) = 1.28$, $p > .250$, $\eta_p^2 = .05$; and no time by inference interaction, $F(1, 23) = 1.22$, $p > .250$, $\eta_p^2 = .05$. Thus, foil memory scores were similar both before (M_{before} = 0.21, SE = 0.02) and after successful inference retrieval (M_{after} = 0.17, SE = 0.02). In addition, foil memory scores were similar both before (M_{before} = 0.21, SE = 0.03) and after unsuccessful inference retrieval (M_{after} = 0.21, SE = 0.02).

Discussion

The results of Experiments 1 and 2 provide evidence that flexible retrieval processes required for successful associative inference also produce increases in false memories as a result of source misattributions: memory errors increased significantly *after* but not *before* successful compared with unsuccessful inferential retrieval. This pattern was observed both when the test of directly learned and associative inference items was two-alternative forced-choice (Experiment 1), and also when a third "neither" option was provided (Experiment 2); the effect was also robust across both a 24-hr study-test delay (Experiment 1) and a 48-hr study-test delay (Experiment 2).

The finding that source misattributions occurred more often for correct versus incorrect inferences constitutes evidence for a link between processes that support associative inference and those that contribute to false memories; if there were no such link, then source memory errors would not differ for correct and incorrect inferences. This finding alone, however, does not allow us to distinguish whether integrative encoding or flexible retrieval is responsible for the observed increased of source memory errors related to successful inference. However, the finding that the observed increase in false memories for correct inference occurred only *after* the associative inference test was given implicates flexible retrieval, rather than integrative encoding, as the key process responsible for the boost in false memories. Furthermore, foil memory scores (i.e., detail questions for which participants chose the unrelated foil option) showed no relationship to correct versus incorrect inferences either before or after the associative inference test was given. This finding indicates that the observed source memory error effects are selective to previously experienced details, details that seemingly migrated between the AB and BC episodes as a consequence of successful inference.

Experiment 3

In Experiments 1 and 2, we replicated the key effect of successful inference on false memories across minor variations in procedure, suggesting that the effect is reliable. However, the absolute magnitude of the effect is not large, and because the critical tests in Experiments 1 and 2 were administered in immediate succession, we do not know whether the process of recombination during retrieval that supports successful inference results in only a transient change in participants' susceptibility to source memory errors. To further assess the reliability of the key effect, and to determine whether the effects of recombination allowing for successful associative inference on subsequent source memory error lasts beyond the brief interval between the main tests and further shows a longer-lasting effect on participants susceptibility to source memory error, in Experiment 3 we introduced a 30-min delay between the directly learned (AB and BC)/associative inference trials (AC) on the one hand and the second set of detail and source questions on the other.

Method

Participants. Experiment 3 included 24 young adults (mean age = 20, SD = 2.07; 15 women). No participants were excluded from the analyses; thus, our final sample consisted of 24 participants.

Summary of the procedure. Participants came to the lab for two sessions, separated by a 48-hr delay. The design parameters, stimuli, and coding of true and false memories were exactly the same in Experiment 3 as in Experiment 2 with one exception. During the second session, following the test of the directly learned (AB and BC) and associative inference trials (AC), participants completed 30-min of unrelated filler tasks (e.g., simple math problems) before completing the second half of the detail and source questions. As noted earlier, in Experiment 3 we introduced a 30-min delay between the directly learned/associative inference trials and the second set of detail and source questions.

Results and Discussion

Directly learned and associative inference trials. First, we evaluated overall accuracy on directly learned and associative inference trials. Performance on both directly learned and associative inference trials was generally accurate, and there was no significant difference in the proportion of directly learned (M_{direct} = 0.72, SE = 0.02) compared with associative inference trials ($M_{\text{associative inference}}$ = 0.71, SE = 0.02) that participants answered correctly, $t(23)$ = 0.62, $p > .250$, mean difference = 0.01, 95% confidence interval (CI) = [−0.03, 0.06], d = .13). Consistent with previous research (Zeithamova & Preston, 2010) and results from Experiment 1 and 2, we found significantly longer RTs on associative inference trials ($M_{\text{associative inference}}$ = 4,186 ms, SE = 196) compared with directly learned trials (M_{direct} = 3,057 ms, SE = 123), suggesting that there may be an additional recombination-related retrieval mechanism necessary for inferential versus direct retrieval, $t(23) = -7.46$, $p < .001$, mean difference = −1.13, 95% CI[−1.44, −.82], d = 1.52. Furthermore, participants assigned significantly higher confidence ratings to their responses on directly learned (M_{direct} = 3.23, SE = 0.08) compared with associative inference trials ($M_{\text{associative inference}}$ = 2.83, SE = 0.10), suggesting that participants were more confident in their memory for events that they had directly experienced compared with those resulting from recombination, $t(23)$ = 8.97, $p < .001$, mean difference = 0.40, 95% CI[0.31, 0.50], d = 1.83.

False memory. Identical to Experiments 1 and 2, we examined source memory errors for the detail and source monitoring questions with a 2 (Time: before vs. after inference retrieval) × 2 (Inference: correct vs. incorrect inference) repeated-measures ANOVA. Results revealed no main effect of time, $F(1, 23)$ = 0.46, $p > .250$, η_p^2 = .02; no main effect of inference, $F(1, 23)$ = 1.17, $p > .250$, η_p^2 = .05; and a significant time by inference interaction, $F(1, 23)$ = 5.89, p = .023, η_p^2 = .20 (Figure 3). Participants falsely attributed more details to the overlapping event after successful inference retrieval (M_{after} = 0.27, SE = 0.02) than before successful inference retrieval (M_{before} = 0.22, SE = 0.02; $t(23)$ = 3.46, p = .002, mean difference = 0.05, 95% CI[0.02, 0.08], d = .71). Furthermore, participants did not falsely attribute more details to the overlapping event after unsuccessful inference retrieval (M_{after} = 0.21, SE = 0.03) than before unsuccessful inference retrieval (M_{before} = 0.23, SE = 0.03; $t(23) = -0.63$, $p > .250$, mean difference = −0.02, 95% CI[−0.09, 0.05], d = .13). Participants did not falsely attribute more details to the overlapping event before successful inference retrieval (M_{correct} = 0.22, SE = 0.02) than before unsuccessful inference retrieval ($M_{\text{incorrect}}$ = 0.23, SE = 0.03; $t(23)$ = .829, $p > .250$, mean

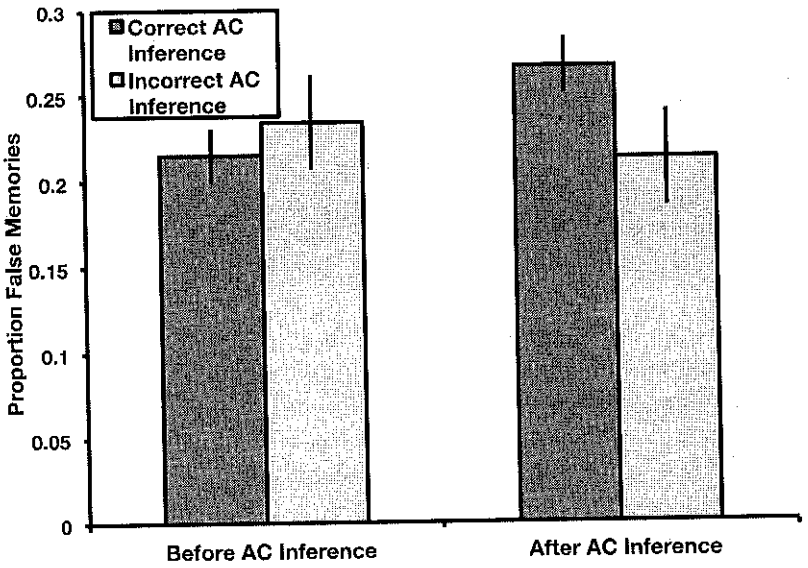

Figure 3. Proportion of false memories in Experiment 3. Performance on detail and source monitoring questions was examined both before and after either successful or unsuccessful inference. It is important that only trials for which participants responded correctly to directly learned trials were included in this analysis. Results revealed a significant time by inference interaction in Experiment 3. Subsequent *t* tests confirm that false memories selectively increased only following successful associative inference. Error bars represent ± 1 *SEM*.

difference = 0.02, 95% CI[−0.03, 0.06], d = .17). Critically, participants falsely attributed more details to the overlapping event after successful inference retrieval ($M_{correct}$ = 0.27, SE = 0.02) than after unsuccessful inference retrieval ($M_{incorrect}$ = 0.21, SE = 0.03; $t(23)$ = 2.49, p = .021, mean difference = 0.05, 95% CI[0.009, 0.10], d = .51), replicating results from Experiment 1 and 2, suggesting again that recombination during retrieval required for successful inference may be linked to source memory errors.

True memory. A 2 (Time: before vs. after inference retrieval) × 2 (Inference: correct vs. incorrect inference) repeated-measures ANOVA on correct responses to the detail and source monitoring questions revealed no main effect of time, $F(1, 23)$ = 0.46, $p > .250$, η_p^2 = .02; no main effect of inference, $F(1, 23)$ = 0.91, $p > .250$, η_p^2 = .04; and no time by inference interaction, $F(1, 23)$ = .042, $p > .250$, η_p^2 = .002. Thus, true memory scores were similar both before (M_{before} = 0.23, SE = 0.02) and after successful inference retrieval (M_{after} = 0.24, SE = 0.02). In addition, true memory scores were similar both before (M_{before} = 0.21, SE = 0.04) and after unsuccessful inference retrieval (M_{after} = 0.22, SE = 0.02).

Foil memory. We conducted a 2 (Time: before vs. after inference retrieval) × 2 (Inference: correct vs. incorrect inference) repeated-measures ANOVA to evaluate participants' foil memory scores. Results revealed no main effect of time, $F(1, 23)$ = 1.74, p = .200, η_p^2 = .07; no main effect of inference, $F(1, 23)$ = 0.62, $p > .250$, η_p^2 = .03; and no time by inference interaction, $F(1, 23)$ = 2.23, p = .150, η_p^2 = .09. Thus, foil memory scores were similar both before (M_{before} = 0.20, SE = 0.02) and after successful inference retrieval (M_{after} = 0.16, SE = 0.01). In addition, foil memory scores were similar both before (M_{before} = 0.16, SE = 0.02) and after unsuccessful inference retrieval (M_{after} = 0.17, SE = 0.02).

Overall, the pattern of results from Experiment 3 was essentially identical to that observed in Experiments 1 and 2: Participants made significantly more source misattributions for correct than incorrect inferences, but only when the detail and source monitoring test was given after the test of directly learned and associative inference items. Because the second source test was administered 30 min after completion of the directly learned and associative inference test, Experiment 3 indicates that the effects we have attributed to flexible recombination during retrieval are not simply transient influences that disappear following a filled delay.

Experiment 4

Although our central theoretical claim of Experiments 1–3 focuses on retrieval-related recombination processes that occurred during the associative inference test for previously unpaired AC items, participants were also tested for AB and BC items that did appear together previously. Thus, it is conceivable that the increase in source memory errors following the associative inference test is attributable to direct retrieval of previously studied pairs (AB, BC) as opposed to retrieval-related recombination processes. Two key features of the data from Experiments 1–3 speak against this possibility. First, if retrieval of directly learned associations were responsible for the increase in false memories, then false memory rates should have been similar for successful and unsuccessful inferential retrieval after the associative inference test, but as noted above memory errors increased significantly following successful compared with unsuccessful inferential retrieval. Second, neither experiment revealed significant differences in the number false memories before compared with after *unsuccessful* inferential retrieval. If testing of directly learned pairs during the associative inference test were responsible for the increased false memory effects after compared with before the associative inference test,

then those effects should have been observed for unsuccessful inference trials. However, no such effects were observed. While the results of Experiments 1–3 thus suggest that the testing of directly learned associations is not responsible for the key effects of successful inference on false memories, in Experiment 4 we assess this possibility empirically by testing directly learned associations only after both sets of detail and source monitoring tests were completed. If, as we have suggested, recombination during retrieval is responsible for observed increases in false memories, then the same critical pattern of results from previous experiments—more source misattributions for correct than incorrect inference items, after but not before the inference test—should be observed in Experiment 4, even though directly learned associations were not tested prior to the detail and source memory tests.

Method

Participants. Experiment 4 included 26 young adults (mean age = 20.70 years, SD = 2.19; 16 women). Two participants were excluded from the true and false memory analyses. One participant was excluded from the true, false, and foil memory analyses because they were 100% accurate on the associative inference trials, and one participant was excluded from all analyses for noncompliance during the second session (e.g., did not make responses during the detail and source monitoring questions); thus, our final sample consisted of 24 participants.

Summary of the procedure. Participants came to the lab for two sessions, separated by a 48-hr delay. The design parameters, stimuli, and coding of true and false memories were exactly the same in Experiment 4 as in Experiment 2, with the one exception. During the second session, following the first half of the detail and source questions, participants were only tested on associative inference trials (AC), thus eliminating the potential effect of retrieving directly learned associations on false memory following successful associative inference. However, to ensure that we still obtained a measure of performance on directly learned items, following the second half of the detail and source questions, participants were tested on directly learned trials (AB and BC).

Results and Discussion

Directly learned and associative inference trials. First, we evaluated overall accuracy on directly learned and associative inference trials. Performance on both directly learned and associative inference trials was generally accurate, and there was no significant difference in the proportion of directly learned (M_{direct} = 0.66, SE = 0.03) compared with associative inference trials ($M_{\text{associative inference}}$ = 0.70, SE = 0.03) that participants answered correctly, $t(24) = -1.01$, $p > .250$, mean difference = −0.04, 95% CI[−0.11, 0.04], d = .20. Consistent with previous research (Zeithamova & Preston, 2010) and results from Experiments 1–3, we found significantly longer RTs on associative inference trials ($M_{\text{associative inference}}$ = 5,140 ms, SE = 242) compared with directly learned trials (M_{direct} = 3,300 ms, SE = 148), suggesting that there may be an additional recombination-related retrieval mechanism necessary for inferential versus direct retrieval, $t(24) = -10.18$, $p < .001$, mean difference = −1.84, 95% CI[−2.21, −1.47], d = 2.04. Furthermore, participants assigned significantly higher confidence ratings to their responses on directly learned (M_{direct} = 3.15, SE = 0.12) compared with associative inference trials ($M_{\text{associative inference}}$ = 2.87, SE = 0.11), suggesting that participants were more confident in their memory for events that they had directly experienced compared with those resulting from recombination, $t(24) = 3.06$, $p = .005$, mean difference = 0.27, 95% CI[0.89, 0.46], d = 0.61.

False memory. Identical to Experiments 1–3, we examined source memory errors for the detail and source monitoring questions with a 2 (Time: before vs. after inference retrieval) × 2 (Inference: correct vs. incorrect inference) repeated-measures ANOVA. Results revealed no main effect of time, $F(1, 23) = 1.60$, $p = .219$, $\eta_p^2 = .07$; no main effect of inference, $F(1, 23) = .011$, $p > .250$, $\eta_p^2 = .00$; and a significant time by inference interaction, $F(1, 23) = 4.79$, $p = .039$, $\eta_p^2 = .17$ (Figure 4). Participants falsely attributed more details to the overlapping event after successful inference retrieval (M_{after} = 0.26, SE = 0.02) than before successful inference retrieval (M_{before} = 0.19, SE = 0.01; $t(23) = 3.20$, $p = .004$, mean difference = 0.07, 95% CI[0.03, 0.12], d = .65). Furthermore, participants did not falsely attribute more details to the overlapping event after unsuccessful inference retrieval (M_{after} = 0.23, SE = 0.03) than before unsuccessful inference retrieval (M_{before} = 0.23, SE = 0.04; $t(23) = .010$, $p > .250$, mean difference = 0.0004, 95% CI[−0.09, 0.09], d = .002). Participants did not falsely attribute more details to the overlapping event before successful inference retrieval (M_{correct} = 0.19, SE = 0.01) than before unsuccessful inference retrieval ($M_{\text{incorrect}}$ = 0.23, SE = 0.04; $t(23) = 1.22$, $p = .231$, mean difference = 0.04, 95% CI[−0.03, 0.10], d = .25). Critically, participants falsely attributed more details to the overlapping event after successful inference retrieval (M_{correct} = 0.26, SE = 0.02) than after unsuccessful inference retrieval ($M_{\text{incorrect}}$ = 0.23, SE = 0.03; $t(23) = 2.37$, $p = .027$, mean difference = 0.03, 95% CI[0.004, 0.64], d = .48), replicating results from Experiments 1–3, suggesting that recombination during retrieval required for successful inference may be linked to source memory errors.

True memory. A 2 (Time: before vs. after inference retrieval) × 2 (Inference: correct vs. incorrect inference) repeated-measures ANOVA on correct responses to the detail and source monitoring questions revealed no main effect of time, $F(1, 23) = 2.53$, $p = .13$, $\eta_p^2 = .10$; no main effect of inference, $F(1, 23) = 1.68$, $p = .21$, $\eta_p^2 = .07$; and no time by inference interaction, $F(1, 23) = .43$, $p > .250$, $\eta_p^2 = .02$. Thus, true memory scores were similar both before (M_{before} = 0.22, SE = 0.03) and after successful inference retrieval (M_{after} = 0.17, SE = 0.02). In addition, true memory scores were similar both before (M_{before} = 0.18, SE = 0.03) and after unsuccessful inference retrieval (M_{after} = 0.16, SE = 0.03).

Foil memory. We conducted a 2 (Time: before vs. after inference retrieval) × 2 (Inference: correct vs. incorrect inference) repeated-measures ANOVA to evaluate participants' foil memory scores. Results revealed no main effect of time, $F(1, 23) = 3.67$, $p = .068$, $\eta_p^2 = .14$; no main effect of inference, $F(1, 23) = 2.47$, $p = .130$, $\eta_p^2 = .10$; and no time by inference interaction, $F(1, 23) = 0.59$, $p > .250$, $\eta_p^2 = .03$. Thus, foil memory scores were similar both before (M_{before} = 0.18, SE = 0.02) and after successful inference retrieval (M_{after} = 0.19, SE = 0.02). In addition, foil memory scores were similar both before (M_{before} = 0.14, SE =

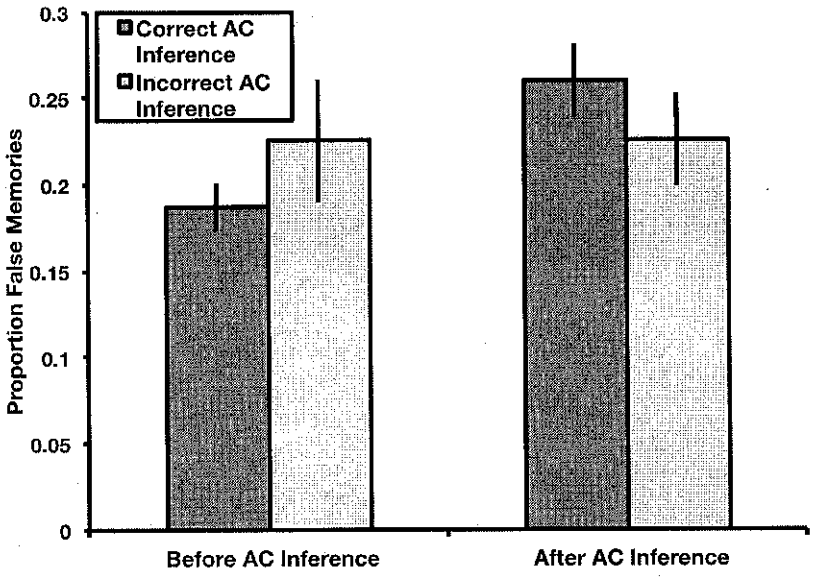

Figure 4. Proportion of false memories in Experiment 4. Performance on detail and source monitoring questions was examined both before and after either successful or unsuccessful inference. It is important that only trials for which participants responded correctly to directly learned trials were included in this analysis. Results revealed a significant time by inference interaction in Experiment 4. Subsequent *t* tests confirm that false memories selectively increased only following successful associative inference. Error bars represent ± 1 *SEM*.

0.02) and after unsuccessful inference retrieval (M_{after} = 0.18, SE = 0.02).

In summary, the results of Experiment 4 replicated the results of Experiment 1–3, while also providing additional evidence that testing of directly learned pairs during the associative inference test was not responsible for the increased false memory effects after compared with before successful associative inference. During the associative inference test for Experiment 4, participants were only tested on previously unpaired AC items (i.e., inference items) before the second set of detail and source questions. Thus, the increase in source memory errors following the associative inference test in Experiment 4 cannot be attributable to direct retrieval of previously studied pairs; rather, the current results support the role of recombination-related retrieval processes in subsequent source memory errors.

General Discussion

The four experiments reported here each provide evidence that flexible retrieval processes that support successful associative inference also produce increases in false memories that result from source misattributions: memory errors increased significantly *after* but not *before* successful compared to unsuccessful inferential retrieval. Experiments 1 and 2 provided evidence that flexible retrieval processes required for successful associative inference also produce increases in source misattributions when the detail/source memory test immediately followed the test of directly learned and associative inference items, whereas Experiment 3 revealed that these effects persisted across a 30-min delay between the associative inference tests and the second source memory test. Experiment 4 revealed the same significant increase in source memory error after but not before successful compared to unsuccessful inferential retrieval as observed in Experiment 1–3 even when directly learned associations were not tested until participants completed all of the detail and source monitoring questions. Furthermore, across all four experiments results revealed that both foil memory and correct memory scores showed no relationship to correct compared with incorrect inference either before or after the directly learned and associative inference test, thereby indicating that the observed effects are specific to the misattribution of previously experienced details to the related event rather than to a general decrease of detail with which the original event was remembered. Thus, the results of all four experiments provide direct evidence supporting the role of flexible retrieval and recombination processes in both successful associative inference and subsequent source memory error. These data thus provide, for the first time, direct experimental support for a key claim of the constructive episodic simulation hypothesis (Schacter & Addis, 2007a, 2007b), namely that the same flexible recombination process that supports an adaptive cognitive process can also increase memory errors that result from combining elements of distinct episodes. More generally, our results add to the mounting evidence that certain kinds of memory errors result from the operation of adaptive constructive processes that are linked to beneficial effects (for reviews, see Howe, 2011; Howe et al., 2016; Newman & Lindsay, 2009; Schacter, 2012; Schacter et al., 2011).

As noted in the Introduction, previous research (cf., Shohamy & Wagner, 2008; Zeithamova & Preston, 2010) indicates that successful associative inference in the AB, BC paradigm used here can result from flexible retrieval and/or integrative encoding (i.e., during study of BC, participants recall the related AC pair and encode an integrated representation (ABC) that they later retrieve

on the associative inference test). If integrative encoding contributes to false memories in our paradigm, then there should be more false memories for successful than unsuccessful inference trials *before* the associative inference test, but such effects were observed only *after* the associative inference test. Note, however, that previous research suggests that integrative encoding primarily supports associative inference when learning occurs across multiple repetitions, by affording multiple opportunities for cross-episode binding (Shohamy & Wagner, 2008; Zeithamova & Preston, 2010). By contrast, our experimental design utilized a single-trial learning paradigm that elicits an additional recombination mechanism during successful inference retrieval (Zeithamova & Preston, 2010). It is thus possible that when there are multiple repetitions during the learning phase, or under other experimental conditions that heighten the contribution of integrative encoding to associative inference, integrative encoding processes contribute to the type of source memory errors observed here. Thus, while the present data provide evidence for a link between flexible retrieval and false memories, they by no means rule out a similar link to integrative encoding under a different set of experimental parameters that are more likely to elicit successful associative inference as a result of integration during encoding. Future research should aim to examine the role of integration during encoding on subsequent source memory error.

Why does successful inferential retrieval result in heightened susceptibility to source memory errors? We suggest that the effects that we have documented here reflect the joint operation of two related but distinct mechanisms: cross-episode binding (e.g., Bridge & Voss, 2014a, 2014b) and retrieval-based reactivation and recombination (e.g., Bridge & Voss, 2014a, 2014b; Hupbach, Gomez, Hardt, & Nadel, 2007; St Jacques & Schacter, 2013). Binding processes that link disparate elements of an episode into a unified representation have been extensively studied in recent years, and have been linked closely to the operation of the hippocampus (e.g., Eichenbaum & Cohen, 2001; Hannula & Ranganath, 2008; Shimamura, 2010). As Bridge and Voss (2014b) point out, however, most such studies have focused on binding of elements *within* an episode. Bridge and Voss (2014b) studied *cross-episode* binding processes, and provided evidence that participants sometimes bind elements from distinct episodes (e.g., a face from one episode and a scene from another), resulting in memory error (for additional evidence linking binding processes to memory distortions, see Lew & Howe, 2016). We suggest that such cross-episode binding in our paradigm occurs most often and most extensively for episodes that result in successful, as opposed to unsuccessful, associative inference. That is, when people make a correct inference about the relationship between elements of events that have not been experienced together previously (i.e., AC), they may more fully bind details from the two episodes, such that details from one episode (AB) migrate to and become incorporated in the overlapping (BC) episode.

However, this binding account alone cannot explain the key finding from our experiments that increased false memories were observed for successful compared to unsuccessful inference trials only when the detail/source memory test was given *after* the associative inference test, and it is this finding that has led us to implicate a role for flexible retrieval and recombination processes in increased source memory errors. These observations fit well with prior findings that reactivating or retrieving memories can be a potent source of memory distortion if novel information is incorporated into a memory during the retrieval process (e.g., Chan, Thomas, & Bulevich, 2009; Gershman, Schapiro, Hupbach, & Norman, 2013; Gordon, Thomas, & Bulevich, 2015; Hupbach et al., 2007; Hupbach, Gomez, & Nadel, 2011; St Jacques, Olm, & Schacter, 2013; St Jacques & Schacter, 2013), possibly related to processes associated with memory reconsolidation that render a memory labile and prone to distortion during retrieval (Chan & LaPaglia, 2013; Dudai, 2012; Hardt, Einarsson, & Nader, 2010). From this perspective, in our experimental paradigm source memory errors arise when overlapping AB and BC relationships (along with their corresponding contextual details) are reactivated and flexibly recombined in order to encode the novel inference between the previously unrelated A and C items. Indeed, and consistent with our results, Bridge and Voss (2014b) only observed evidence for memory distortion associated with cross-episode binding following an active (vs. passive) retrieval condition. In line with the current results, retrieval-related recombination may thus result in heightened rates of source memory error following successful compared to unsuccessful inference because inferring the relationship between the nonoverlapping A and C items requires both a) reactivating distinct AB and BC episodes and b) flexibly recombining the nonoverlapping A and C items—during which contextual details from the AB episode are more fully bound to the BC episode and visa versa—resulting in memory distortions associated with cross-episode binding as a consequence of flexible retrieval and recombination processes. An important task for future research is to explore and clarify exactly how the recombination process supporting successful inference produces such erroneous memories. While previous evidence supports the idea that memory errors can result from erroneously combining details of individual episodic or autobiographical memories (e.g., Burt et al., 2004; Devitt et al., 2016; Odegard & Lampinen, 2004), the present studies provide novel evidence that the same flexible recombination mechanism that supports an adaptive cognitive process, such as associative inference, also increases subsequent memory errors.

Although we are not aware of any prior studies that have specifically linked successful associative inference with memory errors, as noted earlier previous research has linked memory reactivation processes with source misattributions and related kinds of memory errors. The studies noted earlier by Bridge and Voss (2014a, 2014b) suggest that simply coactivating memories during retrieval can lead to source misattributions, wherein coactivation of existing memory traces produces cross-episode binding of peripheral features from each episode. Although these results are consistent with the results reported here, it is unlikely that simple coactivation of elements from different episodes is sufficient to account for our key results. Our data speak against a simple coactivation hypothesis specifically because only trials for which participants were able to successfully reactivate both AB and BC episodes (as assessed by the test for directly learned associations) were used in the false memory analyses. Thus, both AB and BC events should have been successfully reactivated during the inference test. Accordingly, if coactivation of AB and BC events accounted for the increase in source memory error, we would not expect to see a significant difference between successful inference and unsuccessful inference after the associative inference test.

Because we observed such a difference, we suggest that successful inference requires an additional retrieval-related recombination process that results in increased source memory error. Indeed, in each of our experiments we observed significantly longer RTs on associative inference trials than on directly learned trials, which is in line with the arguments of Zeithamova and Preston (2010), suggesting that there is an additional retrieval mechanism necessary for inferential versus direct retrieval following single-trial learning. Coactivation of memories at test clearly can lead to source misattributions (Bridge & Voss, 2014a, 2014b), and it may be a contributing factor and perhaps even a necessary condition for increased source memory errors in the current paradigm. Nonetheless, coactivation of elements from distinct episodes during retrieval does not appear to a sufficient condition for producing the increase in source misattributions in the current paradigm. Alternatively, coactivation may have an effect during encoding such that participants bring to mind overlapping AB pairs during BC encoding thereby linking the two related events. However, if this were the case we would expect to see elevated source memory error before successful inference, which was not the case in our experiments, as we emphasized in the discussion of integrative encoding.

Although we have emphasized throughout the distinction between integrative encoding and flexible retrieval, and provided in the Introduction explicit predictions regarding outcomes that distinguish between these processes, it is important to emphasize we are not advocating that a simple encoding-retrieval dichotomy can account for the results observed here. Students of memory have long recognized that that encoding processes involve retrieval and vice versa. With respect to the present paradigm, integrative encoding requires some amount of retrieval (i.e., during study of BC, participants retrieve an overlapping AB pair to encode an integrated ABC representation), and flexible retrieval results in some degree of encoding (i.e., cross-episode binding). Nonetheless, the pattern of results observed here indicates a sharp difference in patterns of false memory before and after the associative inference test, which we have attempted to characterize in terms of the joint operation of cross-episode binding and flexible retrieval processes.

We have emphasized throughout that the current results fit well with an emerging theoretical picture in which various kinds of memory distortions are viewed as products of adaptive constructive processes (Schacter, 2012) that serve a range of cognitive functions, including simulating future experiences (Dudai & Carruthers, 2005; Schacter & Addis, 2007a, 2007b; Suddendorf & Corballis, 2007), solving problems (Howe, Garner, Charlesworth, & Knott, 2011), memory updating (Hardt et al., 2010; Hupbach et al., 2007, 2011; St Jacques et al., 2013), and extracting gist or meaning (Brainerd & Reyna, 1990; Koutstaal, 2006; Schacter, 2001; for recent reviews, see Howe, 2011; Howe et al., 2016; Newman & Lindsay, 2009; Schacter et al., 2011; Schlichting & Preston, 2015). Here we have focused on associative inference, which serves the adaptive function of allowing us to make new connections, and decisions about novel situations, based on flexibly retrieving and recombining information acquired in distinct though related prior experiences (Zeithamova, Schlichting, & Preston, 2012). Neuroimaging studies have linked the retrieval-based recombination process that supports associative inference to hippocampal function (Zeithamova & Preston, 2010; Zeithamova, Dominick, & Preston, 2012), but the nature of hippocampal contributions to the kinds of false memories associated with successful inference is unknown. Future research aimed at delineating the neural basis of the costs and benefits associated with flexible retrieval and recombination would enhance our understanding of the nature and functions of episodic memory.

References

Alba, J. W., & Hasher, L. (1983). Is memory schematic? *Psychological Bulletin, 93,* 203–231. http://dx.doi.org/10.1037/0033-2909.93.2.203

Bartlett, F. C. (1932). *Remembering: A study in experimental and social psychology.* New York, NY: Cambridge University Press.

Brainerd, C. J., & Reyna, V. F. (1990). Gist is the grist: Fuzzy-trace theory and the new intuitionism. *Developmental Review, 10,* 3–47. http://dx.doi.org/10.1016/0273-2297(90)90003-M

Brainerd, C. J., & Reyna, V. F. (2005). *The science of false memory.* New York, NY: Oxford University Press. http://dx.doi.org/10.1093/acprof:oso/9780195154054.001.0001

Bransford, J. D., Barclay, J. R., & Franks, J. J. (1972). Sentence memory: A constructive versus interpretive approach. *Cognitive Psychology, 3,* 193–209. http://dx.doi.org/10.1016/0010-0285(72)90003-5

Bransford, J. D., & Franks, J. J. (1971). The abstraction of linguistic ideas. *Cognitive Psychology, 2,* 331–350. http://dx.doi.org/10.1016/0010-0285(71)90019-3

Bridge, D. J., & Voss, J. L. (2014a). Hippocampal binding of novel information with dominant memory traces can support both memory stability and change. *The Journal of Neuroscience, 34,* 2203–2213. http://dx.doi.org/10.1523/JNEUROSCI.3819-13.2014

Bridge, D. J., & Voss, J. L. (2014b). Active retrieval facilitates across-episode binding by modulating the content of memory. *Neuropsychologia, 63,* 154–164. http://dx.doi.org/10.1016/j.neuropsychologia.2014.08.024

Burt, C. D. B., Kemp, S., & Conway, M. (2004). Memory for true and false autobiographical event descriptions. *Memory, 12,* 545–552. http://dx.doi.org/10.1080/09658210344000071

Chan, J. C. K., & LaPaglia, J. A. (2013). Impairing existing declarative memory in humans by disrupting reconsolidation. *Proceedings of the National Academy of Sciences of the United States of America, 110,* 9309–9313. http://dx.doi.org/10.1073/pnas.1218472110

Chan, J. C. K., Thomas, A. K., & Bulevich, J. B. (2009). Recalling a witnessed event increases eyewitness suggestibility: The reversed testing effect. *Psychological Science, 20,* 66–73. http://dx.doi.org/10.1111/j.1467-9280.2008.02245.x

Devitt, A. L., Monk-Fromont, E., Schacter, D. L., & Addis, D. R. (2016). Factors that influence the generation of autobiographical memory conjunction errors. *Memory, 24,* 204–222. http://dx.doi.org/10.1080/09658211.2014.998680

Dudai, Y. (2012). The restless engram: Consolidations never end. *Annual Review of Neuroscience, 35,* 227–247. http://dx.doi.org/10.1146/annurev-neuro-062111-150500

Dudai, Y., & Carruthers, M. (2005). The Janus face of Mnemosyne. *Nature, 434,* 567. http://dx.doi.org/10.1038/434567a

Eichenbaum, H., & Cohen, N. J. (2001). *From conditioning to conscious recollection: Memory systems of the brain.* New York, NY: Oxford University Press.

Gallo, D. A. (2006). *Associative illusions of memory: False memory research in DRM and related tasks.* New York, NY: Psychology Press.

Gershman, S. J., Schapiro, A. C., Hupbach, A., & Norman, K. A. (2013). Neural context reinstatement predicts memory misattribution. *The Journal of Neuroscience, 33,* 8590–8595. http://dx.doi.org/10.1523/JNEUROSCI.0096-13.2013

Gordon, L. T., Thomas, A. K., & Bulevich, J. B. (2015). Looking for answers in all the wrong places: How testing facilitates learning of misinformation. *Journal of Memory and Language, 83,* 140–151. http://dx.doi.org/10.1016/j.jml.2015.03.007

Hannula, D. E., & Ranganath, C. (2008). Medial temporal lobe activity predicts successful relational memory binding. *The Journal of Neuroscience, 28,* 116–124. http://dx.doi.org/10.1523/JNEUROSCI.3086-07.2008

Hardt, O., Einarsson, E. O., & Nader, K. (2010). A bridge over troubled water: Reconsolidation as a link between cognitive and neuroscientific memory research traditions. *Annual Review of Psychology, 61,* 141–167. http://dx.doi.org/10.1146/annurev.psych.093008.100455

Howe, M. L. (2011). The adaptive nature of memory and its illusions. *Current Directions in Psychological Science, 20,* 312–315. http://dx.doi.org/10.1177/0963721411416571

Howe, M. L., Garner, S. R., Charlesworth, M., & Knott, L. (2011). A brighter side to memory illusions: False memories prime children's and adults' insight-based problem solving. *Journal of Experimental Child Psychology, 108,* 383–393. http://dx.doi.org/10.1016/j.jecp.2010.08.012

Howe, M. L., Wilkinson, S., Garner, S. R., & Ball, L. J. (2016). On the adaptive function of children's and adults' false memories. *Memory, 24,* 1062–1077. http://dx.doi.org/10.1080/09658211.2015.1068335

Hupbach, A., Gomez, R., Hardt, O., & Nadel, L. (2007). Reconsolidation of episodic memories: A subtle reminder triggers integration of new information. *Learning & Memory, 14,* 47–53. http://dx.doi.org/10.1101/lm.365707

Hupbach, A., Gomez, R., & Nadel, L. (2011). Episodic memory updating: The role of context familiarity. *Psychonomic Bulletin & Review, 18,* 787–797. http://dx.doi.org/10.3758/s13423-011-0117-6

Koutstaal, W. (2006). Flexible remembering. *Psychonomic Bulletin & Review, 13,* 84–91. http://dx.doi.org/10.3758/BF03193817

Lew, A. R., & Howe, M. L. (2016). Out of place, out of mind: Schema-driven false memory effects for object-location bindings. *Journal of Experimental Psychology: Learning, Memory, and Cognition.* Advance online publication. http://dx.doi.org/10.1037/xlm0000317

Loftus, E. F. (2003). Make-believe memories. *American Psychologist, 58,* 867–873. http://dx.doi.org/10.1037/0003-066X.58.11.867

Loftus, E. F. (2005). Planting misinformation in the human mind: A 30-year investigation of the malleability of memory. *Learning & Memory, 12,* 361–366. http://dx.doi.org/10.1101/lm.94705

Loftus, E. F., Miller, D. G., & Burns, H. J. (1978). Semantic integration of verbal information into a visual memory. *Journal of Experimental Psychology: Human Learning and Memory, 4,* 19–31. http://dx.doi.org/10.1037/0278-7393.4.1.19

Madore, K. P., Addis, D. R., & Schacter, D. L. (2015). Creativity and memory: Effects of an episodic-specificity induction on divergent thinking. *Psychological Science, 26,* 1461–1468. http://dx.doi.org/10.1177/0956797615591863

Madore, K. P., & Schacter, D. L. (2014). An episodic specificity induction enhances means-end problem solving in young and older adults. *Psychology and Aging, 29,* 913–924. http://dx.doi.org/10.1037/a0038209

McClelland, J. L. (1995). Constructive memory and memory distortions: A parallel-distributed processing approach. In D. L. Schacter (Ed.), *Memory distortion* (pp. 69–90). Cambridge, MA: Harvard University Press.

Newman, E. J., & Lindsay, S. D. (2009). False memories: What the hell are they for? *Applied Cognitive Psychology, 23,* 1105–1121. http://dx.doi.org/10.1002/acp.1613

Odegard, T. N., & Lampinen, J. M. (2004). Memory conjunction errors for autobiographical events: More than just familiarity. *Memory, 12,* 288–300. http://dx.doi.org/10.1080/09658210244000621

Okado, Y., & Stark, C. E. L. (2005). Neural activity during encoding predicts false memories created by misinformation. *Learning & Memory, 12,* 3–11. http://dx.doi.org/10.1101/lm.87605

Preston, A. R., Shrager, Y., Dudukovic, N. M., & Gabrieli, J. D. E. (2004). Hippocampal contribution to the novel use of relational information in declarative memory. *Hippocampus, 14,* 148–152. http://dx.doi.org/10.1002/hipo.20009

Roediger, H. L., III. (1996). Memory illusions. *Journal of Memory and Language, 35,* 76–100. http://dx.doi.org/10.1006/jmla.1996.0005

Roediger, H. L., & McDermott, K. B. (1995). Creating false memories: Remembering words not presented in lists. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 21,* 803–814. http://dx.doi.org/10.1037/0278-7393.21.4.803

Schacter, D. L. (1996). *Searching for memory: The brain, the mind, and the past.* New York, NY: Basic Books.

Schacter, D. L. (2001). *The seven sins of memory: How the mind forgets and remembers.* New York, NY: Houghton Mifflin.

Schacter, D. L. (2012). Adaptive constructive processes and the future of memory. *American Psychologist, 67,* 603–613. http://dx.doi.org/10.1037/a0029869

Schacter, D. L., & Addis, D. R. (2007a). The cognitive neuroscience of constructive memory: Remembering the past and imagining the future. *Philosophical Transactions of the Royal Society of London Series B, Biological Sciences, 362,* 773–786. http://dx.doi.org/10.1098/rstb.2007.2087

Schacter, D. L., & Addis, D. R. (2007b). Constructive memory: The ghosts of past and future. *Nature, 445,* 27. http://dx.doi.org/10.1038/445027a

Schacter, D. L., Addis, D. R., Hassabis, D., Martin, V. C., Spreng, R. N., & Szpunar, K. K. (2012). The future of memory: Remembering, imagining, and the brain. *Neuron, 76,* 677–694. http://dx.doi.org/10.1016/j.neuron.2012.11.001

Schacter, D. L., Guerin, S. A., & St Jacques, P. L. (2011). Memory distortion: An adaptive perspective. *Trends in Cognitive Sciences, 15,* 467–474. http://dx.doi.org/10.1016/j.tics.2011.08.004

Schlichting, M. L., & Preston, A. R. (2015). Memory integration: Neural mechanisms and implications for behavior. *Current Opinion in Behavioral Sciences, 1,* 1–8. http://dx.doi.org/10.1016/j.cobeha.2014.07.005

Shaw, J., & Porter, S. (2015). Constructing rich false memories of committing crime. *Psychological Science, 26,* 291–301. http://dx.doi.org/10.1177/0956797614562862

Sheldon, S., McAndrews, M. P., & Moscovitch, M. (2011). Episodic memory processes mediated by the medial temporal lobes contribute to open-ended problem solving. *Neuropsychologia, 49,* 2439–2447. http://dx.doi.org/10.1016/j.neuropsychologia.2011.04.021

Shimamura, A. P. (2010). Hierarchical relational binding in the medial temporal lobe: The strong get stronger. *Hippocampus, 20,* 1206–1216. http://dx.doi.org/10.1002/hipo.20856

Shohamy, D., & Wagner, A. D. (2008). Integrating memories in the human brain: Hippocampal-midbrain encoding of overlapping events. *Neuron, 60,* 378–389. http://dx.doi.org/10.1016/j.neuron.2008.09.023

St Jacques, P. L., Olm, C., & Schacter, D. L. (2013). Neural mechanisms of reactivation-induced updating that enhance and distort memory. *Proceedings of the National Academy of Sciences of the United States of America, 110,* 19671–19678. http://dx.doi.org/10.1073/pnas.1316930110

St Jacques, P. L., & Schacter, D. L. (2013). Modifying memory: Selectively enhancing and updating personal memories for a museum tour by reactivating them. *Psychological Science, 24,* 537–543. http://dx.doi.org/10.1177/0956797612457377

Suddendorf, T., & Corballis, M. C. (2007). The evolution of foresight: What is mental time travel, and is it unique to humans? *Behavioral and Brain Sciences, 30,* 299–313. http://dx.doi.org/10.1017/S0140525X07001975

Szpunar, K. K. (2010). Episodic future thought: An emerging concept. *Perspectives on Psychological Science, 5,* 142–162. http://dx.doi.org/10.1177/1745691610362350

Tulving, E. (2002). Episodic memory: From mind to brain. *Annual Review of Psychology, 53,* 1–25. http://dx.doi.org/10.1146/annurev.psych.53.100901.135114

Zeithamova, D., Dominick, A. L., & Preston, A. R. (2012). Hippocampal and ventral medial prefrontal activation during retrieval-mediated learning supports novel inference. *Neuron, 75,* 168–179. http://dx.doi.org/10.1016/j.neuron.2012.05.010

Zeithamova, D., & Preston, A. R. (2010). Flexible memories: Differential roles for medial temporal lobe and prefrontal cortex in cross-episode binding. *The Journal of Neuroscience, 30,* 14676–14684. http://dx.doi.org/10.1523/JNEUROSCI.3250-10.2010

Zeithamova, D., Schlichting, M. L., & Preston, A. R. (2012). The hippocampus and inferential reasoning: Building memories to navigate future decisions. *Frontiers in Human Neuroscience, 6,* 70. http://dx.doi.org/10.3389/fnhum.2012.00070

Received June 2, 2016
Revision received August 9, 2016
Accepted August 14, 2016 ■

Assignment 3

Prior to beginning this assignment, please review all the required readings from the first three weeks as well as the articles you used in your Week 2 Discipline-Based Literature Review. During this course, you have been developing your knowledge in the area of learning and cognition. In the Week 2 assignment, there was special focus on six topics that influence a wide variety of disciplines in psychology and other fields. In the final week of this course, you will develop a Learning and Cognition Handbook based on these topics.

This week, you will write an extensive review and annotated bibliography on one of the six main course topics below:

- Traditional learning theories: Operant and classical conditioning
- Traditional learning theories: Behaviorism and social learning theory
- Attention and memory
- Decision-Making
- Language acquisition
- Organizational and lifelong learning

The topic you choose should be based on the area in which you would most like to develop your knowledge. Your choice should also consider your current interests in psychology and support your future career goals.

As you prepare this assignment, keep in mind that it is designed to assist you with beginning the process of drafting your Learning and Cognition Handbook, which is due in Week 6. It is recommended that your paper be checked in Grammarly (Links to an external site.) and through Turnitin (Links to an external site.) prior to submission.

Include the following components in your review:

Introduction: Explain your motivation focusing on your chosen topic and describe how this topic aligns with your future career goals.

Research: Research five Scholarly, Peer-Reviewed, and Other Credible Sources (Links to an external site.) in the Ashford University Library focusing on your chosen topic, and provide an annotated bibliography (Links to an external site.). These articles must provide sufficient information so that they will support your work in the Learning and Cognition Handbook. (See the instructions in Week 6 for further clarification.) Provide a complete reference for each of the five articles. Beneath each reference, provide an annotation that explains the theoretical perspectives, historical trends, and/or empirical research within the article that describe and define your chosen construct.

Conclusion: Provide a concluding paragraph that synthesizes the cognitive learning principles and theories found within the articles as they relate to your chosen construct.

The Choosing Your Focus paper

- Must be three to five double-spaced pages in length and formatted according to APA style (Links to an external site.) as outlined in the Ashford Writing Center.

- Must include a title page with the following:
  - Title of paper
  - Student's name
  - Course name and number
  - Instructor's name
  - Date submitted
- Must begin with an introductory paragraph that clearly states the chosen construct.
- Must clearly discuss and explain the chosen construct with critical thought.
- Must include a Research section comprised of the Annotated Bibliography (Links to an external site.).
  - The Formatting Your References List (Links to an external site.) guide offers additional guidance on correctly formatting references for the annotated bibliography.
- Must end with a conclusion that synthesizes the cognitive learning principles and theories found within the articles as they relate to the chosen construct.
- Must use at least five peer-reviewed sources (one per topic).
  - The Scholarly, Peer Reviewed, and Other Credible Sources (Links to an external site.) table offers additional guidance on appropriate source types. If you have questions about whether a specific source is appropriate for this assignment, please contact your instructor. Your instructor has the final say about the appropriateness of a specific source for a particular assignment.

The Role of Cognition in Classical and Operant Conditioning

(Source 1)

Irving Kirsch
University of Connecticut

Steven Jay Lynn
Binghamton University

Michael Vigorito
Seton Hall University

Ralph R. Miller
Binghamton University

For the past 35 years, learning theorists have been providing models that depend on mental representations, even in their most simple, deterministic, and mechanistic approaches. Hence, cognitive involvement (typically thought of as expectancy) is assumed for most instances of classical and operant conditioning, with current theoretical differences concerning the level of cognition that is involved (e.g., simple association vs. rule learning), rather than its presence. Nevertheless, many psychologists not in the mainstream of learning theory continue to think of *cognitive* and *conditioning* theories as rival families of hypotheses. In this article, the data pertaining to the role of higher-order cognition in conditioning is reviewed, and a theoretical synthesis is proposed that provides a role for both automatic and cognitively mediated processes. © 2004 Wiley Periodicals, Inc. J Clin Psychol 60: 369–392, 2004.

Keywords: classical conditioning; operant conditioning

Correspondence concerning this article should be addressed to: Steven Jay Lynn, Psychology Department, Binghamton University, State University of New York, P.O. Box 6000, Binghamton, NY 13902–6000; e-mail: slynn@binghamton.edu.

JOURNAL OF CLINICAL PSYCHOLOGY, Vol. 60(4), 369–392 (2004)

Published online in Wiley InterScience (www.interscience.wiley.com). DOI: 10.1002/jclp.10251

Classical conditioning used to be viewed as a type of learning that involves the acquisition of elicited responses (i.e., responses, like the defensive eye blink, that are preceded reliably by an identifiable eliciting stimulus and that are experienced phenomenologically as automatic or reflexive). Similarly, instrumental (operant) conditioning was regarded as a type of learning that involves the acquisition of emitted responses (i.e., responses, like a wink of the eye, that can occur in the absence of reliable or well-defined antecedent stimuli and are experienced as voluntary). An implicit assumption of these old definitions was that what is acquired is a stimulus–response (S–R) association rather than a belief about the antecedents of an outcome (O, i.e., an expectancy).

When operant and classical conditioning are defined narrowly as types of learning in which S–R associations are formed, one can question whether they exist at all. Thus, Brewer (1974) provocatively titled his review of the conditioning literature, *There is No Convincing Evidence for Operant or Classical Conditioning in Adult Humans*. Contemporary conditioning theorists regard instrumental and classical conditioning as procedures that lead to behavior change (see Bolles, 1979). One advantage of defining instrumental and classical conditioning empirically in this way is that it is theoretically neutral. There can be no doubt but that instrumental and classical conditioning procedures reliably lead to changes in behavior. What are at issue are the inferred processes by which these changes are produced. In contrast to the early views expressed above, most contemporary learning theorists, even those who are mechanistically minded, regard classical conditioning as reflecting S–O (a.k.a. S–S) associations and instrumental learning as reflecting R–O associations (Rescorla, 1988, 1991; also see Kirsch, 1985).

In what he deemed his most important use of the term, Kuhn (1970) characterized paradigms as exemplars of seemingly permanent solutions to particular scientific problems, exemplars that serve as models for subsequent research in the area. Classical and instrumental conditioning are paradigms in this sense of the term. They have provided models for study a variety of phenomena, including phenomena that are central to clinical psychology. Outstanding examples of the application of classical conditioning procedures to clinical concerns include their use to induce (Watson & Rayner, 1920) and treat (Jones, 1924; Wolpe, 1958) phobic anxiety. The application of operant conditioning to clinical phenomena is evident in the creation and elimination of behaviors that might be symptomatic of psychiatric disorders (Haughton & Ayllon, 1965) and in the operation of token economies (Allyon & Azrin, 1968). Similarly, the behavior of rats that have been trained in a Skinner box on a variable ratio schedule of reinforcement can serve as an exemplar for analyzing and studying the behavior of humans standing in front of slot machines in Las Vegas. In the latter case, even the topography is isomorphic, including the lever that is pressed and the cup, in which the reinforcing pellets or chips are delivered, underneath it. However, this topographical similarity is the exception rather than the rule.

Kuhn (1970) noted that scientists can "agree in their identification of a paradigm without agreeing on . . . a full interpretation of it" (p. 44). This particularly is clear with respect to classical and operant conditioning. There can be no doubt that these procedures result in learning and that they have inspired treatments that have been shown to be effective in clinical trials. However, almost since their inception, their interpretation was the focus of intense theoretical debate. The central issue at the core of this debate was the following question: Are these phenomena automatic, mechanistic processes, in which higher-order cognition, if present at all, is merely an epiphenomenon (e.g., Hull, 1943; Pavlov, 1927; Skinner, 1953; Watson, 1913), or are they processes that are mediated cognitively (e.g., Bolles, 1979; Rotter, 1954; Tolman, 1932, 1948)? In recent years, a consensus has emerged that cognitive processes play an important role in learning (Miller

& Oberling, 1998; Rescorla, 1988, 1991). Nevertheless, it will be argued that there are some instances in which learning bypasses entirely higher-order cognition.

Contemporary mechanistic accounts of classical and operant conditioning typically involve the hypothesis that direct associations between the stimulus and outcome representations or the response and outcome representations are formed during conditioning. Obviously the outcome representations activated by these associations constitute a low-level form of outcome expectancy. Thus, there is now virtually universal agreement that conditioning involves the production of expectancies. The remaining theoretical differences about this issue concern the level of cognition and whether these cognitions can be represented adequately as simple associations. Higher-order cognitive alternatives to these mechanistic interpretations have centered on the concept of expectancy as more than just the activation of simple binary associations. An expectancy is a future-oriented belief; it is a belief that something will happen. Therefore, they also have been described as subjective probabilities (Rotter, 1954). From a cognitive perspective (e.g., Kirsch, 1985), instrumental learning situations produce expectancies that particular behaviors will produce particular outcomes (e.g., that food can be found in a particular location), and classical conditioning produces expectancies that certain stimuli will be followed by other stimuli (e.g., that food will be presented soon after the bell is sounded).

Expectancies have been portrayed as S–O associations, but not all S–O associations are expectancies. For example, a particular melody might function as a cue stimulus evoking a representation of a person in one's past. However, this representation is not an expectancy because it does not produce the belief that the person will appear because there also are present contextual cues that tell us that the person will not appear. In addition, there are types of S–R associations that can be regarded as expectancies (Kirsch, 1985). These are associations in which what is invoked by the stimulus is a representation of the response rather than the response itself. In this article, old and relatively recent data pertaining to the role of higher-order cognition in conditioning will be reviewed, and a theoretical synthesis that provides a role for both automatic and cognitively mediated processes will be proposed.

Data Indicating Higher-Order Cognitive Mediation

Tolman's Challenge to Mechanistic Explanations of Learning

Following the ascendancy of behaviorism, mechanistic explanations were prevalent among learning theorists (e.g., Hull, 1943). The major exception was Tolman (1932, 1948), whose research program was aimed largely at providing anomalies for image-free mechanistic learning theories. Tolman and his colleagues produced a large body of data supporting the hypothesis that rats running in mazes behaved as if they had access to information, built cognitive maps of the mazes, and expected to find food in particular locations (Bolles, 1979). The nature of Tolman's challenge to image-free mechanistic theory can be illustrated with the following examples.

Vicarious Trial and Error: "Catching On." Tolman's (1939) studies of vicarious trial and error (VTE) supported his idea of cognitive maps. VTE refers to the behaviors of "hesitating, looking back and forth" that rats engage in at choice points in a maze or discrimination task (e.g., choosing whether food is behind a black or white door) or before going one way or another in a maze. Rats seemed to display more VTEs when they *catch on* to which stimuli to pay attention to in a visual discrimination task or later "make

sure of which stimulus is which." Tolman claimed this behavior indicates that the rat actively selects and compares stimuli in constructing a cognitive map of the task.

"Hypothesis" Experiments. Krech and Crutchfield (1948) defined learning as a "reorganization of the cognitive field" (p. 112). Tolman (1948) credited Krechecsky (Krechevsky, 1932) with designing experiments suggesting that rats develop systematic choices or *hypotheses* in progressing down difficult mazes. For example, rats try a variety of different behaviors, such as choosing right-handed or dark doors, which continues at above-chance levels until a solution to the maze or discrimination task is achieved. Such trial-and-error behavior was viewed as goal directed and was thought to reflect the development of tentative cognitive maps that are subject to revision as learning occurs.

"Place Learning." If a rat forms a cognitive map of a maze, then it should learn something about the relation among stimuli and have the ability to discriminate the *place* where food was located in its learning environment. Tolman, Ritchie, and Kalish (1946) found that rats that were trained to run a maze in a direct path for food, when blocked from running down the original path to the food and confronted with radiating paths, tended to run down the path that was in the direction of where the food was placed originally or selected a path that ran perpendicularly to the side of the room where the food was placed. Accordingly, the rats appeared to have learned the place where the reward was located, allowing the inference that they had formed a cognitive map of the maze.

In a direct test of the hypothesis that rats in mazes learn locations rather than responses, Tolman et al. (1946) alternately placed rats in one of two different start locations. Half of the rats (designated *place learners*) were reinforced for running to the same location, which required a different response (turning left or right) depending on which location they had started from. The others (designated *response learners*) were reinforced for making the same response, which took them to a different location depending on where they had started from. The logic of the study was that the place-learning task should be the easier of the two if rats learn locations, whereas the response-learning task should be easier if rats learn responses. In fact, all of the place learners learned their task within 8 trials, whereas after 72 trials, only 3 of 8 response learners had learned the task. Nevertheless, both tasks were learned by at least some of the subjects, making it clear that rats are opportunistic and are capable of either type of learning.

Response Prevention. Additional support for the cognitive map hypothesis comes from studies in which animals have been prevented from making a response for which they previously have been reinforced. In these studies, the use of an alternate response to reach the same reinforced location is interpreted as support of the cognitive hypothesis. For example, a rat prevented from turning right can traverse the right path of a T maze by turning left in a $\frac{3}{4}$ circle, until it is facing the goal box. Numerous studies have shown that learning occurred when responses were prevented in a variety of ways, including a) immobilizing rats by an administration of curare and testing learning when the drug was no longer active (Girden, 1942), b) crushing rats' motor nerves (Kellogg, Scott, Davis, & Wolf, 1940), and c) lesioning midbrain regions (Beck & Doty, 1957).

Latent Learning. The most controversial research paradigm was that purporting to demonstrate latent learning (e.g., Blodgett, 1929; Tolman & Honzik 1930). These experiments revealed that rats allowed to spend time in a maze without food reinforcement for reaching the goal showed little improvement in the time required to reach the goal or in

the number of errors made. However, immediately after food was dispensed, the rats' error curves "dropped astoundingly," indicating that the rats had learned to navigate the maze even when they were not reinforced for doing so, but that this learning was not expressed behaviorally in the absence of reinforcement. In other experiments, it was shown that rats learned to locate food even when they were satiated (Thistlewaite, 1951) and when exposure to the maze is provided by having the rat ride in a small cart (McNamara, Long, & Wilke, 1956). According to Tolman, these sorts of demonstrations implied the existence of cognitive maps that formed during nonreinforced trials.

Brewer's (1974) Review

In 1974, William Brewer reviewed more than 200 "dissociation" studies, which he claimed distinguished between "conditioning theory" and "cognitive theory" (i.e., between mechanistic and higher-order cognitive interpretations of conditioning). According to Brewer, these studies of autonomic responses, motor responses, and complex responses (e.g., semantic generalization, conditioned meaning, verbal operant conditioning) in humans provide strong support for a cognitive interpretation. Below are the types of studies he reviewed and the logic by which they support a higher-order cognitive interpretation of conditioning.

Informed pairing: Simply informing participants about the CS–US (i.e., S–O) relation, with no actual pairing, results in acquisition of the CR and informing participants of response–reinforcement (i.e., R–O) contingencies produces instrumental learning.

Informed unpairing: After operant or classical conditioning, extinction can be produced by informing participants that the contingencies are no longer in effect, without any actual extinction trials.

Instructed conditioning: Participants instructed to produce a CR in response to a CS or to emit an operant response do so, without any actual conditioning trials.

Instructed nonconditioning: Participants are told to not produce a CR or operant response, following which they are given conditioning trials. Cognitive theory is supported when the response is not emitted.

Instructed extinction: After standard operant or classical conditioning, participants told to stop emitting the learned response do so.

Masking: Misleading instructions can be used to mask CS–US or response–reinforcement relations.

Awareness of contingency: Awareness of contingencies can be assessed and often is found to be correlated with the emission of conditioned responses, the cognitive hypothesis being that they will be emitted only by participants who are aware.

Modified contingency expectancy: After conditioning, participants are provided with information that produces expectations about contingencies that are different from those of the conditioning trials. For example, removing the shock electrodes in an aversive conditioning paradigm should eliminate the expectancy of shock.

Response expectancies: After conditioning in situations in which different responses are possible, participants' hypotheses about the response are assessed and correlated with their CRs. Alternatively, participants' response expectancies can be manipulated by the provision of verbal information.

Reinforcement expectancy: Participants' responses in a classical conditioning paradigm are correlated with information they have been given about the intensity of a strongly aversive US, or their responses are correlated with their hypotheses about the purpose of an ambiguous reinforcer (e.g., a spoken "hmm").

Brewer (1974) interpreted the data from these experiments as indicating "all the results of the traditional conditioning literature are due to the operation of higher mental processes, as assumed in cognitive theory, and that there is not and never has been any convincing evidence for unconscious, automatic mechanisms in the conditioning of adult human beings" (p. 27). A number of later reviews (Boakes, 1989; Lovibond & Shanks, 2002; Shanks & St. John, 1994), focusing primarily on data reported after Brewer's (1974) review, have echoed Brewer's conclusions regarding the failure of research to support mechanistic views of conditioning. Other reviewers, however, have reached alternate interpretations of the data (e.g., see Manns, Clark, & Squire, 2002; Weins & Öhman, 2002, for contrary conclusions), especially regarding the hypothesis that learning requires awareness of the contingencies (Morris, Ohman, & Dolan, 1998; Schacter, 1987).

Rescorla's Reviews of Classical and Operant Conditioning

Two influential reviews by Robert Rescorla, first of classical conditioning (1988) and then of instrumental conditioning (Rescorla, 1991), constituted further challenges to simple mechanistic views of learning. Rescorla's (1988) review entitled, *Pavlovian conditioning: It's not what you think it is*, updated his earlier (1968) conceptualization of classical conditioning as involving the acquisition of information, as opposed to a "low level mechanical process in which the control over a response is passed reflexively from one stimulus to another" (Rescorla, 1988, p. 152). In his early experiments, Rescorla manipulated the contingency (i.e., correlation) between CSs and USs by presenting various combinations of CSs alone, USs alone, and contiguous CS–US pairings. What appeared to be important for the acquisition of conditioned responding was not the total number of contiguous pairings, but the overall relationship between the CS and US. According to this informational hypothesis, behavioral control is established when there is a positive or negative correlation between the CS and US, but not when there is no correlation. Accordingly, conditioned responses are elicited when the CS predicts that the US is likely to occur, but inhibited when the CS predicts that the US is less likely than the USs base rate. It was as if the animals were attuned to the informational value of the CS that established relations among events, just as is predicted by modern conditioning theories, in which cognitions (and expectancies in particular) play a central role.

According to this perspective, animals (including humans) are goal-directed, active information seekers who form rich and varied representations of their environment in the course of responding to an array of stimuli that come to be associated with one another in potentially complex ways. Conditioning is responsive to different properties of the stimuli that organisms encounter, to differences in associability among stimuli, and to the signaling properties of stimuli with respect to the relations that exist among other stimuli. Learning occurs when organisms are *surprised* (Kamin, 1968; Rescorla & Wagner, 1972) and modify their Pavlovian associations in response to the "discrepancy between the actual state of the world and the organism's representation of that state" (Rescorla, 1988; p. 153). Outcomes that are *surprising* provide new information, facilitate a rich representation of the world, and permit conditioning in as few as a single trial. This line of theorizing allows Tolman's concept of expectancy to be applied readily to contemporary associative learning theory. However, this insight did not result in an unquestioned triumph of higher-order cognition. Notably, simple expectancies can be captured in mechanistic formulations such as the Rescorla–Wagner model (e.g., Allan, 1993). Here subjects are assumed insensitive to direct correlations between cues and outcomes, but the asymptotic state of their mechanistic S–O formulation is difficult to distinguish from such a

sensitivity. Moreover, at their current states of development, simple mechanistic models do some things (e.g., predict recency effects and cue competition) better than do higher-order cognitive models (e.g., López, Shanks, Almaraz, & Fernández, 1998). However, as each family of models evolves, it can be seen that there is little that each family (simple mechanism or higher-order cognition) cannot explain in principle. Contrasting families of models, rather than contrasting two specific models, is dangerous because improved models within each family are often in preparation (Miller & Escobar, 2001). Nevertheless, contrasting cognitive and mechanistic approaches encourages model development and highlights critical phenomena.

S–O Associations in Classical Conditioning

Historically (e.g., Hull, 1943), the associations that are learned during conditioning are between stimuli and responses. Thus, they are S–R associations. In simple cognitive theories (including virtually all contemporary conditioning theories), associations are hypothesized to be formed between representations of two stimuli (i.e., S–O associations), between representations of stimuli and responses (S–R), or between representations of responses and outcomes (R–O). S–O (or R–O) associations generally are interpreted as cognitions in which a stimulus (or response) comes to elicit an expectancy for the occurrence of another stimulus. Contemporary research suggests that expectancies in classical conditioning involve associations that vary in complexity. Simple S–O associations are inferred from conditioning procedures that result in conditioned responses following trial-by-trial presentation of a CS in some relationship with the US. More complex associations are inferred from procedures that provide the opportunity to combine two simple S–O associations (e.g., sensory preconditioning or second-order conditioning), and even greater complexity in associative structure is inferred from procedures that allow for the encoding for higher-order relationships (e.g., occasion setting; Miller & Oberling, 1998).

Simple S–O Associations. Credible evidence of S–O associations in classical conditioning is provided by experiments using *sensory preconditioning procedures* (e.g., Rizley & Rescorla, 1972). In this procedure, animals are presented first with contiguous pairings of two neutral stimuli such as a light and a tone. During this initial phase of the procedure, there is no behavioral evidence that the animals associate the two stimuli. Next, only one of the neutral stimuli, for example the tone, is paired with a US until it elicits a CR. When the stimulus that was not paired with a US (the light in this example) is presented alone in the final test phase, it too is found to elicit a CR. Note that because the light was never paired with a US, it was never experienced as contiguous with a response; therefore, it could not have gained eliciting properties as a result of an S–R association. The generally accepted explanation of the sensory-preconditioning phenomenon is that the animals acquire a latent tone–light association in the first phase and a tone–US association in the second phase. When the light is presented in the final test phase, the light evokes a mental representation of the tone, which in turn evokes an expectancy of the US and generates a CR. Although sensory preconditioning procedures provide strong support for simple S–O associations, they also show more complex cognitive processes. That is, these results indicate that rats are able to take two separate S–O associations (Light–Tone and Tone–US) and through a transitive inference process infer a third association (Light–US).

Additional evidence for this simple cognitive account of classical conditioning comes from *US devaluation procedures* (e.g., Holland & Rescorla, 1975). In the first phase,

experimental and control groups are given standard CS–US pairings until CRs are observed. According to early mechanistic views of conditioning, Phase-1 conditioning reflects an S–R (CS–UR) association, but, according to both contemporary mechanistic and simple cognitive accounts, conditioning results in an S–O (CS–US) association, which can be thought of as a mental link between the representations of the stimulus events. To differentiate between the S–R and S–O accounts of conditioning, the value of the US is reduced (devalued) for the experimental group, but not for the control group during Phase 2 of the experiment. For example, in procedures in which the US is food, satiating the animal or conditioning a taste aversion to the US can achieve devaluation. The S–R account predicts that devaluing the US should have no effect on the CSs subsequent ability to elicit CRs because the US is not part of the association that controls conditioned responding. The S–O account, however, includes a *forward-looking* association and a mental representation of the anticipated US. The experimental group, therefore, should expect a devalued US and thereby respond less to the CS relative to the control group. Devaluation experiments involving classical conditioning paradigms as diverse as sexual-approach conditioning in birds (Holloway & Domjan, 1993) and food conditioning in rats (e.g., Holland & Rescorla, 1975) have provided evidence of S–O associations.

A wide range of simple classical conditioning procedures result in robust and reliable conditioned responding. This observation suggests the even simple associative learning may vary in complexity. In most conditioning procedures, the CS onset precedes US onset, but this temporal precedence is not necessary for conditioning to be seen. Conditioned responses occur when a CS is presented simultaneously with, or following, an aversive US in just a few trials, and sometimes after just one trial (e.g., Ayres, Haddad, & Albert, 1987; Mahoney & Ayres, 1976). Conditioning with just one CS–US pairing indicates that temporal contiguity is a sufficient condition for the establishment of a simple association between internal representations of a CS and US. Additional experience with repeated CS–US trials, however, provides the opportunity for the subject to obtain more information through contingency learning, information that may result in new learning involving the causal (Rescorla, 1988) and temporal (Miller & Barnet, 1993) relationship between stimuli or the behavioral expression of that information (Miller & Matzel, 1988).

Regarding variation in the behavioral expression of simple associations, expectancy theory does not have much to contribute. For instance, expectancy theory does not account for the form that the conditioned response takes in conditioning procedures. A number of studies indicate that the form of the conditioned response is influenced by the type of CS used. For example, Holland (1977) found that rats exposed to tone–food pairings developed a head jerk to the tone, whereas rats exposed to otherwise identical light–food pairings exhibited rearing behavior to the light. In addition, the length of the interval between the CS and the US can affect response topology in ways that are not always consistent with *rational* expectancies. With a short tone–food interval, Holland (1980) observed a startle response to the tone, whereas with a long tone–food interval, he saw orientation to the food hopper in response to the tone. Results like these support the view that the activation of conditioned responding involves biologically *preprogramed* (mechanistic) behaviors that are organized around important biological functions (e.g., feeding, mating, and defense) and are elicited by stimuli that anticipate the arrival of a US (Timberlake & Lucas, 1989). Moreover, recent studies have demonstrated that an abstraction such as response variability can be reinforced (e.g., Neuringer, 2002). Expectancy accounts of simple associative learning also fail to explain basic learning phenomenon that point to failures of behavioral expression rather than to failure to form an S–O association. One example is the *US preexposure effect*. Repeated exposure to a US retards the course of classical conditioning later when a CS is paired with the US in the same context. The US

preexposure effect apparently occurs when the context accrues sufficient excitatory strength to block conditioning to the discrete CS (Randich & LoLordo, 1979). This result usually is interpreted as reflecting the failure of a subject to acquire a CS–US association because the added CS was redundant and provided no new information. However, Matzel, Brown, and Miller (1987) demonstrated that unreinforced exposure to the context after CS–US training reduced the US preexposure effect, suggesting that the CS–US associations indeed were formed during the training phase, but was not expressed in behavior.

Higher-Order Associations. Although the context can serve as a CS in a CS–US association when the context is the best predictor of the US, it also can enter a higher-order relationship with a CS–US relationship when the latter is embedded within the context. In one study, Bouton and King (1983) found that when a discrete CS was paired with a shock US in one context and extinguished in another context, the subjects showed conditioned fear of the discrete CS when retested in the original training context. This renewal of conditioned fear was not evident in subjects that experienced training and extinction in the same context. Thus, expectancies can be altered by the contexts in which test trials occur. Furthermore, independent assessment of the ability of the context to elicit conditioned responding indicated that demonstrable excitatory and inhibitory conditioning of the context was not necessary for the context to control fear to the discrete CS. These results were interpreted as supporting the role of the context as an occasion setter. Occasion setters are viewed as stimuli that provide information about when a CS–US contingency is in effect (Schmajuk & Holland, 1998). Occasion setters are not restricted to contextual stimuli, but can occur in other procedures that provide two-level hierarchical arrangements of discrete events. For example, in the *serial feature–positive discrimination procedure*, a discrete stimulus (the feature) precedes another discrete stimulus (the CS) when the latter is paired with a US, but not when it is presented without a US. Evidence suggests that the discrete CS enters a simple association with the US, but its expression is dependent upon a conditional cue function of the feature. Interestingly, when the feature is presented simultaneously with the other CS instead of preceding it, simple associations appear to develop between the feature and the US rather than higher-order conditional associations (Ross & Holland, 1981). Moreover, just as occasion setters can disambiguate otherwise ambiguous CSs, so too can higher-order occasion setters disambiguate otherwise ambiguous first-order occasion setters (Arnold, Grahame, & Miller, 1991).

R–O and S–(R–O) Associations in Operant Conditioning

R–O Associations. Traditional cognitive explanations of behavioral change in instrumental conditioning procedures posit the formation of an association between an emitted response and an outcome that has followed it in the past (R–O; Bolles, 1979). These R–O associations are the basis of outcome expectancies, that is, they support expectancies that an emitted response will lead to a particular outcome. Devaluation procedures, from which evidence of S–O associations in classical conditioning was derived, also have been adapted for instrumental conditioning procedures, in which they provide evidence of R–O associations. In these instrumental learning studies, an outcome (e.g., sucrose solution or food pellets) can be devalued by pairing it with the administration of a substance or toxin that creates a food aversion, for example. In one study using simple schedules of reinforcement (i.e., a single manipulandum), rats that learned to press a lever for food on a ratio schedule of reinforcement (Phase 1) and then experienced a devaluation of the reinforcer outcome in the home cage (Phase 2) responded less on the lever in a

subsequent extinction test (Phase 3) compared to rats that did not experience the devaluation (Dickinson, Nicholas, & Adams, 1983). Rescorla and colleagues have used concurrent schedules of reinforcement such that in the first phase rats were trained to emit one response for one type of outcome (e.g., food pellets) and a second response for another outcome (e.g., sucrose) followed by home–cage devaluation of one of the outcomes. These studies also have demonstrated consistently a reduction in responding for the devalued outcome during the test phase (e.g., Colwill & Rescorla, 1985a; 1985b). Such results are not predicted by an S–R mechanistic account of conditioning because the S–R association that presumably was selected in Phase 1 should have been unchanged by the home–cage devaluation of the outcome, and as a result, responding should have persisted in the Phase-3 test. The S–O mechanistic and cognitive accounts, however, explain it in terms of the devaluation procedure reducing the value of the reinforcer outcome and consequently operant responding motivated by the expectation of the outcome responding should decline.

S–(R–O) Associations. In his review of associative relations in instrumental learning, Rescorla (1991) made a strong case for the establishment of associations that went beyond simple binary stimulus–response or response–outcome associations. In addition to S–R and R–O associations, Rescorla posited S–(R–O) associations, which can be understood as expectancies of particular outcomes (O) when certain responses (R) are emitted in the presence of an occasion setting (discriminative) stimulus (S). For example, in the typical operant conditioning procedure with rats, the discriminative stimuli of an operant chamber occasions the expectation that responding will lead to imminent arrival of food. Higher-order cognitive accounts describe instrumental conditioning as resulting in motivated action directed toward an expected goal (outcome), whereas contemporary mechanistic theories describe instrumental conditioning as resulting in motivated action because it was reinforced previously. In both cases, S–(R–O) associations are at the heart of modern conditioning theory. In contrast, prior S–R mechanistic explanations speculated that the reinforcing outcome selects a stimulus–response association, but does not become a component of the association—the prior reinforcement history simply compels the rat to respond in the presence of distinctive stimuli.

Cognitive Mediation in Clinical Research

Much of the data pertaining to the debate about higher-order cognitive mediation is derived from basic research on animal behavior. However, there also have been studies of clinical phenomena in which this issue has been addressed. In particular, studies of systematic desensitization and placebo analgesia have evaluated automatic and cognitive accounts of these phenomena.

Systematic Desensitization. Systematic desensitization (Wolpe, 1958) is a treatment for phobic anxiety that was inspired by Clark Hull's (1943) theory of conditioning. Phobic anxiety is posited to be a classically conditioned response. The idea behind the therapy is to associate the anxiety-arousing cues (the CS) with a new CR, one that is incompatible with anxiety (e.g., relaxation). Another explanation of systematic desensitization is extinction. According to the extinction hypothesis, in the absence of reinforcement by an aversive event (i.e., a US), repeated (or prolonged) exposure to the anxiety cues causes the CR (anxiety) to extinguish. One nice feature of the extinction hypothesis is that it works for other exposure-based treatments (e.g., flooding), as well as systematic desensitization.

The idea that exposure treatments are due to automatic conditioning processes has been tested in three studies (Gauthier, Laberge, Dufour, & Fevre, 1987; Kirsch & Henry, 1977; Southworth & Kirsch, 1988). In the first of these, Kirsch and Henry (1977) compared the effects of systematic desensitization and two credible expectancy modification procedures. One of these procedures was designed specifically to rule out conditioning hypotheses. In an *operant desensitization* condition, visualizations of anxiety-related scenes were paired with painful electric shocks, which subjects were told would "punish the anxiety." This expectancy modification procedure was as effective as standard systematic desensitization in reducing public-speaking fear. Because aversive stimuli are assumed to be the USs leading to the acquisition of fear as a CR, the substantial degree of fear reduction produced by *operant desensitization* cannot be accounted for by extinction or counter conditioning. Furthermore, because the addition of the electric shock was the only procedural difference between *operant* and traditional desensitization, it is reasonable to suspect that the effects of the two procedures were due to a common causal mechanism. Substantial correlations between pretreatment ratings of treatment credibility and treatment-outcome measures suggest that expectancy modification was the common causal agent.

A second test of mechanistic explanations of exposure treatments was a study of the effects of *in vivo* exposure on agoraphobia (Southworth & Kirsch, 1988). Over a two-week period, participants in this study were give ten sessions of *in vivo* exposure, during which they were asked to walk away from their homes until they became anxious and then to turn around and return. Half of the participants were told that the purpose of this was to lower their anxiety. The others were told that the purpose was to assess their anxiety and that treatment would not begin until after the two-week period. Clients provided with therapeutic expectancies showed substantially greater improvement and improved more rapidly than those who were led to believe that *in vivo* exposure was for the purpose of assessment, even when the distance and time walked was equated between the two groups. These data indicate that the therapeutic effects of *in vivo* exposure can be suppressed by disguising its therapeutic intent. These data were replicated by Gauthier et al. (1987) in a study of dental phobia. The findings of both of these studies are problematic for not only traditional S–R models of learning, but contemporary conditioning theories as well.

Placebo Effects. According to classical conditioning models of placebo effects (Ader & Cohen, 1991; Herrnstein, 1962; Wickramasekera, 1980), active medications are USs and the vehicles in which they are delivered (i.e., the pills, capsules, syringes, etc.) are CSs. The medical treatments that people experience during their lives constitute conditioning trials, during which the vehicles are paired with their active ingredients. These pairings endow the pills, capsules, and injections with the capacity to evoke therapeutic effects as CRs.

There are a number of studies demonstrating that classical conditioning procedures can enhance placebo analgesia (Voudouris, Peck, & Coleman, 1985, 1989, 1990). The placebo effect was enhanced in these studies by surreptitiously lowering the intensity of a pain stimulus whenever a part of the body treated with a placebo anesthetic was stimulated. The lowered intensity of the pain stimulus was the US and the placebo was the CS. This procedure increased the pain-reducing effect of the placebo, when subsequently it was tested without lowering the intensity of the pain stimulus.

Although the Voudouris et al. (1985, 1989, 1990) studies convincingly demonstrated conditioned enhancement of placebo pain relief, they did not discriminate between S–O mechanistic and higher-order cognitive interpretations of this phenomenon. In a follow

up to these studies, Montgomery and Kirsch (1997) investigated the effect of verbal information on this conditioning procedure. Recall that in the initial studies, the lowering of the intensity of the pain stimulus was done surreptitiously. Participants did not know that the intensity was lowered, and they therefore attributed the reduction in pain to the effect of the supposed topical anesthetic. Montgomery and Kirsch replicated this effect, but they also included an *informed-pairing* control condition in which participants were told that the intensity of the stimulus was being lowered. As in the condition replicating the original studies, participants in the informed-pairing condition were given trials in which reduced pain was paired with the application of a placebo. However, they also were given accurate information about how the reduction in pain was being produced. This verbal information completely reversed the effect of conditioning trials on the placebo response, which is not anticipated by even modern conditioning theories. In addition, regression analyses indicated that the effects of conditioning trials were mediated completely by participants' verbally rated expectancies.

In contrast to these data, conditioning with various drugs as USs has been reported to result in CRs that are the opposite of the URs (Siegel, 1983). For example, conditioning trials with morphine as the US produces increased sensitivity to painful stimuli as a response to the CS, and conditioning trials with tranquilizers like chlorpromazine as the US produces increased activity as a response to the CS. These data have been interpreted as indications of compensatory CRs, failures to identify accurately the actual US and/or UR when these drugs are administered (Donahoe & Palmer, 1994; Eikelboom & Stewart, 1982; Siegel, Baptista, Kim, McDonald, & Weise-Kelly, 2000). On one hand, these interpretations render this phenomenon consistent with current conditioning theories. On the other hand, regardless of interpretation, these data are inconsistent with a classical conditioning model of placebo effects. If increased pain sensitivity and activation are the CRs that are acquired when morphine and chlorpromazine are administered, then the pain-reducing effect of placebo morphine and the sedating effects of placebo tranquilizers cannot be CRs produced by the same mechanism. These data especially are important because the effects of placebo analgesics and tranquilizers are particularly well established.

In contrast, these data are not inconsistent with expectancy accounts of placebo effects because those effects are consistent with people's expectations. Most people expect morphine to reduce pain and tranquilizers to decrease activity, and the placebo effect is consistent with those expectations. Eikelboom and Stewart provided an account of how both mimetic and compensatory conditioned drug effects could arise from S–O mechanistic conditioning, but it also is possible that expectancy produces mimetic conditioned drug effects that are strong enough to override the conditioned effects of compensatory drug effects. In addition, expectancies can produce two conflicting response tendencies: an automatic mimetic response and a voluntary compensatory response. For example, in addition to an automatic response decrement, placebo alcohol can produce a voluntary compensatory response that is associated with the motivation to resist the expected deleterious effects, especially when the potential outcome is highly consequential (Vogel-Sprott & Fillmore, 1999).

Data Indicating Automatic Conditioning

Taken together, the data reviewed above provide clear evidence of cognitive mediation in both classical and operant conditioning. Other data, however, reveal conditioning phenomena that do not appear to be mediated cognitively. The data reviewed in this section seem to be explained more easily by automatic S–R processes and are consistent with a stimulus substitution model of conditioning (Pavlov, 1927).

Evaluative Conditioning

Evaluative conditioning occurs when a neutral conditional stimulus (CS) is paired with an affectively valenced, liked or disliked, unconditional stimulus (US) and results in a transfer of affect from the US to the CS (see Baeyens, Eelen, Crombez, & Van den Bergh, 1992). Two aspects of evaluative conditioning have been thought to distinguish it from traditional autonomic conditioning: (a) conditioning without awareness (Baeyens, Eelen, & Van den Bergh, 1990; Martin & Levey, 1978) and (b) resistance to extinction (Baeyens, Eelen, Van den Bergh, & Crombez, 1989). Although mechanistic interpretations of much of the evaluative conditioning literature have been challenged (e.g., Davey, 1994; Field & Davey, 1999; Lovibond & Shanks, 2002; Shanks & St John, 1994), studies of the evaluative conditioning of taste properties of odors (e.g., Stevenson, Prescott, & Boakes, 1995; Stevenson, Boakes, & Prescott, 1998) have yielded more convincing evidence of conditioning without awareness, leading Lovibond and Shanks to speculate that the gustatory system possesses special learning characteristics that operate outside of awareness. Studies of learning during anesthesia in nonhuman animals (e.g., Rabin & Rabin, 1984) also provided support for the possibility of learning without awareness.

S–R Persistence Following Devaluation

Recall that devaluation procedures were cited as support for a cognitive account of instrumental (as well as classical) conditioning. Specifically, devaluation of a stimulus that had been used to reinforce a particular response resulted in reduction of the response in simple (Dickinson, Nicholas, & Adams, 1983) and concurrent (Colwill & Rescorla, 1985a) schedules of reinforcement. However, the equally important observation that outcome devaluation does not always affect instrumental performance provides support for an S–R mechanistic interpretation of instrumental learning. When rats are trained on simple schedules of reinforcement, outcome devaluation reduces responding maintained on ratio, but not interval schedules of reinforcement (e.g., Dickinson et al., 1983). Thus, depending on the schedule of reinforcement, instrumental performance can be autonomous of its outcome. One explanation is that simple interval schedules fail to establish R–O associations because, unlike ratio schedules, the relationship between responding and rate of outcomes received is weak at moderate and high rates of responding (Dickinson, 1989). A seemingly contradictory finding is that interval schedules are effective in producing behaviors that are sensitive to outcome devaluation when they are used in concurrent schedules of reinforcement (Colwill & Rescorla, 1985a). Dickinson (1989) posited that the availability of different behaviors and their consequent outcomes in concurrent procedures provide the opportunity to learn strong response–outcome correlations. Thus, when there is sufficient opportunity to learn response–outcome correlations (e.g., simple ratio schedules and concurrent schedules), R–O associations control performance. However, when the response–outcome correlation is weak (e.g., simple interval schedules), S–R associations maintain performance.

Persistence following devaluation also has been observed in procedures that typically yield expectancy-based performance. Adams (1982) demonstrated that extended training on a ratio schedule renders outcome devaluation ineffective. Behavioral autonomy following extended practice appears not to be a result of the increase in the number of training reinforcers (Adams, 1982; Dickinson,1989), nor of simple repetition. Repetition of responding, for example, does not preclude expectancy-based performance when different behavior–outcome relationships are experienced in the same session (i.e., concurrent schedules; Colwill & Triola, 2002) or in alternating sessions (Colwill & Rescorla,

1985a; 1988). Dickinson (1989) had noted that the change from goal-directed expectancy to behavior that is autonomous of its outcomes might reflect changes in the response–outcome correlations that are experienced with extensive training in simple ratio schedules. Thus, the variability in rate of responding (and the consequent rate of reinforcement) that occurs early in training is reduced considerably with extended training (Dickinson, 1985). These observations suggest that, in the absence of consistent behavior–outcome correlations, instrumental performance is maintained by an S–R mechanistic process and is autonomous of the consequent outcomes.

Interestingly, a recent study suggests that behavioral autonomy from outcome status also can be observed in situations with consistent behavior–outcome correlations. Using the concurrent procedure, Dickinson, Wood, and Smith (2002) found that devaluation reduced instrumental performance when the outcome was food, but not when it was ethanol. This result is consistent with the view that alcohol-seeking behavior is maintained by S–R mechanistic habitual responding rather than goal-directed expectancy.

There also is evidence that cognitive and mechanistic processes can contribute to the same action. Although the response reduction after devaluation reported by Colwill and Rescorla (1985a, 1985b) strongly supports an explanation in terms of cognitive R–O associations, there are aspects of their data that are consistent with an S–R mechanistic interpretation. Devaluation of the reinforcing stimulus decreased the associated response, but it did not eliminate it altogether after brief or extended training. So why did the rats work at all to receive an outcome that had been devalued through taste aversion? A plausible explanation is that an S–R association maintained the residual responding (Colwill & Rescorla, 1985b; Dickinson, 1989; Nevin & Grace, 2000).

Resistance to Change and Excessive Behavior

Persistence of instrumental performance following outcome devaluation indicates that under some circumstances instrumental performance is resistant to post-conditioning changes in the consequent outcome. Resistance to change, or what Nevin has termed *behavioral momentum*, also is observed when response contingencies are altered in multiple schedules of reinforcement (e.g., Nevin & Grace, 2000). In this procedure, two or more schedules of reinforcement are correlated with distinctive stimuli. For example, responding is reinforced in the presence of one stimulus under a VI − 1 min schedule and in the presence of a second stimulus under a VI − 3 min schedule. The two stimulus–response–outcome contingencies are presented successively and separated by a brief time-out period. When noncontingent food is introduced in the time-out periods to disrupt performance, behavior under the richer schedule (VI − 1 min) is disrupted much less than behavior under the leaner schedule (VI − 3 min). This differential resistance to change also is observed when reinforcer value, rather than rate, is varied across stimulus situations. Nevin has shown that the overall rate of responding is determined by the response–outcome contingency, but resistance to change is determined by the overall rate of outcomes obtained in the stimulus situation (Nevin, Tota, Torquato, & Shull, 1990). The latter result was interpreted as indicating that resistance to change is modulated by a simple association between the discriminative stimulus (S^D) and the outcome. Good evidence for S^D–O associations come from transfer tests used to assess R–O associations (Colwill & Rescorla, 1988; Colwill & Triola, 2002). For example, an S^D present during one outcome will enhance performance of another response if it was trained with the same outcome, but not if it was trained with a different outcome. This result indicates that in instrumental conditioning procedures both R–O expectancy and simple S^D–O associations control instrumental performance (Colwill & Rescorla, 1988).

A dramatic example of the mechanistic control of behavior comes from studies demonstrating that a consistent history of intermittent delivery of reinforcers in a discriminative context (S^D) can generate bizarre and excessive (*adjunctive*) behavior in animals and humans (Falk, 1994). For example, when food-deprived rats are reinforced with food pellets on a fixed-interval schedule, they develop concurrent, excessive drinking (polydipsia). The consistent intermittency of food-pellet delivery is the important factor since schedule-induced drinking also occurs when the food is presented in the absence of an R–O contingency (i.e., a fixed time schedule). This procedure for producing chronic and excessive oral drug self-administration under strong stimulus control has been proposed as an animal model of drug abuse (e.g., Falk & Tang, 1988). Discriminative control of drug intake, whether it reflects resistance to change in the face of the disruptive effects of excessive drug use (Nevin & Grace, 2000) or schedule-induced self-administration (Falk & Tang, 1988), may be analogous to the phenomenon of relapse in humans. Relapse in recovering addicts is much more likely when drug abusers return to a situation previously associated with drug-taking behavior (Brownell, Marlatt, Lichtenstein, & Wilson, 1986) and is much less likely when drug users are removed suddenly from their drug-taking context to a radically different context. An example of the latter is that a very small percentage of soldiers who became addicted to heroin in Vietnam relapsed within three years of their return home (Robins, Helzer, Hesselbrock, & Wish, 1980; however, see Fish, 1998, for an alternative explanation).

Second-Order Conditioning

Evidence of S–R associations also is provided by studies of second-order conditioning procedures. In this procedure, animals are conditioned with standard pairings of a CS (e.g., a tone) and a US pairings (the first-order conditioning phase). In the next phase (the second-order conditioning phase), a second CS (e.g., light) is paired with the first-order CS that was conditioned in Phase 1, but with the US omitted. The result is that this second CS elicits CRs. A cognitive account explains the conditioning of the second-order light CS in much the same way it explained the sensory preconditioning experiment: After sufficient light–tone pairings, the light arouses a mental representation of the tone, which in turn arouses the expectation of a US, thereby generating a CR. However, unlike the sensory preconditioning results, an S–R association also can account for second-order conditioning; the second-order light CS may be conditioned as a result of contiguous pairings with the CR that was elicited by the tone CS. To differentiate these two accounts, Rizley and Rescorla (1972) extinguished responding to the tone after the second-order conditioning phase and subsequently presented the second-order light CS in a final test phase. The cognitive account predicts that extinction of the first-order tone CS should abolish responding to the second-order CS because the former should no longer arouse an expectancy of the US. The results failed to confirm this prediction, suggesting that the second-order conditioning, at least in this situation, was most likely a result of an association between the second-order CS and the CR elicited by the first-order CS. The ineffectiveness of US devaluation in reducing the CR-eliciting properties of second-order USs also provides evidence of S–R associations (Holland & Rescorla, 1975).

Conditioned Taste Aversions and Flavor Preferences

In the taste-aversion conditioning procedure, a taste stimulus (CS) is paired with a substance that produces gastrointestinal malaise (US). Because of this pairing, the subject

avoids consumption of the taste the next time it is encountered. Why? One explanation is that the taste is avoided because there is an expectation that consumption of the taste will cause gastrointestinal malaise (Rozin & Zellner, 1985). That is, behavior is guided by the information (danger) provided by the taste CS. An expectancy explanation of taste-aversion learning, however, does not account for the affective and hedonic changes that have been observed in humans and animals (Berridge, 2000). Pleasant tastes (e.g., sugar solutions) elicit positive affective facial and ingestive responses, but when paired with gastric malaise, there is a shift to negative defensive reactions (e.g., gaping and head shaking) that typically are seen with distasteful substances. In one study (Pelchat, Grill, Rozin, & Jacobs, 1983), a sweet taste was paired with treatments to produce upper-intestinal discomfort (LiCl toxicosis), lower-intestinal discomfort (lactose ingestion), or pain (electric shock). All three treatments led to avoidance of the taste stimulus, but only LiCl shifted the hedonic reaction to the taste stimuli from *like* to *dislike*. Whereas avoidance of the taste stimuli in the absence of shifts in hedonic quality may reflect an expectancy of the negative consequences of ingestion, the observed changed in the sensory evaluation of an initially preferred taste suggests an S–R automatic, noncognitive conditioning process (Rozin & Zellner, 1985).

Similar results have been observed in studies of conditioned flavor preferences. Rats learn to prefer a novel, mildly sweet flavor CS paired with intragastric infusions of nutrients (e.g., glucose) over another mildly sweet flavor CS paired with intragastric infusions of water (e.g., Drucker, Ackroff, & Sclafani, 1994). Evidence suggests that such conditioned flavor preferences sometimes reflect an increased positive hedonic value instead of an anticipation of the positive consequences of ingestion. For example, flavor preferences conditioned with intragastric nutrient infusion as a US are very resistant to extinction in both deprived and nondeprived animals (Drucker et al., 1988), enhance sham-feeding responses (Myers & Sclafani, 2001a), and shift taste reactivity toward responses typically seen with higher concentrations of sweet solutions (Myers & Sclafani, 2001b). However, as with conditioned taste aversions, not all conditioned flavor preferences appear to be mediated by changes in the hedonic properties of the flavors. When the CSs are initially unpalatable flavors rather than mildly sweet flavors, robust conditioned flavor preferences are not accompanied by enhanced positive hedonic reactions (Myers & Sclafani, in press). Together, these taste-aversion and flavor-preference studies suggest that the modulation of food choice through conditioning reflects two different processes—anticipation of the consequences of ingestion and a more mechanistic change in sensory evaluation (Rozin & Zellner, 1985; Myers & Sclafani, in press).

Conditioning with Subliminally Presented CSs

Additional evidence of automatic conditioning without awareness comes from experiments where the CSs are visual stimuli presented subliminally. Visual stimuli presented briefly (less than 300 msec) and immediately followed by a masking visual stimulus are not perceived consciously, yet are evaluated effectively as measured by explicit ratings or elicited autonomic responses. Words with extreme negative (e.g., *cancer*) or positive (e.g., *friend*) valence, when presented subliminally, elicit affective reactions that habituate with repeated subliminal presentations (Dijksterhuis & Smith, 2002). Phobic individuals presented with subliminal fear-relevant stimuli show increased electrodermal responses (Öhman & Soares, 1994). Because participants are not aware of visually masked stimuli if used as a CS in a Pavlovian conditioning paradigm and conditioned responding is observed, then conditioning will have occurred necessarily without the awareness of a

CS–US contingency. Several studies have reported conditioned responding to masked visual CSs despite lack of awareness of the CS or the CS–US contingency. For example, conditioned electrodermal responses have been observed when masked stimuli of *angry* faces or *threatening* animals were paired with an unpleasant shock US in nonphobic participants (Esteves, Parra, Dimberg, & Öhman, 1994; Öhman & Soares, 1998; Parra, Esteves, Flykt, & Öhman, 1997) . Masked stimuli that did not have an initial negative evaluation (e.g., pictures of a happy face or a flower), however, did not result in a conditioned electrodermal response (Öhman & Soares, 1998). Thus, it appears that automatic conditioning occurs only to preattentively perceived CSs that evoke some affective negative evaluation.

Conditioned Immunosuppression

Robert Ader (1985) conducted experiments that are procedurally similar to taste-aversion conditioning, but instead of conditioning a taste aversion, he conditioned immunosuppression. In this procedure, rats are allowed to consume a sweet tasting solution (CS) just before cyclophosphamide injections (US). The drug cyclophosphamide reduces the number of T-lymphocytes produced by the immune system of rats. When rats drink the taste CS in the absence of the drug, they show a reduced number of T-lymphocytes relative to control animals that received noncontingent pairings of the sweet taste and drug. The observation of conditioned immunosupression suggests that a normal, adaptive physiological function can be brought under control of an arbitrary stimulus such as a taste. It seems highly unlikely that rats could expect immunosupression or even have any representation of the phenomenon.

Conditioning in Simple Organisms

Evidence for automatic S–R associations also are provided by studies of conditioning in simple animals. For example, *Aplysia californica*, a large marine snail with a relatively simple nervous system of only a few hundred neurons, shows learned behavior in both classical and instrumental conditioning procedures (Carew, Hawkins, & Kandel, 1983). This simple invertebrate has an external gill that is withdrawn reflexively into a body cavity for protection. A mild tactile stimulus applied to tissue surrounding the gill produces little defensive gill withdrawal. However, after pairing this mild stimulus (CS) with a strong shock to the tail (US), the CS reliable elicits a robust gill-withdrawal response. It seems unlikely that an organism with such a simple nervous system would be capable of forming representations.

Conclusions

Data concerning two interpretations of classical and operant conditioning have been reviewed. One is the hypothesis that conditioning is an S–R mechanistic process in which expectancy and other cognitive factors are, at best, epiphenomena. From this perspective, conditioning trials produce conditional responses and perhaps expectancies, but there is no causal relation between expectancy and response. The other is cognitive theory, including S–O associations, according to which expectancy is hypothesized to mediate the effects of conditioning. From this perspective, conditioning trials produce expectancies, and it is the expectancy that produces the response.

Most traditional operant and classical conditioning phenomena can be explained by either S–R mechanistic or cognitive accounts (both simple S–O and higher order), and experiments that have been designed as critical tests of these rival hypotheses have yielded mixed results. On one hand, this review indicates abundant data disconfirming mechanistic S–R hypotheses and supporting cognitive interpretations in some situations. These data have led contemporary conditioning theorists to abandon earlier formulations in favor of approaches that are more consistent with cognitive theories. On the other hand, there are data indicating the occurrence in other situations of conditioned associations that are unlikely to be mediated cognitively. How can these data be reconciled?

Early debates about the nature of conditioning were based on the premise that it was exclusively either cognitive or mechanistic. A less parochial interpretation suggests that there are two types of conditioning processes, those that are mediated cognitively and those that are not. In addition, there appear to be learning processes that are not based on conditioning at all (e.g., learning by observation or through verbal communication). These aspects of the data suggest an unparsimonious proliferation of unconnected processes.

Conversely, there are data indicating important commonalities between conditioning and other forms of learning. Two examples of these are the informed-pairing and informed-unpairing studies reviewed by Brewer (1974). Participants in these studies who had been informed verbally about environmental contingencies behaved as they would have had they actually undergone conditioning or extinction trials. Similarly, verbally induced expectancies have been shown to produce patterns of responding that emulate various schedules of reinforcement (e.g., Wasserman & Shaklee, 1984). These data suggest a coordinated system of learning processes rather than a proliferation-independent learning mechanisms.

Cognition in complex organisms evolved from and incorporated more simple learning processes. Clearly, classical and operant conditioning of simple S–R associations are among the most basic processes. However, behavioral flexibility requires greater complexity. Thus, more complex organisms have evolved the ability to form representations (i.e., based on both R–O and S–O relationships) via conditioning procedures, as well as the ability to infer those relationships from other sources of information. It can be speculated that the more complex the organism, the smaller the role of automatic conditioning processes and the greater the role of representational cognition. This speculation is consistent with the data reviewed in this article showing that the provision of information generally overrides the effect of conditioning trials, especially in human participants, but also in laboratory animals.

Finally, the construct of *set* may bridge the apparent divide between automatic conditioning processes and representational cognitive processes. Simple S–R associations may be thought of as response sets. They are functionally anticipatory in that they prepare the organism for efficient automatic emission of the response when the appropriate stimulus conditions are encountered (Kirsch & Lynn, 1999). Similarly, S–O and R–O associations can be thought of as stimulus sets that prepare the organism to perceive environmental stimuli in particular ways. Examples include placebo effects and the effect of set on perceptions of ambiguous stimuli. Explicit expectancies are consciously accessible stimulus and response sets. Stimulus and response sets that are not consciously accessible could be thought of as implicit expectancies, although in doing so one might risk the danger that they could be reified gratuitously as a higher-level construct implying unconscious cognitions. Classical and operant conditioning are two of the means by which response sets are formed, but they also can be acquired vicariously though observation and through the provision of verbal information.

References

Adams, S.D. (1982). Variations in the sensitivity of instrumental responding to reinforcer devaluation. Quarterly Journal of Experimental Psychology, 3B, 77–98.

Ader, R. (1985). Conditioned taste aversions and immunopharmacology. Annals of the New York Academy of Sciences, 443, 293–307.

Ader, R., & Cohen, N. (1991). The influence of conditioning on immune responses. In R. Ader & D.L. Felten (Eds.), Psychoneuroimmunology (2nd ed., pp. 611–646). San Diego, CA: Academic.

Allan, L.G. (1993). Human contingency judgment: Rule-based or associative? Psychological Bulletin, 114, 435–448.

Allyon, T., & Azrin, N.H. (1968). The token economy: A motivation system for therapy and rehabilitation. New York: Appleton-Century-Crofts.

Arnold, H.M., Grahame, N.J., & Miller, R.R. (1991). Higher-order occasion setting. Animal Learning & Behavior, 19, 58–64.

Ayres, J.J., Haddad, C., & Albert, M. (1987). One-trial excitatory backward conditioning as assessed by conditioned suppression of licking in rats: Concurrent observations of lick suppression and defensive behaviors. Animal Learning & Behavior, 5, 212–217.

Baeyens, F., Eelen, P., Crombez, G., & Van den Bergh, O. (1992). Human evaluative conditioning: Acquisition trials, presentation schedule, evaluative style and contingency awareness. Behaviour Research and Therapy, 30, 133–142.

Baeyens, F., Eelen, P., & Van den Bergh, O. (1990). Contingency awareness in evaluative conditioning: A case for unaware affective-evaluative learning. Cognition and Emotion, 4, 3–18.

Baeyens, F., Eelen, P., Van den Bergh, O., & Crombez, G. (1989). Acquired affective evaluative value: Conservative but not unchangeable. Behaviour Research and Therapy, 27, 279–287.

Beck, E.C., & Doty, R.W. (1957). Conditioned flexion reflexes acquired during combined catalepsy and de-efferentiation. Journal of Comparative Physiological Psychology, 50, 211–216.

Berridge, K.C. (2000). Measuring hedonic impact in animals and infants: Microstructure of affective taste reactivity patterns. Neuroscience and Biobehavioral Reviews, 24, 173–198.

Blodgett, H.C. (1929). The effect of the introduction of reward upon the maze performance of rats. University of California Publications in Psychology, 4, 113–134.

Boakes, R.A. (1989). How one might find evidence for conditioning in adult humans. In T. Archer & L.G. Nilsson (Eds.), Aversion, avoidance and anxiety: Perspectives on learning and memory. England: Lawrence Erlbaum Associates.

Bolles, R.C. (1979). Learning theory (2nd ed.). New York: Holt, Rinehart & Winston.

Bouton, M.E., & King, D.A. (1983). Contextual control of the extinction of conditioned fear: Tests for the associative value of the context. Journal of Experimental Psychology: Animal Behavior Processes, 9, 248–265.

Brewer, W.F. (1974). There is no convincing evidence for operant or classical conditioning an adult humans. In W.B. Weimer & D.S. Palermo (Eds.), Cognition and the symbolic processes (pp. 1–42). Hillsdale, NJ: Lawrence Erlbaum Associates.

Brownell, K.D., Marlatt, G.A., Lichtenstein, E., & Wilson, G.T. (1986). Understanding and preventing relapse. American Psychologist, 41, 765–782.

Carew, T.J., Hawkins, R.D., & Kandel, E.R. (1983). Differential classical conditioning of a defensive withdrawal reflex in Aplysiacalifornica. Science, 219, 397–420.

Colwill, R.M., & Rescorla, R.M. (1985a). Post-conditioning devaluation of a reinforcer affects instrumental responding. Journal of Experimental Psychology: Animal Behavior Processes, 11, 120–132.

Colwill, R.M., & Rescorla, R.A. (1985b). Instrumental responding remains sensitive to reinforcer devaluation after extensive training. Journal of Experimental Psychology: Animal Behavior Processes, 11, 520–536.

OPEN ACCESS Freely available online

PLOS | ONE

Effect of Circadian Phase on Memory Acquisition and Recall: Operant Conditioning vs. Classical Conditioning

Source 2

Madeleine V. Garren, Stephen B. Sexauer, Terry L. Page*

Department of Biological Sciences, Vanderbilt University, Nashville, Tennessee, United States of America

Abstract

There have been several studies on the role of circadian clocks in the regulation of associative learning and memory processes in both vertebrate and invertebrate species. The results have been quite variable and at present it is unclear to what extent the variability observed reflects species differences or differences in methodology. Previous results have shown that following differential classical conditioning in the cockroach, *Rhyparobia maderae*, in an olfactory discrimination task, formation of the short-term and long-term memory is under strict circadian control. In contrast, there appeared to be no circadian regulation of the ability to recall established memories. In the present study, we show that following operant conditioning of the same species in a very similar olfactory discrimination task, there is no impact of the circadian system on either short-term or long-term memory formation. On the other hand, ability to recall established memories is strongly tied to the circadian phase of training. On the basis of these data and those previously reported for phylogenetically diverse species, it is suggested that there may be fundamental differences in the way the circadian system regulates learning and memory in classical and operant conditioning.

Citation: Garren MV, Sexauer SB, Page TL (2013) Effect of Circadian Phase on Memory Acquisition and Recall: Operant Conditioning vs. Classical Conditioning. PLoS ONE 8(3): e58693. doi:10.1371/journal.pone.0058693

Editor: Troy Zars, University of Missouri, United States of America

Received October 18, 2012; **Accepted** February 5, 2013; **Published** March 22, 2013

Funding: This work was supported by National Science Foundation grant #IOS-1145605 to TLP. The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.

Competing Interests: The authors have declared that no competing interests exist.

* E-mail: Terry.L.Page@Vanderbilt.Edu

Introduction

In the past decade, several studies have indicated that circadian clocks may have varied effects on learning and memory. In some cases, the ability to form a memory may be independent of circadian phase, but phase may function as a contextual cue (time-stamping) such that recall and performance are better at 24-hour intervals following learning as demonstrated in hamsters [1] and rats [2–4]. In other cases, recall appears to be largely independent of the phase of testing, but memory acquisition or consolidation may depend on the circadian phase of training as shown in mollusks [5,6], insects [7–9], fish [10], and mice [11,12].

There have also been reports that disruption of the circadian system by phase-shifting ("jet-lag") can impair memory in rats [13–15] and that internal phase relationships are important for learning in humans [16]. Finally, two recent studies presented data indicating that abolition of circadian cycling in hamsters impairs performance in a declarative memory task [17] and that ongoing circadian oscillations in the hippocampus are necessary for long-term memory stability following fear conditioning in mice [18].

In summary, it seems clear that the circadian system can have widespread effects on various aspects of learning and memory including acquisition, retention, and recall; however, at this point numerous questions remain both about the mechanisms by which the circadian system regulates these processes and about the functional/adaptive significance of this novel feature of circadian organization. One of the problems with sorting out the various results to come to clear understanding of underlying principles of the circadian system's role in associative memory formation is that the experiments have used various species, various conditioning paradigms, and various stimuli for reinforcement. Thus it is unclear whether differences in results reflect fundamental differences in the role of the circadian system in learning and memory or, alternatively, simply reflect a "hodge-podge" of species and methodological differences that obscures any underlying general principles.

The cockroach may be an excellent model for addressing these issues. Cockroaches can be trained both by classical and operant conditioning paradigms using virtually identical stimuli for reinforcement [7,19,20]. Thus we eliminate much of the variability that plagues comparisons among published studies and are able to focus on differences in circadian regulation of various forms of associative memory. Using a differential classical conditioning protocol it has been shown that the circadian system regulates olfactory learning and memory in the cockroach *Rhyparobia (Leucophaea) maderae* [7]. In this study, the effect of training and testing at different circadian phases on performance in an odor discrimination test was investigated. When the cockroaches were allowed to choose between two odors (peppermint and vanilla), naïve animals showed a clear preference for vanilla at all circadian phases. The results indicated there was no circadian modulation of initial odor preference or ability to discriminate between odors. Training involved differential classical conditioning in which peppermint odor was associated with a positive unconditioned stimulus (US+) of sucrose solution and vanilla odor was associated with a negative unconditioned stimulus (US−) of saline solution. It was found that cockroaches conditioned in the early subjective night showed a strong preference for peppermint and retained the memory for at least two days. Animals trained and tested at other

circadian times (CT) showed significant deficits in performance for both short-term and long-term memory. At CT 2 (early subjective day) the deficit was profound and animals that had been trained at this phase were behaviorally indistinguishable from naïve, untrained animals. In contrast, recall of a learned memory was independent of the phase of testing – animals trained at CT 14 were able to recall at CT 2.

In the present study we show that *R. maderae* can also be trained via an operant conditioning protocol that utilizes the same sensory cues that were used for classical conditioning. Further we show that, unlike classical conditioning, with operant conditioning animals are able to acquire memories at any circadian phase but that their ability to recall long-term memories is tied to the phase of training. The results indicate that the impact of circadian regulation of learning and memory is strongly dependent on the form of training.

Results

Operant Conditioning can Establish Both Short and Long-term Memories

We first wanted to determine if *R. maderae* could indeed learn by operant conditioning. Conditioning involved placing animals that had been isolated from food and water for 6–7 days in a cylindrical plastic container with two odor choices on opposite sides of the arena. Peppermint, which is an aversive odor, was associated with a standardized slice of apple as a reward. The second odor was an attractant (vanilla) that was paired with apple made inaccessible by covering with fine mesh netting. The arena was housed in very dim red light and the animal's behavior was monitored with an infra-red video camera. Typically in the initial trial, animals would "visit" the inaccessible apple slice at the vanilla 4–6 times before they approached the peppermint and consumed the apple associated with the aversive odor. In subsequent trials a reduction in the number of visits to vanilla prior to acquiring the apple at peppermint was taken a measure of learning. In initial experiments the animals were trained and tested at CT 14, a phase when they have been shown to be capable memory formation by classical conditioning [7]. Memory was evaluated by the performance at 5 minutes, 90 minutes, 48 hours and 9 days (216 h). Following the initial training trial and consumption of the apple slice, animals consistently showed a significantly reduced number of visits to vanilla prior to the visit to peppermint and the receipt of the reward in the 5-minute trial. Little change in performance occurred in subsequent trials indicating that animals were capable of both short-term and long-term memory (Fig. 1A). In additional experiments in which animals were given three training trials on each of two consecutive days at CT 14, performance was excellent at both one-week and two-week tests (Fig. 1B). Notably, in previous results with classical conditioning (3 training trials) long-term memories were generally more labile lasting only 3–4 days [7] while the present data indicate that memories formed via operant conditioning persisted for over a week with little decrement.

In view of the fact that there is a robust circadian regulation of memory acquisition via classical conditioning [7] we anticipated that animals would not be successful at forming the associative memory if trained at CT 2 (a phase where they appear to be incapable of memory acquisition via classical conditioning). Surprisingly, when animals were trained at CT 2 using our operant conditioning protocol they performed just as well as the animals trained at CT 14 (Fig. 1C) exhibiting both short-term and long-term associative memory and there was no evidence of any significant deficit in the ability to perform the task.

As a control to demonstrate that the changes in odor preference were in fact due to an association between the apple reward and the peppermint odor, the protocol was revised such that animals received no positive reward for peppermint visits by making the reinforcement inaccessible at both odor sources. As shown in Fig. 1D there was no decrease in the preference for vanilla when the apple reward at peppermint was not available. The results confirmed that the changes in odor preference reflected the formation of an associative memory.

Associative Memory Formation is Independent of Reward

While the odor sources used in the operant task (peppermint and vanilla) were the same as those used to demonstrate a circadian rhythm in effectiveness of classical conditioning [7], in the earlier experiments the positive unconditioned stimulus was a sucrose solution rather than apple. Thus one potential explanation for the difference in the impact of circadian phase on memory formation was that the sensory information from the apple stimulus was processed differently from the sucrose solution, in one case being subject to circadian regulation (sucrose) while perception of the other (apple) was independent of circadian phase. Therefore we repeated our experiments utilizing a 20% sucrose solution as a reward to match more closely the positive US we had used previously for classical conditioning. The results are shown in Fig. 2. When sucrose was offered as a reward, it was just as effective as the apple in modifying odor preference behavior at both CT 14 (Fig. 2A) and at CT 2 (Fig. 2B). As an additional control we showed that when the sucrose that was paired with the peppermint odor was also covered with netting to prevent the cockroach from receiving the reward at the peppermint odor, there was no change in odor preference (Fig. 1C). The results confirmed the shift in odor preference was due to an association between the peppermint odor and the sucrose reward. These observations show that both short-term and long-term associative memories in which the peppermint odor is associated with a reward are formed equally well at CT 14 and at CT 2 in an operant conditioning task independent of whether the reward is apple or sucrose.

The results suggested that either there is no effect of the circadian system on memory formation or that the phasing of the effect was quite different for the two forms of learning. In order to distinguish between these two possibilities, we trained animals at two additional circadian times (CT 8 and CT 20). Fig. 3 plots the Learning Indices for all four circadian times memory. There was no significant dependence of performance on circadian phase. With regard to short-term memory the learning indices were nearly identical at all circadian times. There is more variability in the results for long-term memory (e.g., performance at CT 8 was somewhat better than at other phases); however, there were no statistically significant dependence of performance on the circadian phase of training.

In view of the fact that both the classical and operant conditioning protocols were closely matched in terms of the stimuli used, we found it surprising that memory formation via classical conditioning exhibited a robust circadian rhythm while memory formation following operant conditioning appeared to be completely independent of the circadian system. However, the experiments involving classical conditioning utilized a differential conditioning protocol in which the peppermint was associated with a positive unconditioned stimulus (sucrose) while the vanilla was associated with a negative unconditioned stimulus (saline). Thus a possible explanation of the differences we were finding was that the circadian modulation in classical conditioning was due to circadian regulation of the response to the aversive (saline)

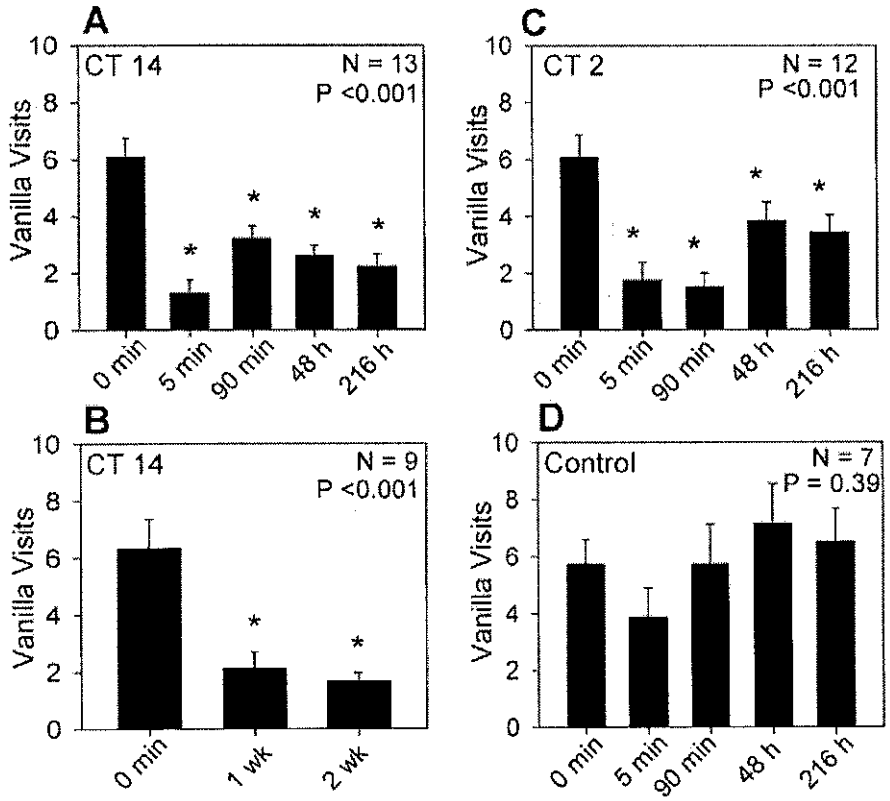

Figure 1. Each panel plots the number of times (Mean±SEM) the animals visited a vanilla odor prior to visiting peppermint as a function of the training/testing time. A, animals were subjected to training sessions in the early subjective night (CT14) and were rewarded with a slice of apple when they visited the peppermint. Prior to any reward (0 min) animals exhibited a clear preference for vanilla. In subsequent trials animals showed a significant reduction in vanilla visits prior to visiting peppermint. B, Animals were subjected to two consecutive days of training at CT 14 (three trails in each session with a 5 minute inter-trial interval. There was a highly significant reduction in the number of visits to vanilla made prior to the visit to peppermint both one and two weeks later compared to the initial trial (0 min.). C, when trained at CT 2, animals exhibited a similar reduction in vanilla visits to the animals trained at CT 14. D, when access to the reward at peppermint was prevented during training (CT14), there was no significant change in the number of vanilla visits. P-values for the ANOVA are indicated in the figure. Bars marked with * indicate a statistically significant difference ($p<0.05$) when compared to the initial number of vanilla visits (Holm-Sidak post-hoc test).
doi:10.1371/journal.pone.0058693.g001

stimulus. In order to test this, we trained animals to an operant conditioning task in which the peppermint was paired with sucrose and the vanilla was paired with an accessible saline solution. Animals still exhibited excellent performance in both short-term and long-term memory tests whether they were trained at CT 2 or CT 14 (Fig. 4). Notably, on initial visits to vanilla the animals did

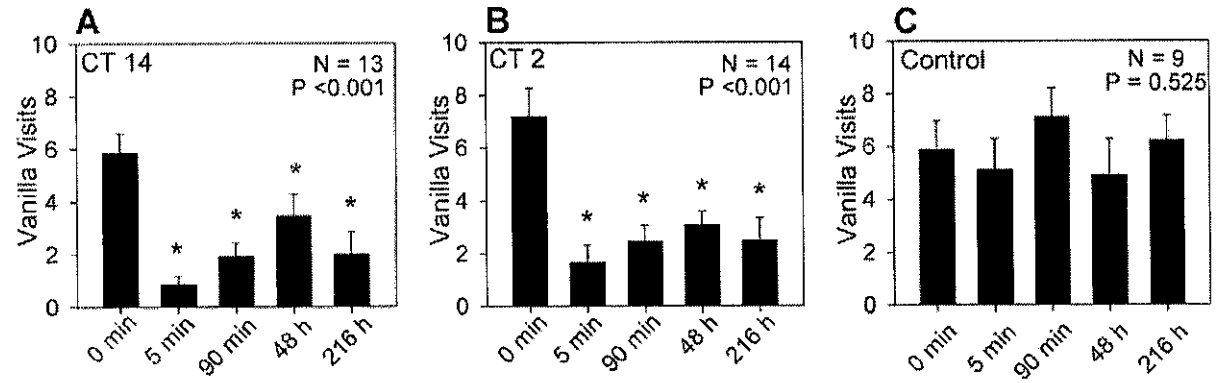

Figure 2. Plots the number of times (Mean±SEM) animals visited the vanilla odor prior to visiting peppermint as a function of the training/testing sequence when trained at CT 14 (A) or CT 2 (B). Sucrose rather than apple was offered as a reward. C, when access to the sucrose reward at peppermint was prevented during training (CT14), there was no significant change in the number of vanilla visits. P-values for the ANOVA are indicated in the figure. Bars marked with * indicate a statistically significant difference ($p<0.05$) when compared to the initial number of vanilla visits (Holm-Sidak post-hoc test).
doi:10.1371/journal.pone.0058693.g002

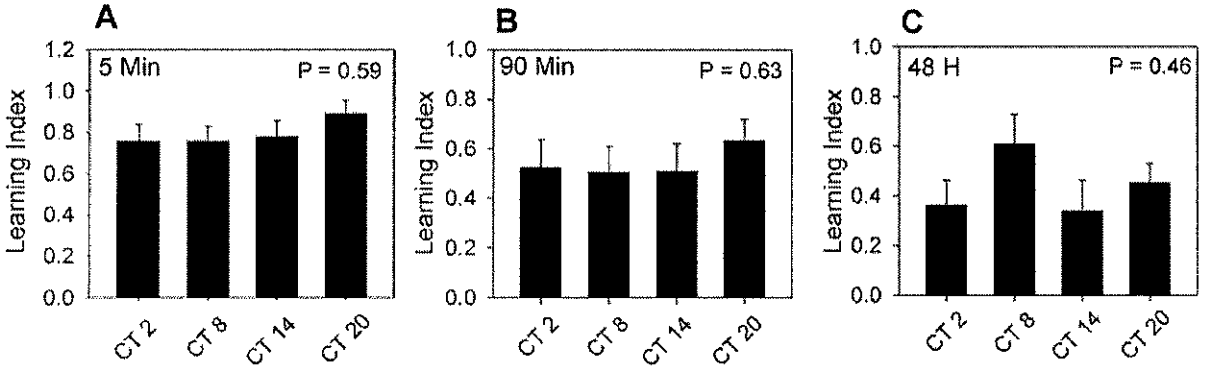

Figure 3. Plots of learning index as a function of the circadian phase of training and testing for 5 min, 90 min, and 48 h memory tests. Analysis of variance showed no significant dependence of performance on phase of training for measures of either short-term or long-term memory. Numbers of animals for each circadian phase ranged from 10 to 15.
doi:10.1371/journal.pone.0058693.g003

appear to contact the saline solution (but did not drink it) indicating they were in fact exposed to the negative reinforcement. Support for this contention was obtained in trials in which we offered sucrose solution instead of saline at the vanilla. Four of five animals consumed the sucrose at the vanilla odor prior to a visit to peppermint showing that they were sampling the reinforcement solution presented at vanilla before moving to peppermint.

The results suggested that the difference in the circadian regulation of classical and operant conditioning was not dependent on differences in the olfactory or gustatory cues the animals were exposed to in the two training protocols.

Temporal Regulation of Recall

Previous results with differential classical conditioning [7] showed that recall of an acquired memory was independent of circadian phase. Animals trained at CT 14 were able to perform well on the task even if they were tested at CT 2 (a time when they were incapable of memory acquisition). The results were quite different when we trained animals at CT 14 in the operant conditioning task and tested them 12, 24, 36, or 48 hours later. Recall was significantly better when animals were tested at CT 14 (24 and 48 h tests) than when they were tested at CT 2 (12 and 36 h tests) (Fig. 5A). The results suggested that circadian phase at the time of training is a contextual cue influencing the ability to perform the task. In order to confirm that successful recall was tied to the phase of training, we trained animals at CT 2 and tested them at either 12, 24, 36, or 48 hours after training. In this case recall was better if the animals were tested at times corresponding to CT 2 rather than CT 14 (Fig. 5B). In essence, animals perform better when tested at the same circadian phase at which they were trained (independent of the phase of training) suggesting that circadian phase is an important contextual cue for memory recall.

Discussion

In classical (Pavlovian) conditioning animals learn about the relationship between two stimuli, while in operant (instrumental) conditioning it is the relationship between stimuli and the consequences of the animal's own behavior that is critical. While the two forms of associative learning are operationally distinct, the basic question of how these different forms of learning are related and whether or not they involve the same or fundamentally different underlying processes is still uncertain [21]. While the analysis of classical conditioning has proceeded rapidly, there is much less information available on mechanisms of operant conditioning. However, recent data do suggest that while there are similarities, significant differences exist at the cellular/

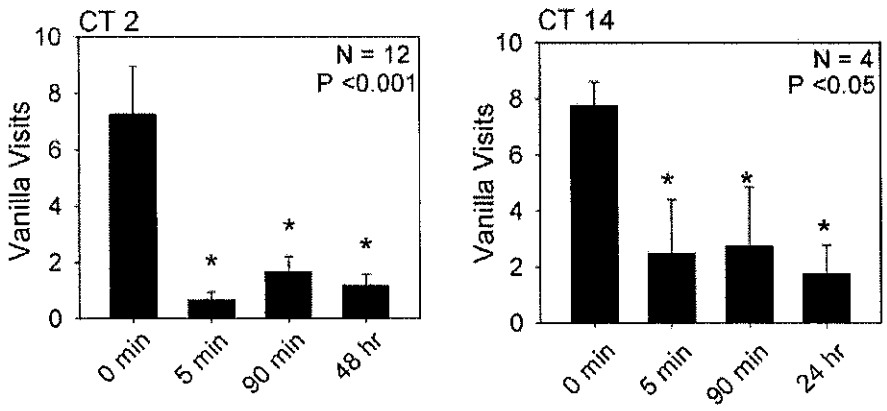

Figure 4. Plots vanilla visits as a function of training/testing time when the peppermint odor was paired with the positive reinforcement of sucrose solution and the vanilla odor was paired with an accessible negative reinforcement of saline. Left panel, training at CT 2; right panel training at CT 14. P-values for the ANOVA are indicated in the figure. Bars marked with * indicate a statistically significant difference ($p<0.05$) when compared to the initial number of vanilla visits (Holm-Sidak post-hoc test).
doi:10.1371/journal.pone.0058693.g004

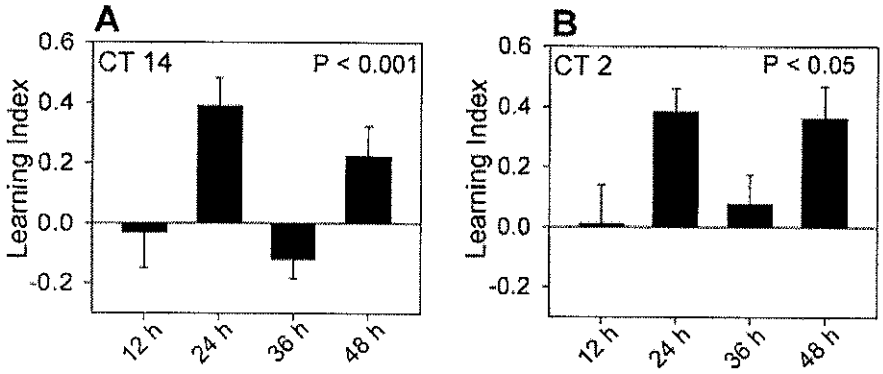

Figure 5. Plots the learning index for animals trained at either CT 14 (A) or CT 2 (B) and tested 12, 24, 36, or 48 h after training. Animals tested at the same circadian time as training performed better than those tested at a phase 12 hours different from the phase of training. Numbers of animals for each time point ranged from 14 to 17 in A and 10 to 11 in B. Analysis of variance showed significant dependence of performance on the time of testing for training at both CT 2 and CT 14.
doi:10.1371/journal.pone.0058693.g005

molecular level [22–24]. The extent to which these differences in mechanism may be reflected in differences in circadian regulation is unclear. With regard to the role of the circadian system in learning and memory, studies have addressed questions of memory acquisition (short-term memory); memory consolidation (long-term memory) and long-term memory recall utilizing both classical and operant conditioning. The outcomes of these studies have been varied.

Circadian Regulation of Memory Formation

Although the number of studies is limited, based on previously published reports, it appears that the role of the circadian system in the regulation of learning and memory may generally be different for classical and operant conditioning. For memory acquisition, circadian regulation appears to be prevalent in classical conditioning paradigms (mice [11]; zebrafish [10]; cockroaches [7]; fruit flies [8]; bees [9]). The one apparent exception to this generalization occurred in golden hamsters (*Mesocricetus auratus*) subjected to conditioned place preference or conditioned place avoidance [25,26].

In contrast to the general finding that memory acquisition during classical conditioning varies with circadian phase, the results from operant conditioning studies have suggested that learning and short-term memory are independent of the circadian system (rats [2,3]; marmosets [4]; *Aplysia* [6]). Similarly, the formation of long-term memories appears to depend on the circadian phase of training in classical conditioning while, with one exception [6], long-term memory formation appears to be independent of circadian phase in operant conditioning paradigms.

On the basis of these published results, one might suggest that there potentially generalizable differences between operant and classical conditioning in the way in which the circadian system regulates memory formation. One reason to be cautious is that in some cases it may be difficult to distinguish whether or not an animal is relying exclusively on operant or classical features of memory acquisition. Another major problem is that the responses to the two mechanisms for associative memory formation had never been examined in the same species and generally involved very different training methods (and thus very different sensory inputs and behavioral outputs). Therefore differences could be dismissed as variation due to differences between species or methodology. The results presented here however, indicate that in the cockroach, *Rhyparobia maderae*, the differences in the role of the circadian system in regulating the formation of new memories can likely be attributed to fundamental differences in the way in which memories are formed by the two types of conditioning. Experiments with classical conditioning in the cockroach demonstrated that memory acquisition in an odor discrimination task is regulated by the circadian system [7]. In contrast, the results presented here show that both short- and long-term memory formation via operant conditioning in a very similar odor discrimination task are independent of circadian phase. These results lend support to the notion that the circadian regulation of memory formation may be different between memories that arise from classical conditioning and those that are formed via operant conditioning. The data raise a variety of general questions of interest. The first concerns the mechanisms by which circadian clocks "gate" memory formation in classical conditioning, and by the same token, why it doesn't appear to impact memory formation with operant conditioning. In the cockroach, the difference in effectiveness between classical and operant conditioning at CT 2 is evident within 5 minutes after the training. Similarly, in mice [11], zebrafish [10], and fruit flies [8] circadian regulation modifies performance early in the process of memory acquisition. The data suggest that whatever the regulatory role of the circadian system, its impact is significant very early (within minutes) in acquisition process (which provides an experimentally attractive limited time window for further study).

At present there are only three studies that appear to directly approach the question of how the circadian system regulates memory formation. In one recent study involving a novel object recognition task in Siberian hamsters (*Phodopus sungorus*), it was shown that the $GABA_A$ receptor antagonist, pentylenetetrazol, was able to restore learning deficits caused by disruption of the circadian system. The results indicated that GABAergic signaling controlled by the circadian clock in the suprachiasmatic nucleus may be responsible for suppressing memory acquisition at inappropriate times of day [17]. Interestingly, as the authors note, this hypothesis could also explain the observation that SCN-lesioned rodents generally improved or failed to have a negative effect on learning [27,28]. Conceptually similar results were obtained in another study on zebrafish, though the details differed. In the zebrafish the data suggested that night-time melatonin secretion from the pineal gland was responsible for suppression of memory acquisition at night, and removal of the pineal or treatment with melatonin receptor antagonists abolished memory deficits at night [10]. Thus in both of these cases a circadian clock

appears to actively suppress early stages of memory formation during part of the circadian cycle, and destruction of the clock or pharmacological interference with the output signal rescues the learning deficit. The targets of suppression are not yet clear. At these early stages (i.e., short-term memory), protein synthesis is unnecessary for recall or performance, thus it is unlikely that regulation of transcription or translation is involved. In contrast, in diurnal *Aplysia* results indicate that at night when the animals exhibit a deficit in long-term memory, the circadian clock actively suppresses persistent MAPK activation and thus the transcriptional activation necessary for long-term memory while leaving the processes of memory acquisition and short-term memory unaffected [29].

In summary, in these markedly different species, mechanisms of circadian regulation of memory formation appears to be quite diverse in detail but do exhibit the common feature that the clock appears to be actively suppressing memory formation at "inappropriate" times of day. On the other hand, no clear picture emerges to answer the question of what mechanistic differences between memory formation and retrieval could account for differential regulation of operant and classical conditioning by the circadian system.

Circadian Regulation of Long-term Memory Recall

With regard to the circadian regulation of recall of long-term memories, the contrast between operant and classical conditioning appears to be less consistent. Studies have indicated three different outcomes for the role of the circadian system in recall long-term memory recall following classical conditioning In mice, recall ability following context or tone cued fear conditioning was better during the subjective day, independent of the time of training [11,18]. The results suggested the circadian system either limits access to long-term memory stores to a fixed set of circadian phases or modulates down-stream processes related to performance. In contrast, in the golden hamster, following classical conditioning (conditioned place preference or avoidance), recall was better when the test was done at the same circadian phase as the training independent of whether animals were trained in the night or the day [1,25,26]. In this case, the results suggested that circadian phase of training became a contextual cue that determined performance during testing. Finally, recall following classical conditioning in the zebrafish [10] or the cockroach [7] appeared to be independent of the time of testing.

Similar variability has been evident on studies of operant conditioning. Following operant conditioning in the cockroach, recall was better when the test was done at the same circadian phase as training indicating (as in classical conditioning in the hamster). This suggests that circadian phase of training was a contextual cue that determined performance during testing. On the other hand, in *Aplysia* recall of long-term memory after operant conditioning appeared to be independent of the time of testing [6]. Overall, the data suggest that the variability in the ability to recall a consolidated memory at various points in the circadian cycle is not readily tied to a particular mode of training.

Adaptive Significance

The second general question concerns the adaptive significance of circadian control and why circadian regulation of classical and operant conditioning should be different. One speculative suggestion that emerged from classical conditioning studies was that memories are only profitable when formed in the environmental context in which they will be used [7]. The data from cockroaches are largely consistent with this notion. In the case of classical conditioning, the suppression of memory formation occurs at a time when the animals are least active and thus least likely to be using olfactory cues in foraging behavior. As a consequence, formation of memories by completely external intervention is suppressed at times when the animal is not normally engaged in olfactory driven behavior and the associative memory is not likely to be useful in guiding future behavior.

Conversely, if the animal is *voluntarily* out foraging (even at an unlikely time as might happen when food is scarce [30]) and is rewarded, then memory of the success of the behavior becomes useful and is tied to the circadian phase at which that particular olfactory environment was profitable. At other times of day, when the olfactory or reward environment is likely to have changed, the memory is not used to guide behavior. This is reminiscent of the early work on honey bees [31] and later work with birds [32,33] and fish [34,35] where the animals were shown to select feeding sites at those specific times of day when they had previously been successful. The notion here then is that the role of the circadian system in regulation of learning and memory is to limit formation of new memories or utilization of established memories to only those times of day when they are likely to be profitably used in the future.

Materials and Methods

Animals

Colonies of *Rhyparobia maderae* (more commonly known as *Leucophaea maderae)* were maintained in 12-h light/dark cycles (LD 12:12). One week before the experiment began, six to twelve young adult males were isolated from food and water and housed together in a round plastic container 9 cm tall and 24.5 cm in diameter. All animals were transferred to constant darkness at end of the last complete light period prior to training. The experiments were conducted in an environmental room under dim red light at 24.5°C. The animals remained in a light-tight box in constant darkness (DD) until the conclusion of the experiment.

Operant Conditioning

The methods were adapted from [19] and [7]. The strategy was to reward animals for visiting an aversive peppermint odor rather than an attractive vanilla odor – the reward was either a small slice of apple or 20% sucrose solution. Animals were trained and tested in a circular Plexiglas arena 30 cm in diameter and 9.5 cm tall, the sides of which were smeared with petroleum jelly to prevent escape. This arena was housed in a closed box, the interior of which was illuminated with dim darkroom safelight that limited visible light wavelengths to greater than 600 nanometers (Kodak 1A or GBX-2; Eastman- Kodak, Rochester, NY). Light intensity was adjusted with a rheostat to a final intensity at the floor of the testing arena less than 0.1 $\mu E\text{-}m^{-2}\text{-}scc^{-1}$ (LiCor Photometer). Odors were provided by placing a 1.5-cm-square piece of filter paper, saturated with 20 µl of either vanilla or peppermint extract, into a 1.5 ml microfuge tube aerated with small holes. The microfuge tube was connected to a petri dish lid 3.1 cm in diameter. For training and testing, one vanilla odor and one peppermint odor source were placed at opposite sides of the arena. A small cup in each Petri dish lid (made from a microfuge tube lid) held either a 75 µl sucrose reward or a slice of apple. A "standard" apple reward was prepared by inserting glass tubing, 0.5 cm in diameter, through the apple. The cylindrical core of apple retrieved from the tube was sliced with two razor blades separated by the 1 mm width of a microscope slide to produce 1 mm-thick apple slices that weighed approximately 0.01 g. The reward was covered with a fine mesh netting on the vanilla apparatus but was not covered on the peppermint apparatus, allowing the animal

access to the positive reinforcement at peppermint only. In one series of experiments as noted in the results, the vanilla odor was paired with a negative reinforcement of an accessible 20% NaCl solution.

Individuals were placed in the center of the arena and covered with a Petri dish until the training trial began. Animals were observed using an infrared-sensitive CCD camera (Sony XC-77; Sony, Tokyo, Japan). When the timer was started, the animal was allowed to run freely in the arena between the two odor sources. The animal was expected to visit the vanilla odor first before visiting the peppermint, since it has been shown that cockroaches have an innate preference for the vanilla odor [7,19]. In order to ensure that all subjects in the study exhibited a naïve preference for vanilla and to eliminate inactive individuals, each animal was required to visit the vanilla at least three times before visiting the peppermint or it was dropped from further training trials. A visit to vanilla was recorded when the animal probed the netting covering the reward with its mouthparts. The number of vanilla visits was recorded until the animal first visited the peppermint and received the positive reinforcement which concluded the training trial.

Training and Testing Schedule

Circadian Times (CT) of training and testing were estimates based on the time of lights-off of the prior light cycle (designated as CT 12) and assumed a freerunning period of 24 hours. Training was conducted at either: CT 0–3 (corresponding to the early subjective day); CT 6–9 (late subjective day); CT 12–15 (early subjective night); or CT 18–21 (late subjective night). For most experiments, each training session consisted of three "training trials," The first was designated as time 0, the second at 5 minutes after the end of the first, and a third 60–120 minutes after the second trial was complete (referred to as 90 minutes in the results). We note that the variability of timing in the third training trial could have introduced more inter-individual variability in performance [36,37] but was necessary for sufficient numbers of animals to be trained in one session. Subsequent trials were carried out either 12, 24, 36, or 48 hours, or 9 days later. For one experiment we subjected animals to training sessions on two consecutive days. For each of the two days there were 3 trials with an interval of 5 minutes between each. Testing on these animals was carried out one and two weeks after the last training trial.

When the interval between two training trials was 5 minutes, animals were allowed to remain in the testing arena. After the remaining trials, the animals were returned to the group housing container and placed in the DD light-tight box. Individuals were identified by unique patterns of dots on the pronotum made with white paint. Animals were given 12 minutes to complete each training trial (i.e., acquire the reward), with the exception of the initial training trial, in which an 8-minute limit was set to eliminate inactive animals. For each trial, the number of visits the animal made to the vanilla source before reaching the peppermint was recorded.

Data Analysis

The data were analyzed with Sigma Plot® statistical software (Version 11, Systat Software, Inc. 2007). The number of vanilla visits in each training trial relative to the initial training trial was used as a measure of learning, and was analyzed with a repeated measures one-way ANOVA, using the Holm-Sidak method as a post-hoc test. The mean number of vanilla visits for each training trial was compared against the mean number for the initial trial and tested to determine if there was a significant difference between the means at the $\alpha = 0.05$ level.

In order to compare groups, a learning index (LI) was developed to quantify the learning for each individual animal. The learning index was calculated by: $LI = (V_0 - V_T)/(V_0 + V_T)$ where V_0 is the number of initial vanilla visits, and V_T is the number of vanilla visits in a testing trial. A higher LI indicated better performance (maximum value of 1). An LI score of 0 indicated no change in odor preference. LIs were compared using a one-way ANOVA.

Author Contributions

Conceived and designed the experiments: TLP MVG SBS. Performed the experiments: MVG SBS. Analyzed the data: TLP MVG SBS. Wrote the paper: TLP.

References

1. Cain SW, Chou T, Ralph MR (2004) Circadian modulation of performance on an aversion-based place learning task in hamsters. Behav Brain Res 150: 201–205.
2. Cain SW, Ko CH, Chalmers JA, Ralph MR (2004) Time of day modulation of conditioned place preference in rats depends on the strain of rat used. Neurobiol Learn Mem 81: 217–220.
3. Manrique T, Molero A, Ballesteros MA, Moron I, Gallo M et al. (2004) Time of day-dependent latent inhibition of conditioned taste aversions in rats. Neurobiol Learn Mem 82: 77–80.
4. Valentinuzzi VS, Neto SPD, Carneiro BTS, Santana KS, Araujo JF et al. (2008) Memory for time of training modulates performance on a place conditioning task in marmosets. Neurobiol Learn Mem 89: 604–607.
5. Fernandez RI, Lyons LC, Levenson J, Khabour O, Eskin A (2003) Circadian modulation of long-term sensitization in *Aplysia*. Proc Natl Acad Sci USA 100: 14415–14420.
6. Lyons LC, Rawashdeh O, Katzoff A, Susswein AJ, Eskin A (2005) Circadian modulation of complex learning in diurnal and nocturnal *Aplysia*. Proc Natl Acad Sci USA 102: 12589–12594.
7. Decker S, McConnaughey S, Page TL (2007) Circadian regulation of insect olfactory learning. Proc Natl Acad Sci USA 104: 15905–15910.
8. Lyons LC, Roman G (2009) Circadian modulation of short-term memory in *Drosophila*. Learn Mem 16: 19–27.
9. Lehmann M, Gustav D, Galizia CG (2011) The early bee catches the flower - circadian rhythmicity influences learning performance in honey bees, *Apis mellifera*. Behav Ecol Sociobiol 65: 205–215.
10. Rawashdeh O, de Borsetti NH, Roman G, Cahill GM (2007) Melatonin suppresses nighttime memory formation in zebrafish. Science 318: 1144–1146.
11. Chaudhury D, Colwell CS (2002) Circadian modulation of learning and memory in fear-conditioned mice. Behav Brain Res 133: 95–108.
12. Valentinuzzi VS, Kolker DE, Vitaterna MH, Ferrari EAM, Takahashi JS et al. (2001) Effect of circadian phase on context and cued fear conditioning in C57BL/6J mice. Anim Learn Behav 29: 133–142.
13. Tapp WN, Holloway FA (1981) Phase-shifting circadian-rhythms produces retrograde-amnesia. Science 211: 1056–1058.
14. Fekete M, Vanrec JM, Niesink RJM, Dewied D (1985) Disrupting circadian-rhythms in rats induces retrograde-amnesia. Physiol Behav 34: 883–887.
15. Devan BD, Goad EH, Petri HL, Antoniadis EA, Hong NS, et al. (2001) Circadian phase-shifted rats show normal acquisition but impaired long-term retention of place information in the water task. Neurobiol Learn Mem 75: 51–62.
16. Wright KP, Hull JT, Hughes RJ, Ronda JM, Czeisler CA (2006) Sleep and wakefulness out of phase with internal biological time impairs learning in humans. J Cognitive Neurosci 18: 508–521.
17. Ruby NF, Hwang CE, Wessells C, Fernandez F, Zhang P et al. (2008) Hippocampal-dependent learning requires a functional circadian system. Proc Natl Acad Sci USA 105: 15593–15598.
18. Eckel-Mahan KL, Phan T, Han S, Wang HB, Chan GCK et al. (2008) Circadian oscillation of hippocampal MAPK activity and cAMP: implications for memory persistence. Nature Neurosci 11: 1074–1082.
19. Sakura M, Mizunami M (2001) Olfactory learning and memory in the cockroach Periplaneta americana. Zool Sci 18: 21–28.
20. Watanabe H, Kobayashi Y, Sakura M, Matsumoto Y, Mizunami M (2003) Classical olfactory conditioning in the cockroach *Periplaneta americana*. Zool Sci 20: 1447–1454.
21. Baxter DA, Byrne JH (2006) Feeding behavior of *Aplysia*: A model system for comparing cellular mechanisms of classical and operant conditioning. Learn Mem 13: 669–680.
22. Putz G, Bertolucci F, Raabe T, Zars T, Heisenberg M (2004) The S6KII (rsk) gene of *Drosophila melanogaster* differentially affects an operant and a classical learning task. J Neurosci 24: 9745–9751.

(Source 3)

Eur Arch Psychiatry Clin Neurosci (2016) 266:291–305
DOI 10.1007/s00406-015-0593-1

CrossMark

ORIGINAL PAPER

Classical conditioning in borderline personality disorder: an fMRI study

Classical Conditioning

Annegret Krause-Utz[1,2,3] · Jana Keibel-Mauchnik[1] · Ulrich Ebner-Priemer[1,4] · Martin Bohus[1] · Christian Schmahl[1]

Received: 16 May 2014 / Accepted: 15 March 2015 / Published online: 27 March 2015

Abstract Previous research suggests disturbed emotional learning and memory in borderline personality disorder (BPD). Studies investigating the neural correlates of aversive differential delay conditioning in BPD are currently lacking. We aimed to investigate acquisition, within-session extinction, between-session extinction recall, and reacquisition. We expected increased activation in the insula, amygdala, and anterior cingulate, and decreased prefrontal activation in BPD patients. During functional magnetic resonance imaging, 27 medication-free female BPD patients and 26 female healthy controls (HC) performed a differential delay aversive conditioning paradigm. An electric shock served as unconditioned stimulus, two neutral pictures as conditioned stimuli (CS+/CS−). Dependent variables were blood-oxygen-level-dependent response, skin conductance response (SCR), and subjective ratings (valence, arousal). No significant between-group differences in brain activation were found [all p(FDR) > 0.05]. Within-group comparisons for CS+$_{unpaired}$ > CS− revealed increased insula activity in BPD patients but not in HC during early acquisition; during late acquisition, both groups recruited fronto-parietal areas [p(FDR) < 0.05]. During extinction, BPD patients rated both CS+ and CS− as significantly more arousing and aversive than HC and activated the amygdala in response to CS+. In contrast, HC showed increased prefrontal activity in response to CS+ > CS during extinction. During extinction recall, there was a trend for stronger SCR to CS+ > CS in BPD patients. Amygdala habituation to CS+$_{paired}$ (CS+ in temporal contingency with the aversive event) during acquisition was found in HC but not in patients. Our findings suggest altered temporal response patterns in terms of increased vigilance already during early acquisition and delayed extinction processes in individuals with BPD.

Keywords Borderline personality disorder · Classical conditioning · Prefrontal cortex · Amygdala · Insula

Annegret Krause-Utz and Jana Keibel-Mauchnik have contributed equally to this work.

Electronic supplementary material The online version of this article (doi:10.1007/s00406-015-0593-1) contains supplementary material, which is available to authorized users.

✉ Christian Schmahl
christian.schmahl@zi-mannheim.de

1 Department of Psychosomatic Medicine and Psychotherapy, Medical Faculty Mannheim, Central Institute of Mental Health Mannheim, Heidelberg University, J 5, 68159 Mannheim, Germany

2 Institute of Psychology, Leiden University, Leiden, The Netherlands

3 Leiden Institute for Brain and Cognition, Leiden, The Netherlands

4 Department of Sport and Sport Science and House of Competence, KIT - Karlsruhe Institute of Technology, Karlsruhe, Germany

Introduction

Conditioning is a basic associative learning process that plays an important role in the development and maintenance of psychiatric disorders as well as in behavior therapy, especially exposure therapy.

Numerous studies have applied experimental fear conditioning paradigms to examine the (neurobiological) mechanisms underlying associative emotional learning [1–31]. In differential delay conditioning paradigms, one of the two initially neutral stimuli (CS+) is temporally paired with an aversive unconditioned stimulus (US), while the other is not (CS−). Many experimental settings do not only

involve *acquisition* (learning) and within-session extinction but also between-session extinction (*extinction recall*) and *reacquisition* or *reinstatement*, i.e., a renewed exposure to the original CS–US contingency.

There is convergent evidence from both animal research and human studies that the amygdala, insula, and medial prefrontal cortex (mPFC) including the anterior cingulate cortex (ACC) play central roles in the acquisition of fear responses [1, 4, 5, 9, 15]. Several studies in healthy humans further reported differential responses (to CS+ > CS−) in the inferior frontal gyrus, rostral, and caudal orbitofrontal cortex (OFC), thalamus, hippocampus, cerebellum, striatum (including putamen), as well as in sensory cortices [6, 10, 16, 18, 20, 22, 24–28]. The mPFC is assumed to play a central role in the integration of sensory and affective stimuli and in the coordination of memory storage both during the acquisition and the extinction of emotional (fear) memories [11, 14, 28, 31]. Extinction is thought to involve activity-dependent potentiation of synaptic transmission in the mPFC resulting in an inhibition of amygdala-dependent responses [11, 14]. Animals with lesions of the mPFC are unable to extinguish conditioned responses [4, 15]. In healthy humans, increased activation in the mPFC [20, 24, 26], OFC [17], amygdala (e.g., [7, 18, 20]), and insula [2, 17] could be observed during extinction (for a review see [28]). For the amygdala, a rapid habituation, i.e., decrease in reactivity to unpleasant stimuli was observed over the course of the acquisition phase [7, 8].

Within a 'brain network of emotion regulation,' the amygdala and insula have been critically implicated in the processing of negative emotions, while the ACC is thought to play a critical role in both the generation and regulation of emotions [9, 32]; prefrontal areas including the ventrolateral and dorsolateral PFC (vlPFC, dlPFC), OFC, and dorsomedial PFC are thought to be involved in the down-regulation of affective arousal [32].

In anxiety disorders, acquired fear responses to an initially neutral stimulus (e.g., place, object, person) previously paired with an US appears to be not successfully extinguished [33–35]. Patients with posttraumatic stress disorder (PTSD), for example, showed amygdala hyperreactivity and increased skin conductance responses to the CS+ during extinction recall [34]. Deficits in associative emotional learning are assumed to underlie clinical features such as a persistent fear and heightened affective arousal, even in contexts where no actual threat is present.

To our knowledge, no study so far has investigated the neural correlates of aversive differential delay conditioning in patients with borderline personality disorder (BPD). BPD is a severe psychiatric disorder with high rates of trauma and pronounced difficulties in emotion regulation including affective hyperarousal even in normative neutral situations [36, 37]. Experimental research in patients with BPD suggests altered learning and memory in aversive emotional contexts [38, 39]. On the neural level, there is growing evidence for both structural and functional alterations in fronto-limbic brain regions which play a major role in affecting regulation and learning processes (e.g., amygdala, hippocampus, anterior cingulate) including amygdala hyperactivity and diminished recruitment of the prefrontal areas during emotional challenge (for reviews see [40, 41]).

In a previous psychophysiological study [42], we investigated skin conductance responses (SCR) as well as arousal and valence ratings during an aversive differential delay conditioning paradigm in patients with BPD and healthy controls (HC). We additionally assessed levels of peri-experimental dissociation, i.e., disruptions of usually integrated functions of consciousness, memory, body awareness, and perception. In this previous study, BPD patients with low levels of dissociation showed regular conditioning responses in terms of a differential SCR to CS+ > CS− similar to HC. Of note, patients with high state dissociation failed to show regular conditioning, which suggests that dissociation may modulate emotional learning in BPD.

Recently, Kamphausen and colleagues [43] assessed SCR and brain activity during an instructed fear task in patients with BPD and HC during functional magnetic resonance imaging (fMRI). Before participants were brought to the MR scanner, they were informed that they would see different stimuli either indicating a safe situation or potential threat (electrodermal stimulation). The electrodermal stimulation was in fact only applied during this instruction but not during the experimental procedure. During fMRI, both healthy participants and BPD patients showed differential fear responses in terms of stronger SCR to stimuli indicating potential threat compared to stimuli indicating the save situation. However, only HC but not BPD patients showed amygdala habituation and an increase in activity in the mPFC during instructed fear. Findings of this fMRI study suggest that altered activity in a fronto-limbic brain circuitry might underlie disturbed emotional learning during an instructed fear task in patients with BPD.

The aim of the present study was to investigate the neural correlates of emotional learning in BPD patients applying an aversive differential (danger versus safety signal) delay conditioning paradigm during fMRI. Between-session extinction and re-acquisition were assessed after 72 h. Based on previous neuroimaging studies, we expected stronger activation in the amygdala and insula during acquisition and reacquisition and less activation in the mPFC during within-session and between-session extinction. We were further interested in habituation effects, i.e., decreases in activity over the course of each conditioning phase. Based on previous research [7, 8, 43], we expected a habituation of the amygdala during the acquisition phase.

Methods and materials

Participants

Thirty-four female individuals fulfilling DSM-IV criteria for BPD as assessed by the international personality disorder examination (IPDE) [44] and 32 female HC aged between 18 and 45 years participated in our study. Exclusion criteria for the patient group were a lifetime diagnoses of alcohol or substance dependence, psychotic disorder or bipolar I disorder, and current major depression as assessed by the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I) [45]. In addition, patients had to be free of psychotropic medication for at least 4 weeks prior to investigation. Exclusion criteria for the HC group were the presence of any current or lifetime axis I or axis II disorder, and current or past psychotherapy. Diagnostic interviews were administered by trained and experienced psychologists. Inter-rater reliability for BPD was $\kappa = 0.69$ for SCID-I (primary diagnosis) and $\kappa = 0.77$ for IPDE. Patients were recruited at the Central Institute of Mental Health, Department of Psychosomatic Medicine and Psychotherapy in Mannheim/Germany or via announcements on BPD-associated Web sites. The HCs were randomly selected from the resident register of the city of Mannheim.

All participants underwent diagnostic assessment using the SCID-I [45] and the IPDE [44]. To obtain a psychopathological characterization of the patient sample, trait dissociation by the German version of the Dissociative Experience Scale (Fragebogen zu Dissoziativen Symptomen, FDS; [46]) and global BPD symptom severity by the Borderline Symptom List (BSL-95; [47]) were assessed.

Demographic and clinical characteristics (trait dissociation, BPD symptom severity, comorbid diagnoses) of the full sample can be found in Supplemental Table 1. There were no significant differences regarding age [BPD (Mean ± SD): 27.6 ± 7.5; HC: 27.8 ± 7.5], sex (all female), and ethnicity (all Caucasian). All participants were right-handed. There was a statistical trend for significant differences in level of education. As expected, both groups differed significantly in clinical measures of trait dissociation and BPD symptom severity.

From this initial sample, one HC and five BPD patients dropped out of the study before completion of the experiment. From the remaining sample, a subset of neuroimaging data had to be discarded due to movement artifacts or technical problems during MR scanning. The final neuroimaging data set comprised data from 26 HC and 27 BPD patients for the first (conditioning) session, 22 HC and 21 BPD patients for extinction recall, and 17 HC and 24 BPD patients for reacquisition. Since some participants did not show an adequate skin conductance response (SCR) > 1 μS during the scanning procedure, SCR data of 21 HC and 21 BPD from the first (conditioning) session and SCR of 15 HC and 14 BPD from the second session (extinction recall, reacquisition) could be included into the final psychophysiology analysis.

Demographic and clinical characteristics (trait dissociation, BPD symptom severity) of all subsamples can be found in Supplemental Table 2 (subsamples neuroimaging dataset) and Supplemental Table 3 (subsamples SCR dataset). There were no significant differences regarding age and educational level in any subsample. As expected, within all subsamples, BPD patients reported significantly more trait dissociation and BPD symptom severity.

Experimental design

The experimental design was an aversive differential delay conditioning procedure which is depicted by Fig. 1. Dependent variables were the blood-oxygen-level-dependent (BOLD) signal, SCR, and valence/arousal ratings. The conditioned stimuli (CS) were two neutral graphic patterns (blue square and yellow triangle). The US consisted of unpleasant, but tolerable electrical stimulation to the right thumb. Stimulus duration was 5.8 s (s), and the unconditioned stimulus (US) lasted for 2.8 s and was administered 3 s after CS onset. The inter-trial interval (ITI) lasted between 8 and 12 s, randomly generated. CS+ (CS paired with the US) and CS− (non-reinforced CS) were counterbalanced across participants and presented in pseudorandom order with the constraint of a maximum of two consecutive presentations of the CS+ (danger signal) or CS− (safety signal).

The US (electrical stimulation) was delivered through a copper electrode of an electrical stimulus generator (Digitimer, DS7A, Wewyn Garden City, UK). Before the experiment, for each participant, the level of pain stimulation was individually determined. To this end, participants were given a series of stimuli, starting with a mild stimulus, which was gradually increased to (1) detection threshold, (2) pain threshold, and (3) pain tolerance. This procedure was repeated three times. Finally, we delivered stimuli that were 80 % above pain threshold. Participants were asked to rate the intensity of pain on a Likert scale ranging from 0 = not painful to 10 = extremely painful. For the experiment, we used stimulation intensities that were rated with at least 7/10. Therefore, the experimental level of pain was objectively different, but subjectively both groups received the same level of painful stimulation. This procedure was chosen due to the fact of higher pain threshold in BPD [48, 49].

The first session (conditioning session) started with a phase in which participants were familiarized with the CS+ and CS− (*'familiarization'*: 10 CS+, 10 CS−). During the following *acquisition* phase, 18 CS+ paired with the US

Table 1 Brain activity during the different conditioning phases in healthy controls (HC) and in patients with borderline personality disorder (BPD)

Conditioning phase	Group	T contrast	MNI coordinates (*X*, *Y*, *Z*)	*K*	Hemisphere	Label (Brodmann area)	Lobe	*T* value	*Z* value	*p* value
Early acquisition	HC	CS+	0, −66, 12	110	R	Posterior cingulate	Limbic	6.12	4.71	0.037
			−27, −48, −15	49	L	Fusiform gyrus (BA37)	Occipital	6.25	4.77	0.021
			−18, −9, −12*	53	L	Parahippocampal gyrus/uncus/amygdala	Limbic	5.33	4.03	<0.05, ROI
			−24, −3, −24*		L		Limbic	4.83	3.78	
			18, −15, −15*	33	R	Parahippocampal gyrus (BA28)	Limbic	4.83	3.78	<0.05, ROI
		CS+ > CS−	No significant clusters at p(FDR) < 0.05							
	BPD	CS+	No significant clusters at p(FDR) < 0.05							
		CS+ > CS−	33, 39, 15	178	R	Middle frontal gyrus	Frontal	7.26	5.28	0.003
			27, 51, 3		R	superior frontal gyrus	frontal	4.17	3.60	0.003
			−60, 3, 3	49	L	Superior temporal gyrus	Temporal	5.29	4.30	0.022
			−66, −27, 12	61	L	Superior temporal gyrus (BA42)	Temporal	4.90	4.07	0.035
			66, −33, 18	89	R	Superior temporal gyrus (BA42)	Temporal	4.84	4.03	0.036
			−33, 15, 3*	16	L	Insula	Sub-lobar	4.32	3.70	<0.05, ROI
			−39,−3,−9*		L			3.63	3.22	<0.05, ROI
Late acquisition	HC	CS+	48, 33,−6*	34	R	Inferior frontal gyrus (BA47)	Frontal	4.80	4.00	<0.05, ROI
		CS+ > CS−	−30, −66, −51	1716	L	Inferior semilunar lobule	Cerebellum	6.32	4.84	0.004
			−33, −78, −30			declive	posterior	6.14	4.75	0.004
			−39, −63, −42			cerebellar tonsil		5.91	4.63	0.004
			45, −48, 51	606	R	Inferior parietal lobule (BA40)	Parietal	6.16	4.76	0.004
			63, −42, 24					5.59	4.46	0.004
			48, −45, 30					5.35	4.33	0.004
			54, 21, 33	178	R	Middle frontal gyrus (BA46)	Frontal	5.62	4.48	0.004
			54, 30, 18			inferior frontal gyrus (BA46)	frontal	4.27	3.67	0.008
			42, 6, 57			inferior frontal gyrus		4.26	3.66	0.008
			−60, −45, 24	216	L	Inferior parietal lobule (BA40)	Parietal	5.29	4.29	0.004
			−42, −51, 33					4.51	3.82	0.006
			−33, −51, 36					4.38	3.74	0.007
			39, 42, 30	35	R	Middle frontal gyrus	Frontal	4.95	4.10	0.005
			48, 27, −9*	85	R	Inferior frontal gyrus (BA47)	Frontal	4.93	4.08	<0.05, ROI
			−54, 21, 0*	41	L	Inferior frontal gyrus (BA47)	Frontal	4.22	3.63	<0.05, ROI
			−51, 15, −6*				frontal	4.16	3.59	<0.05, ROI
	BPD	CS+	48, 15, 45	49	R	Middle frontal gyrus (BA8)	Frontal	6.76	5.09	0.011
			39, 57, 3	98	R	Middle frontal gyrus (BA8)	Frontal	5.88	4.65	0.027
			60, −24, −24	32	R	Inferior temporal gyrus	Temporal	5.36	4.36	0.028
			−27, −66, −30	158	L	Pyramis	Cerebellum	5.15	4.24	0.030
			−57, −36, −6	38	L	Middle temporal gyrus	Temporal	4.83	4.04	0.040
			6, 39, 42	87	R	Medial frontal gyrus (BA8)	Frontal	5.06	4.18	0.040

Table 1 continued

Conditioning phase	Group	T contrast	MNI coordinates (*X*, *Y*, *Z*)	*K*	Hemisphere	Label (Brodmann area)	Lobe	*T* value	Z value	*p* value
		CS+ > CS−	48, 15, 45	208	R	Middle frontal gyrus (BA8)	Frontal	7.08	5.55	0.001
			48, 39, 30		R	Middle frontal gyrus (BA46)	frontal	4.44	3.80	0.017
			−18, −102, 3	831	L	Middle occipital gyrus (BA18)	Occipital	6.29	4.86	0.004
			39, 57, 3	120	R	Middle frontal gyrus (BA9)	Frontal	5.99	4.71	0.004
			30, 12, 3	46	R	Inferior frontal gyrus	Frontal	4.93	4.11	0.012
			33, 21, −9		R			3.89	3.42	0.027
			60, −42, 42	58	R	Inferior parietal lobule (BA40)	Parietal	4.51	3.84	0.016
			6, 36, 42	32	R	Medial frontal gyrus (BA8)	Frontal	4.44	3.79	0.017
			−45, 54, −3	45	L	Middle frontal gyrus (BA10)	Frontal	4.39	3.77	0.018
Extinction	HC	CS+	48, 48, −3*	R	18	Middle frontal gyrus	Frontal	5.57	4.45	<0.05, ROI
			−39, 15, 45*	L	31	Middle frontal gyrus	Frontal	5.01	4.13	<0.05, ROI
			9, 36, 24*	R	57	Orbito frontal gyrus	Frontal	4.86	4.04	<0.05, ROI
			33, 63, 18*					4.63	4.63	<0.05, ROI
			21, 63, 21*					3.87	3.87	<0.05, ROI
			−45, 39, −9*	L	20	Inferior frontal gyrus (BA 47)	Frontal	4.54	3.84	<0.05, ROI
			−27, 57, 24*	L	15	Middle frontal gyrus	Frontal	4.23	3.64	<0.05, ROI
			45, 48, 27*	R	14	Middle frontal gyrus	Frontal	4.13	3.57	<0.05, ROI
			51, 45, 21*	R		middle frontal gyrus	frontal	4.12	3.56	<0.05, ROI
			42, 36, 24*	R	9	Middle frontal gyrus	Frontal	4.07	3.53	<0.05, ROI
		CS+ > CS−	24, 66, 21*	R	28	Superior frontal gyrus (BA10)	Frontal	3.96	3.45	<0.05, ROI
			15, 63, 24*	R		Superior frontal gyrus	frontal	3.90	3.41	<0.05, ROI
			−45, 21, 45*	L	7	Middle frontal gyrus	Frontal	3.83	3.36	<0.05, ROI
	BPD	CS+	45, 42, −12*	R	99	Orbitofrontal gyrus (BA11)	Frontal	4.76	4.00	<0.05, ROI
			30, −6, −12*	R	5	Amygdala	Limbic	3.90	3.43	<0.05, ROI
		CS+ > CS−	No significant clusters p(FDR) < 0.05							
Extinction recall	HC	CS+	12, −63, 0	2235	R	Lingual gyrus	Occipital	10.14	6.12	<0.001
			6, −78, 15		R	lingual gyrus	Occipital	9.14	5.82	<0.001
			−12, −84, 3		L	cuneus (BA 17)	occipital	7.16	5.09	<0.001
			−36, 42, −9	17	L	Middle frontal gyrus	Frontal	5.00	4.04	0.002
			−39, −63, −39	31	L	Tuber	Cerebellum	4.79	3.92	0.003
			51, −48, 3	33	R	Middle temporal gyrus	Temporal	3.80	3.30	0.016
			63, −51, −9		R	middle temporal gyrus (BA21)	Temporal	3.76	3.27	0.017
			−27, −3, −27	10	L	Parahippocampal gyrus	Limbic	4.38	3.67	0.006
			−24, 51, 39	4	L	Superior frontal gyrus (BA9)	Limbic	3.86	3.34	0.014
			−36, −24, −15	6	L	Parahippocampal gyrus	Limbic	3.80	3.30	0.016
		CS+ > CS−	No significant clusters $p < 0.05$							

Table 1 continued

Conditioning phase	Group	T contrast	MNI coordinates (*X*, *Y*, *Z*)	*K*	Hemisphere	Label (Brodmann area)	Lobe	*T* value	Z value	*p* value
	BPD	CS+	12, −72, 9	449	R	Cuneus (BA17)	Occipital	6.78	4.78	0.032
			45, 9, −9*	49	R	Insula (BA38)	Sub-lobar	4.34	3.58	0.05, ROI
			−27, −3, −27	3	L	Parahippocampal gyrus	Limbic	4.38	3.67	0.009
		CS+ > CS−	24, 39, −18*	7	R	Orbitofrontal gyrus (BA11)	Frontal	4.90	3.89	<0.05, ROI
			6, 18, 51*	2	R	medial frontal gyrus (BA8)	Frontal	4.00	3.37	<0.05, ROI
Re-acquisition	HC	CS+	51, −45, 18	13705	R	Superior temporal gyrus,	Temporal,	11.53	6.01	<0.001
			−51, −24, 24		L	insular cortex (BA13)	Sub-lobar	11.09	5.91	<0.001
			−66, −27, 30		L			9.78	5.60	<0.001
			48, 12,0*					8.88		<0.05, ROI
			30, 57, 18	219	R	Superior frontal gyrus/middle frontal gyrus (BA9,	Frontal	7.78	5.02	<0.001
			27, 51, 24		R	BA10)	Frontal	6.88	4.70	<0.001
			42, 54, 12		R		Frontal	6.22	4.43	<0.001
					R					
			−39, 45, 24	74	L	Middle frontal gyrus (BA10, BA9)	Frontal	4.94	3.84	0.001
			−27, 51, 30		L			4.90	3.82	0.001
			−48, 45, 15		L			4.74	3.73	0.001
			−30, 3, −18*	45	L	Parahippocampal gyrus (BA34)	Limbic	5.10	3.92	<0.05, ROI
			−24, 0, −24*		L	amygdala/uncus	Limbic	5.14	3.94	<0.05, ROI
		CS+ > CS−	−3, 51, −6*	151	L	Medial frontal gyrus (BA10)	Frontal	6.80	4.28	<0.05, ROI
			0, 39, −18*		R			5.67	3.88	<0.05, ROI
			12, 45, −12*		R			4.98	3.60	<0.05, ROI
			−21, 42, 45*	L	19	Superior frontal gyrus	Frontal Frontal	5.18	3.68	<0.05, ROI
			−15, 42, 39*			superior frontal gyrus		5.03	3.62	<0.05, ROI
			−27, 24, 51*	10	L	Middle frontal gyrus (BA8)	Frontal	4.59	3.42	<0.05, ROI
	BPD	CS+	9, 3, 39*	19*	R*	Anterior cingulate (BA24)	Limbic	4.64	3.84	<0.05, ROI
			39, 54, −15*	16*	R*	Orbitofrontal gyrus (BA11)	Frontal	4.38	3.67	<0.05, ROI
		CS+ > CS−	36, 51, 12*	10	R	Middle frontal gyrus (BA10)	Frontal	4.28	3.61	<0.05, ROI

Clusters were determined applying an extended threshold of $p < 0.05$ FDR corrected on the whole-brain voxel-wise level. Clusters indicated by an *asterisk* were detected by region of interest (ROI) analyses with predefined anatomical masks. Sizes of subsamples were 26 for session 1 (familiarization, acquisition, extinction), 22 for extinction recall, and 17 for re-acquisition. *Note L* left, *R* right, *k* cluster size of contiguous voxels, *MNI* Montreal Neurological Institute, *R/L* right/left, *BA* Brodmann area, *CS+* conditioned stimulus that was sometimes paired with the unconditioned stimulus (US) but here only in absence of the US, *CS−* conditioned stimulus never paired with US

and 36 CS− never paired with the US were presented. Furthermore, 18 CS+ were presented but not paired with the US (catch trials). Catch trials were used to differentiate between BOLD responses to the CS+ and to the pain stimulus (US). During the *extinction* phase, 18 CS+ and 18 CS− were presented. The second session (reacquisition session) took place 72 h later and consisted of the *extinction recall* phase (18 CS+ and 18 CS−) and a *reacquisition* phase (18 CS−, 9 CS+, 9 CS+/US).

Apparatus and physiological recordings

Stimulus delivery was controlled by a PC running Presentation software (Neurobehavioral Systems, San Francisco). Physiological data acquisition was controlled by an Ibook running Vitagraph version 4.61 (Becker Meditec, Karlsruhe, Germany). Physiological channels were recorded at a rate of 256 Hz in continuous mode using the Vitaport II system (Becker). SCR was obtained from 10-mm (sensor diameter) Ag/AgCl electrodes (Marquette Hellige GmbH, Freiburg, Germany) filled with an isotonic EDR jelly TDE-246 (Steffens, Berlin, Germany) and placed on the thenar and hypothenar of the non-dominant hand (constant voltage method with 0.5 V).

Procedure

The study was approved by the ethical review committee of the University of Heidelberg, Germany, in accordance with the Declaration of Helsinki. All subjects gave written informed consent after receiving a description of the study and scanning procedure. All participants were paid for study participation (10 €/h). The experiment was conducted between 2007 and 2009 at the Central Institute of Mental Health in Mannheim, Germany.

All participants underwent diagnostic assessments including the SCID-I and IPDE by trained diagnosticians and completed clinical questionnaires (FDS, BSL) as described above. The conditioning procedure took place on two different days within a fixed time interval. On a first day, the conditioning session (familiarization phase, acquisition phase, extinction phase) was conducted during fMRI. Immediately before the experiment, the level of pain stimulation was individually determined for each participant as described in detail above. The reacquisition session (extinction recall phase and reacquisition phase) took place 72 h later in the same MR scanner.

Throughout the scanning procedure, participants viewed stimuli presented on a screen by a projector via a mirror mounted on the head coil. Participants were not informed about CS–US contingencies and were told to passively view the stimuli. After each phase (familiarization acquisition, extinction, extinction recall, and reacquisition), participants rated the valence and arousal of the CSs to test awareness of the CS–US contingency. In addition, participants rated their acute dissociation on scales ranging from 1 = very calm to 9 = very arousing, 1 = very pleasant to 9 = very unpleasant, and 1 = no dissociative symptoms to 9 = very intense dissociative symptoms. Valence/arousal ratings of the CS were obtained by presenting the CS together with a visual analogue scale, and patients used a keypad to move the cursor and to choose a number.

fMRI assessment

fMRI was conducted by a Siemens MAGNETOM Vision 1.5 Tesla whole body scanner (Siemens Medical Solution, Erlangen, Germany). Head movement artifacts and scanning noise were restricted using head cushions and headphones within the scanner coil. To acquire BOLD signals (T2-weighted echo planar imaging, EPI), a standard protocol (see also [27]) with the following parameters was used: repetition time (TR): 3.77 s, echo time (TE): 45 ms; flip angle: 90°, matrix: 64 × 64, slice thickness: 3 mm, slice gap: 1 mm, FOV: 220 × 220 mm, and number of slices: 35. There were 100 images for familiarization, 320 for acquisition, and 160 for all other phases.

Data analysis

Psychometrics

Means and standard deviations of the BSL and the FDS were analyzed using t tests. For group comparisons, significance levels were set to $p < 0.05$. All analyses were conducted with SPSS (Version 22.0 for Windows; SPSS Inc., Chicago, IL).

Ratings

To compare valence and arousal ratings, repeated-measures analyses of covariance (rm-ANCOVA) were performed for the mean of each phase with group (HC, BPD) as between-subject factor, stimulus type (CS+, CS) as within-subjects factor, and present-state dissociative experience as covariate. A Greenhouse-Geisser correction was employed in case the sphericity assumption was not met. Statistical analysis was performed using SPSS 22.0 (SPSS Inc, Chicago, IL). An alpha level of $p < 0.05$ determined statistical significance.

Skin conductance response (SCR)

The electrodermal activity was determined according to published guidelines [50] with the program EDR-Para (Schäfer, Wuppertal). Phasic SCRs were defined as the response

magnitude (maximum deflection) within a 1- to 4-s time frame (first interval response, FIR). SCRs lower than 0.05 μS were scored as zero. The FIR was counted as missing when the SCR was clearly initiated prior to CS onset. If such an anticipatory response was superimposed on a stimulus-related response, the scoring method (B) as described by Boucsein ([50], p. 136) was used. SCRs were log-transformed to reduce skewness [50]. To compare SCR to CS+ and CS− type × group, rm-ANCOVAs were performed for the mean of each phase with present-state dissociative experience as covariate. A Greenhouse-Geisser correction was employed if the sphericity assumption was not met. Statistical analysis was performed using SPSS 22.0 (SPSS Inc, Chicago, IL). An alpha level of <0.05 determined statistical significance.

fMRI data

Functional data were analyzed with SPM5 (Welcome Department of Imaging Neuroscience, University College London, UK, 2005). The first four images at the beginning of each trial were discarded to enable the signal to achieve steady-state equilibrium between radiofrequency pulse and relaxation.

Within preprocessing, the EPI time series were realigned and unwarped to the mean to correct for intra-subject's head movements. Mean images were normalized to an MNI (Montreal Neurological Institute, www.bic.mni.mcgill.ca) echoplanar imaging template with affine registration followed by nonlinear transformation with 25 mm cutoff, medium regularization and 16 iterations, resampled with trilinear interpolation, and written in 3 × 3 × 3 mm^3 isotropic voxels. The normalization parameters determined for the mean functional volume were then applied to the corresponding functional image volumes for each participant. Finally, images were smoothed with a Gaussian kernel of 8 mm full-width at half-maximum. The data were high-pass filtered (1/128 Hz cutoff) to remove low-frequency signal drifts.

The consecutive statistical analyses of the fMRI data relied upon the general linear model (GLM) to model effects of interest as implemented in SPM5. For each subject, the following events of interest were defined as regressors of interest: US, CS−, $CS+_{paired}$, and $CS+_{unpaired}$ for each phase of the conditioning paradigm: familiarization, acquisition, extinction, extinction recall, and reacquisition phase.

In order to assess the temporal course of brain activation, we split up the acquisition phase (of the first conditioning session) into an early and late acquisition phase. In addition, we investigated habituation effects during acquisition by modeling linear time effects for $CS+_{unpaired}$, $CS+_{unpaired} > CS-$, and $CS+_{paired}$ (CS paired with US) using the time modulation function as implemented in SPM ($p < 0.05$).

Task-related activity (BOLD response) for each event was modeled by convolving a vector of the event onset times with a canonical hemodynamic response function (HRF). The regressors of interest were included together with time as covariate in a GLM to yield parameter estimates for brain activation related to the different event types (US, CS−, $CS+_{paired}$, $CS+_{unpaired}$). T contrast images for $CS+_{paired} > CS-$ and $CS- > CS+_{paired}$ were computed for each conditioning phase (familiarization, early acquisition, late acquisition, extinction, extinction recall, reacquisition) separately.

At the second level (group level), the resulting first level contrast images were entered into random effect models: *Within-group* (one sample) *t* tests were used to compare brain activity during CS+ to brain activity in response to CS−. *Between-group* (two sample) *t* tests were used to analyze group differences in response to CS+, CS−, as well as $CS+_{unpaired} > CS-$ during the different conditioning phases. Clusters were determined using an extent threshold of $p < 0.05$ on the voxel-wise whole-brain level. The false discovery rate (FDR) correction was applied to correct for multiple comparisons.

Region of interest analyses Based on our a priori hypotheses (stronger activation in the amygdala and insula during acquisition and reacquisition and less activation in the mPFC during within-session and between-session extinction in BPD patients than HC), regions of interest analyses (ROIs) were performed for the following brain areas: amygdala, ACC, hippocampus, insula, OFC, dfPFC, and vmPFC. Anatomically based ROIs of these regions were created using the Masks for Regions of Interest Analysis (MARINA) software program (Bertram Walter, Bender Institute of Neuroimaging, University of Giessen, Germany). These masks were then used for small volume corrections.

In a separate model, state dissociation ratings were included as a covariate in order to test the effect of this

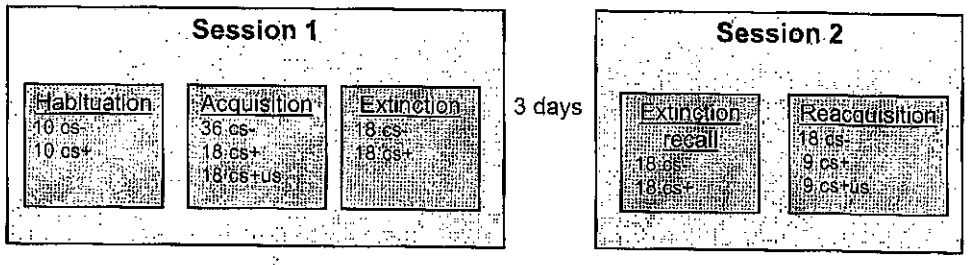

Fig. 1 Experimental design: Ratings of valence and arousal were performed after each of the five experimental phases. *CS+* conditioned stimulus paired with the US; *CS−* non-reinforced conditioned stimulus, *US* unconditioned stimulus

variable on the results. Furthermore, a subgroup analysis with BPD patients with versus without current PTSD compared to HC was computed.

Results

Ratings

Means and standard deviations of arousal (a) and valence (b) ratings during acquisition, extinction, extinction recall, and reacquisition in HC and BPD patients are depicted in Fig. 2.

For arousal ratings after acquisition, a significant effect of type [$F(1,34) = 23.929$, $p < 0.001$] but no significant interaction with group was found [$F(1,34) = 0.001$, $p = 0.973$]: All participants showed a higher rating of the CS+ compared to the CS−. For the arousal ratings after extinction and extinction recall, no significant effect of type [$F(1,30) = 0.250$, $p = 0.621$; $F(1,24) = 0.566$, $p = 0.459$] and no significant interaction with group were found [$F(1,30) = 0.009$, $p = 0.925$, $F(1,24) = 0.452$, $p = 0.508$]. After extinction, BPD patients rated both stimuli as more aversive than the HC indicated by a significant effect of group [$F(1,30) = 5.565$, $p = 0.025$].

For the arousal ratings after reacquisition, a significant effect of type [$F(1,24) = 18.315$, $p < 0.001$] and no significant interaction with group were found [$F(1,24) = 0.337$, $p = 0.567$]: All participants showed a more aversive rating of the CS+ compared to the CS− (see Fig. 2a).

For the valence ratings after acquisition, a significant effect of type [$F(1,34) = 15.199$, $p < 0.001$], with more aversive rating of the CS+ than the CS−, and a significant interaction effect type × group [$F(1,34) = 4.223$, $p = 0.048$] were found. For the valence ratings after extinction and extinction recall, no significant effect of type [$F(1,30) = 0.334$, $p = 0.568$; $F(1,24) = 1.189$, $p = 0.286$] and no interaction with group were found [$F(1,30) = 0.021$, $p = 0.886$, $F(1,24) = 1.156$, $p = 0.293$]. After extinction, again, a significant effect of group was found for the valence ratings [$F(1,30) = 14.944$, $p = 0.001$]: BPD patients rated both stimuli as more aversive than the HC. For the valence ratings after reacquisition, a significant effect of type [$F(1,24) = 14.138$, $p = 0.001$] but no interaction with group was found [$F(1,24) = 0.199$, $p = 0.660$]: All participants showed a more aversive rating of the CS+ compared to the CS− (see Fig. 2b).

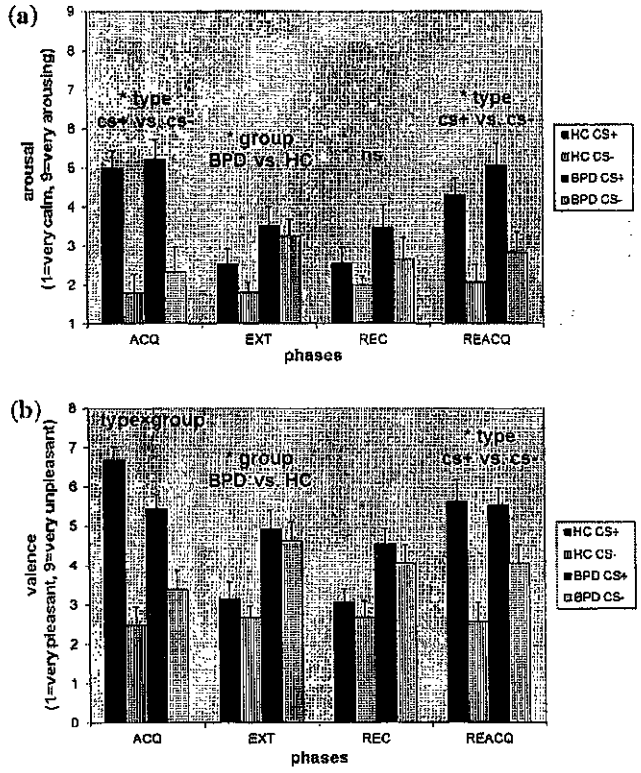

Fig. 2 Results of the arousal (**a**) and valence (**b**) ratings. *ACQ* acquisition, *EXT* extinction, *REC* extinction recall, *REACQ* reacquisition, *HC* healthy controls, *BPD* BPD patients, *CS+* conditioned stimulus paired with the US, *CS−* non-reinforced conditioned stimulus, significant at $^{*}p < 0.05$, $^{\#}p < 0.10$

Skin conductance response (SCR)

Means and standard deviations of SCR during acquisition, extinction, extinction recall, and reacquisition in HC and BPD patients are shown in Fig. 3. During acquisition, no significant effect of type [$F(1,34) = 2.291$, $p = 0.139$, $\eta^2 = 0.063$] and no significant interaction with group were found [$F(1,34) = 0.039$, $p = 0.844$]. During extinction, no significant interaction with group was found [$F(1,33) = 0.962$, $p = 0.334$], but a significant effect of type [$F(1,33) = 9.895$, $p = 0.003$] with participants shows stronger responses to the CS+ than to the CS−. During extinction recall, no significant effect of type [$F(1,20) = 0.209$, $p = 0.652$] and a trend for the interaction effect type x group were found [$F(1,20) = 3.194$, $p = 0.089$], with BPD patients showing descriptively a stronger response to CS+ compared to the CS−, whereas HCs showing descriptively a stronger response to CS− compared to the CS+. During reacquisition, no significant effect of type [$F(1,20) = 1.967$, $p = 0.176$, $\eta^2 = 0.090$] and no interaction with group were found [$F(1,20) = 1.768$, $p = 0.199$, $\eta^2 = 0.081$].

fMRI

Between-group t tests

No significant group differences on the whole-brain level could be detected at a threshold level of $p < 0.05$ after correction for multiple comparisons (FDR correction).

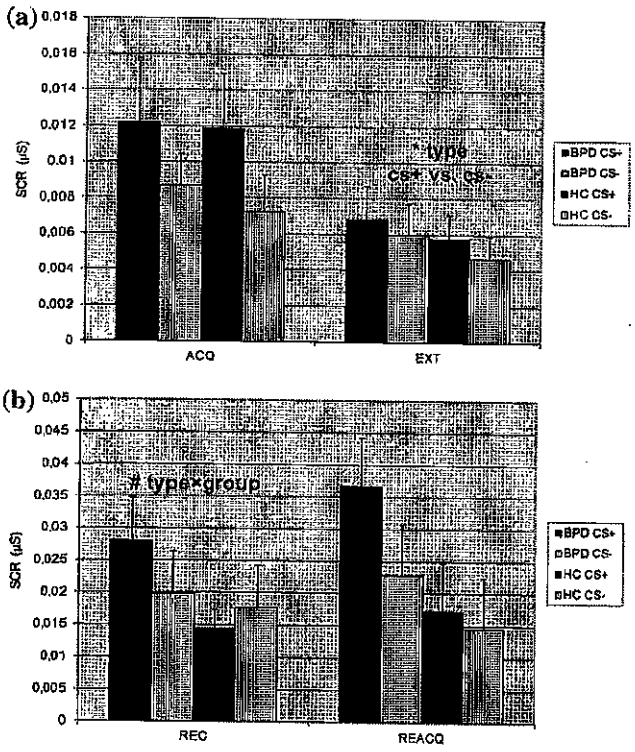

Fig. 3 Skin conductance response. *Note ACQ* acquisition, *EXT* extinction, *REC* extinction recall, *REACQ* reacquisition, *HC* healthy controls, *BPD* BPD patients, *CS+* conditioned stimulus paired with the US, *CS−* non-reinforced conditioned stimulus

Within-group t tests

Results for brain activity in response to $CS+_{unpaired}$ and $CS+_{unpaired} > CS-$ can be found in Table 1. In the following, results are reported for each group and for each conditioning phase separately [p(FDR) < 0.05].

Healthy control group

During *early acquisition*, no significant clusters were observed for the $CS+_{unpaired} > CS-$ contrast. During presentation of the $CS+_{unpaired}$, HC recruited the fusiform gyrus (BA37) and right posterior cingulate. Our ROI analyses further revealed significant $CS+_{unpaired}$-related activation in the bilateral parahippocampal gyrus and left amygdala.

During *late acquisition*, HC showed significant stronger activation in response to the $CS+_{unpaired}$ than to CS− in the bilateral inferior frontal gyrus (BA46 and BA47), bilateral inferior parietal lobule, right middle frontal gyrus (BA46), and left cerebellum.

During *extinction*, HC exhibited significant differential activation for $CS+_{unpaired} > CS-$ in the right superior frontal gyrus (BA10) and left middle frontal gyrus (clusters revealed by ROI analyses). In addition, our ROI analyses revealed significant $CS+_{unpaired}$-related activation in the right OFC, left inferior frontal gyrus (BA47), and bilateral middle frontal gyrus.

During *extinction recall*, no significant clusters were found for the $CS+_{unpaired} > CS-$ contrast. In response to the $CS+_{unpaired}$, HC activated the right lingual gyrus and left cuneus, the left middle frontal gyrus and superior frontal gyrus (BA9), the left parahippocampal gyrus, right middle temporal gyrus, and left tuber (cerebellum).

During *reacquisition*, our ROI analyses revealed significant clusters in the bilateral medial frontal gyrus (BA10), left superior frontal gyrus, and left middle frontal gyrus (BA8) for the $CS+_{unpaired} > CS-$ contrast. In response to the $CS+_{unpaired}$, HC showed recruitment of the bilateral superior temporal gyrus and insular cortex (BA13), bilateral middle frontal, and superior frontal. For the $CS+_{unpaired}$ contrast, ROI analyses additionally revealed significant clusters in the left amygdala and left parahippocampal gyrus.

BPD group

During *early acquisition*, BPD patients showed significant differential activation for $CS+_{unpaired} > CS-$ in the right superior and middle frontal gyrus as well as in the bilateral superior temporal gyrus. ROI analyses further revealed significant stronger activation in the left insula (see Fig. 4a) for the $CS+_{unpaired} > CS-$ contrast.

During *late acquisition*, BPD patients exhibited significant stronger activation in response to $CS+_{unpaired} > CS-$ in the bilateral middle and medial frontal gyrus (BA8, BA9, BA10, BA46), right inferior frontal gyrus, left middle occipital gyrus (BA18), and right inferior parietal lobule (BA40). In response to the $CS+_{unpaired}$, in addition, patients recruited the right inferior temporal gyrus and left middle temporal gyrus.

During the *extinction* phase, no significant clusters were observed for the $CS+_{unpaired} > CS-$ contrast. In response to $CS+_{unpaired}$, BPD patients activated the right orbitofrontal gyrus (BA 11) and right amygdala (see Fig. 4b).

During *extinction recall*, BPD patients exhibited $CS+_{unpaired}$-related activation in the right cuneus and left parahippocampal gyrus as well as in the right insula. For the $CS+_{unpaired} > CS-$ contrast, ROI analyses revealed stronger activation in the orbitofrontal gyrus (BA11) as well as medial frontal gyrus (BA8) in the patient group.

During *reacquisition*, we found significant clusters in the middle frontal gyrus (BA10) for the $CS+_{unpaired} > CS-$ contrast as well as clusters in the orbitofrontal gyrus (BA11) and ACC (BA24) for the $CS+_{unpaired}$ contrast.

Linear habituation

Complete results for linear time effects during the acquisition phase in each group can be found in Supplemental Table 4.

(SOURCE 4)

Person-Centered & Experiential Psychotherapies
Vol. 10, No. 3, September 2011, 198–209

Routledge
Taylor & Francis Group

Operant Conditioning

Bridging the positions of Rogers and Skinner: The role of nonlinear dynamic systems

Lisbeth Sommerbeck*

Clinical psychologist, Bornholm Psychiatric Center, Denmark

(*Received 11 December 2010; final version received 21 March 2011*)

This paper aims to bridge the gap between the positions of Carl Rogers and B. F. Skinner by demonstrating that they are not as antithetical as they are ordinarily assumed to be, and as the two men themselves tended to think. The author uses the concept of nonlinear dynamic systems as the principal concept in her effort to bridge the gap between the work of Rogers and Skinner and consequently introduces the concept of nonpredictivity to replace the concept of nondirectivity as being a more correct and basic characterization of classical client-centered therapy. The concept of nonpredictivity allows client-centered therapy to be framed, in an overarching and general way, by B. F. Skinner's principle of operant conditioning without doing violence to Rogers' concern about client autonomy. More specifically, the paper argues that Rogers' theory of therapy is not antithetical to Skinner's theory of personality and that client-centered therapy therefore can be practiced seamlessly by therapists who, like the author, believe that operant conditioning is a better explanatory concept for human behavior than the actualizing tendency.

Keywords: Rogers and Skinner; nonlinear dynamic systems; nonpredictive; nondirective

Brückenschlag zwischen den Positionen von Rogers und Skinner: die Rolle nicht-linearer dynamischer Systeme

Dieser Artikel will die Kluft zwischen den Positionen von Rogers und Skinner schliessen. Diese sind nicht so anti-thetisch, wie man normalerweise annimmt und wie die beiden selbst meinten. Ein Konzept nicht-linearer dynamischer Systeme soll die Kluft zwischen Rogers' und Skinners' Werk schliessen. Das Konzept der Nicht-Vorhersagbarkeit soll das Konzept der Nicht-Direktivität ersetzen; ersteres ist eine korrektere und grundlegendere Charakterisierung der klassischen Klientzentrierten Therapie. Das Konzept der Nicht-Vorhersagbarkeit ermöglicht es, Klientzentrierte Therapie in umfassender und genereller Weise in Skinners Prinzip des Operanten Konditionierens einzubetten, ohne dass man Rogers' Sorge um die Autonomie der Klienten Gewalt antut. Rogers' Therapietheorie ist keine Antithese zu Skinners Theorie der Persönlichkeit und Klientzentrierte Therapie kann daher nahtlos von Therapeuten praktiziert werden, die wie die Autorin überzeugt sind, dass Operantes Konditionieren ein besseres Erklärungskonzept für menschliches Verhalten ist als die Aktualisierungstendenz.

*Email: lisbethsommerbeck@privat.dk

ISSN 1477-9757 print/ISSN 1752-9182 online

DOI: 10.1080/14779757.2011.599516
http://www.informaworld.com

Estableciendo un puente entre las posturas de Rogers y Skinner: El rol de los sistemas dinámicos ni lineales

Este escrito tiene como fin establecer un puente entre las posiciones de Carl Rogers y B. F. Skinner al demostrar que no son tan antagónicas como normalmente se las considera, y como ambos autores tendían a considerarlas. La autora utiliza el concepto de sistemas dinámicos no lineares como el concepto principal en su esfuerzo para salvar la distancia entre la obra de Rogers y la de Skinner, y por ello introduce el concepto de no predictibilidad para reemplazar el concepto de no directividad, considerándolo una caracterización más exacta del la terapia centrada en el cliente clásica. El concepto de no predictibilidad permite que ubiquemos la terapia centrada en el cliente en un marco más abarcativo y general, por el principio de Skinner de condicionamiento operativo sin violentar la preocupación de Rogers acerca de la autonomía del cliente. Más específicamente, este escrito argumenta que la teoría de Rogers acerca de la terapia no es contraria a la teoría de la personalidad de Skinner y que por lo tanto la terapia centrada en el cliente puede ser aplicada por terapeutas, como la autora, creen que el condicionamiento operante es un concepto que explica el comportamiento humano mejor que la tendencia actualizante.

L'élaboration d'un pont entre les positions de Rogers et de Skinner: Le rôle de systèmes non-linéaires et dynamiques

Cet article vise à créer un pont entre les positions de Carl Rogers et B.F. Skinner en démontrant qu'elles ne sont pas aussi antithétiques qu'on le suppose d'ordinaire ou que les deux hommes avaient tendance à les penser. L'auteure utilise le concept de systèmes non-linéaires et dynamiques comme concept principal dans son effort de créer un pont entre le travail de Rogers et de Skinner. Il s'ensuit qu'elle propose le concept de non-prédictibilité pour remplacer le concept de non-directivité en tant que caractérisation plus juste et plus fondamentale de la thérapie centrée sur le client classique. Le concept de non-predictibilité permet à la thérapie centrée sur le client d'être encadrée, d'une manière globale et générale, par le principe de conditionnement opérant de Skinner sans faire violence à la préoccupation de Rogers concernant l'autonomie du client. Plus spécifiquement, l'article soutient que la théorie de la thérapie de Rogers n'est pas antithétique par rapport à la théorie de la personnalité de Skinner et que la thérapie centrée sur le client peut donc être pratiquée sans heurt par des thérapeutes qui, comme l'auteure, croient que le conditionnement opérant est un concept qui explique mieux le comportement humain que la tendance actualisante.

Fazendo a ponte entre as posições de Rogers e de Skinner: O papel dos sistemas dinâmicos não lineares

Este artigo pretende estabelecer uma ponte entre as posições de Carl Rogers e de B.F. Skinner, ao demonstrar que os dois autores não são antitéticos como habitualmente se julga e como eles próprios tendiam a ver-se um ao outro. A autora recorre ao conceito de sistemas dinâmicos não lineares como sendo central no estabelecimento dessa ponte entre os trabalhos de Rogers e de Skinner e, consequentemente, introduz o conceito de não previsibilidade para substituir a não directividade, considerando o primeiro como permitindo uma caracterização mais correcta e básica da terapia centrada no cliente clássica. O conceito de não previsibilidade permite que se enquadre a terapia centrada no cliente, de forma mais abrangente e geral, através do princípio skinneriano de condicionamento operante, sem violentar a preocupação de Rogers com a autonomia do cliente. Mais concretamente, este artigo pretende que a teoria da terapia de Rogers não é antagónica com a teoria da personalidade de Skinner e que, consequentemente, a terapia centrada no cliente pode ser colocada em prática, sem problemas, por terapeutas que, como o autor, acreditam que o condicionamento operante é um

conceito mais adequado para o comportamento humano do que a tendência actualizante.

ロジャーズとスキナーの立場の橋渡し：非線形力動的システムの役割について

本論文の目的は、カール・ロジャーズとB.F.スキナーの立場が「対立的」ではないことを示し、彼らの間にある相違を埋めることである。ロジャーズとスキナーの業績の間にある相違を埋めるための重要な概念として、非線形力動的システムを用いた。古典的な来談者中心療法におけるより適切で基本的な特徴として、"非指示性"の代わりに"非予測性"を提案した。そして、B.F.スキナーのオペラント条件付けの原理による"非予測性"の概念により、来談者中心療法は、クライエントの自主性に関するロジャーズの懸念を害することなく、より包括的で普遍的に形作られるとし、ロジャーズのセラピーに関する理論がスキナーのパーソナリティ理論と正反対のものではないことを示した。そして、実現傾向よりもオペラント条件付けが人間の行動の説明概念として優れていると信じている治療者であったとしても、来談者中心療が全ての学派にわけ隔てなく実践されうると論じた。

En brobygger mellem Rogers og Skinner: De ikke-lineære dynamiske systemers rolle

Denne artiikel har til formål at bygge bro mellem Carl Rogers' og B. F. Skinners positioner ved at demonstrere at de ikke er så anti-tetiske som almindeligt antaget og som de to mænd selv anså dem for at være. Forfatteren bruger begrebet "ikke-lineære dynamiske systemer" som sit vigtigste begreb i forsøget på at bygge bro mellem Rogers' og Skinners teorier, og som en konsekvens heraf introducerer hun begrebet "ikke-forudsigende" til erstatning for begrebet "ikke-direktiv," idet hun anser førstnævnte for at være en mere basal og korrekt karakterisering af klassisk klient-centreret terapi. Begrebet "ikke-forudsigende" tillader klient-centreret terapi at blive kontekstualiseret, på overordnet og generel vis, af B. F. skinners princip om operant betingning uden at gøre vold på Rogers' omsorg for klientens autonomi. Mere specifikt argumenterer artiklen for at Rogers' teori om terapi ikke er antitetisk til Skinners personlighedsteori og for at klient-centreret terapi derfor kan praktiseres gnidningsfrit af terapeuter der, som forfatteren, tror at operant betingning er et bedre begreb til forklaring af menneskers adfærd end den aktualiserende tendens.

Introduction

Writing this paper has been close to the heart of the author who finds so much of value in Rogers' as well as in Skinner's works that it has become increasingly inconceivable to comprehend their views as being excluding of or in contradiction with each other. Rogers and Skinner were, after all, among the giants of 20th century psychology. It therefore seems most likely that much of value can be found in both men's work.

If Rogers and Skinner could have had a third dialogue, today, informed by late 20th century advances in work with nonlinear dynamic systems (see below), they

might have ended up on the same page and there might not have been the split between the behaviorist orientation and the humanist orientation in psychology that we see today. They agreed (Rogers & Skinner, 1956, 1962/1989) that human behavior is determined, although Rogers had some reservations, because he found that Skinner attached too little importance to the inner, subjective experiences of the individual, particularly the experience of being free and having choices. Rogers did not clarify, though, what kind of importance should be attached to these inner, subjective experiences, apart from stating in various ways that they were important because they felt important and meaningful to the individual. He spoke of a "paradoxical" relationship between scientific determinism and inner, subjective experiences. He said (1962/1989, p. 132):

> I am in thorough agreement with Dr. Skinner that, viewed from the external, scientific, objective perspective, man is determined by genetic and cultural influences. I have also said that in an entirely different dimension, such things as freedom and choice are extremely real ... I see it as being similar to the situation in physics where you can prove that the wave theory of light is supported by evidence, as is the corpuscular theory, though the two of them appear to be contradictory. They are not, at the present state of knowledge, reconcilable, but one would be narrowing his perception of physics to deny one or the other. It is in this same sense that I regard both of these dimensions as real, although they exist in paradoxical relationship. [Physicists, today, would not speak of a paradoxical relationship, but of a complementary relationship between the wave theory and the corpuscular theory of light. (This author's comment)]

However, talking about a paradoxical relationship between scientific determinism and the experience of freedom doesn't really explain anything and, significantly, Rogers did not state that he attached causative agency to inner experiences, or to the experience of being free to make choices, that is, to the experience of having a free will. On the contrary, he said (Rogers, 1962/1989, p. 99): "Behaviors that were formerly dealt with as though they were caused by little homunculi within the individual, or various internal causes, are now seen to have other types of causation."

Rogers' reservation to Skinner's views seems first and foremost to originate in Rogers' feeling of no less than revulsion (1962, p. 126) at the thought that human behavior could be controlled *ad modum* Walden Two (Skinner, 1948). Skinner took the position that as much as he acknowledged the existence of inner, subjective experiences, including the experience of choosing freely, he regarded them as more or less covert or private behaviors, and it was the environmental consequences of a person's behavior, not any kind of inner experiences, that should be studied, because those consequences were the causes (in the form of rewards or punishments) of the behavior. By engineering the environmental consequences of a person's behavior, it was, according to Skinner, possible to control this person's behavior and that prospect was as positive from Skinner's point of view as it was negative from Rogers' point of view.

Basically, however, they were in agreement about the assumption that determinism implies predictability. Today, they would have been informed about Chaos Theory and nonlinear dynamic systems, that is, they would have known that determinism does not necessarily imply predictability. In the light of this knowledge, they might have appreciated that their respective positions were, indeed, compatible. Skinner might have understood that in spite of his being right about the principle of operant conditioning and determinism for living organisms, in general, he could not hope to be able to control the rich and complex behavior of any human individual, in

particular. A Walden Two could never be realized. Rogers, on his side, might have understood that his agreeing with Skinner about determinism did not exclude the soundness and relevance of his nonpredictive therapeutic approach with any individual client.

The impossibility of predictive control of complex sets of behavior

A basic assumption

It is a basic assumption of this paper that the trajectory of the process of the individual human organism's interaction with its environment from conception to death is best modeled by a *nonlinear dynamic system* unlike, for example, the trajectory of the planets around the sun, which is, as physics has long since demonstrated, best modeled by a linear dynamic system.

Nonlinear dynamic systems[1]

Classical Newtonian science is almost exclusively concerned with linear dynamic systems. In such systems, cause–effect relationships can be clearly and exhaustively defined. This allows for very precise predictions of the behavior of a linear dynamic system. With measurements of a few parameters, the trajectory of a planet, for example, can be very precisely predicted. This is so even if no set of measurements can be 100% precise. Physicists and mathematicians say about linear dynamic systems that small differences in initial conditions remain small differences in the future behavior of the system. For all practical purposes, therefore, small differences in initial conditions can be disregarded. Stated otherwise, one can make one's measurements as precise as one wishes one's predictions to be precise. This is the essentially characterizing feature of linear dynamic systems.

Such is not the case with nonlinear dynamic systems. They are said to be sensitively dependent on initial conditions. This means that small differences in initial conditions can develop into big differences in the future behavior of the systems. *The butterfly effect* is the ubiquitously used term for this sensitive dependence on initial conditions. To illustrate what nonlinear dynamic systems are about, it is said that a butterfly flapping its wings in Beijing on Sunday can be decisive for the weather in New York next Saturday. This means that the development of the weather around the globe is a nonlinear dynamic system, which is sensitively dependent on initial conditions, and it is the reason meteorologists cannot predict the weather with better than chance precision for much more than four or five days ahead.[2]

To the surprise of most people, mathematics, that is, the area that is normally thought of as the most predictable of all, is full of nonlinear dynamic systems. Many mathematical equations show sensitive dependence on initial conditions. If one iterates (develops) such equations on a calculator that delivers results with nine decimal points' precision and on a calculator that delivers results with eight decimal points' precision one will find that the iterations on the two calculators sooner or later start to vary widely and seemingly chaotically, as a result of the tiny, one millionth part, difference in initial conditions. *Chaos Theory* is the field of mathematics that is concerned with nonlinear dynamic systems (see, e.g., Gleick, 1987; Lorenz, 1993; Peitgen, Jürgens, & Saupe, 1992). The name is somewhat unfortunate, since there is really nothing "chaotic" about these systems. They are

fully determined systems. The meaning of the term *chaotic* is, more precisely, *unpredictable*. Nonlinear dynamic systems are as determined as linear dynamic systems, but they are as unpredictable as linear dynamic systems are predictable.

Here is another example of a nonlinear dynamic system:

Imagine that you let go of a small bullet at the edge of a large funnel with a hole in the bottom, just a tiny bit larger in diameter than the bullet. You can, of course, on the basis of the law of gravity, predict that it will end up disappearing down the hole in the bottom, but there is no way you can predict its trajectory toward that disappearance. That trajectory is sensitively dependent on the tiniest differences in the way you hold the bullet towards the edge of the funnel at the start, on the tiniest differences in where you hold it, on the tiniest differences in the way you let go of it, and on the tiniest details of irregularities on the side of the funnel. Any of these tiny differences can have a huge effect on the bullet's trajectory. At the same time, these differences are so tiny that there is no way to measure them, just as there is no way to measure their combined differential influence on the bullet's behavior in a way that is sufficiently precise to make it possible to predict the trajectory of the bullet towards its disappearance. One can only make the prediction (obvious to everyone) that the bullet's overall trajectory will be downwards, that it will probably not disappear right away, but instead hit the edge of the hole in the bottom, and be spiraled up again, one time or several times, but still closer to the bottom and still more slowly as it loses energy, until it finally disappears.

At this point, the reader is welcome to associate to the human organism's trajectory through life, since my point with this rather detailed explanation of nonlinear dynamic systems is, as already stated, to postulate that the behavior of the human organism, that is, its continuously changing complex sets of interactions with a continuously changing environment, from conception to death, is a nonlinear dynamic system, and, probably, *the* nonlinear dynamic system, par excellence. The human organism in its environment is, more than anything, like the bullet in the funnel that was described above: Its trajectory through life is as sensitively dependent on initial conditions as the bullet's trajectory on the side of the funnel.

Determined and unpredictable

The consequence of this assumption is that the interactional processes of human beings with their environment are determined *and* unpredictable. It is very important to grasp the idea that something can be both determined and unpredictable. It is important, because our language and thinking is so suffused with the linearity of classical science that we automatically tend to think of determined systems as being synonymous with predictable systems. It is therefore no wonder that Rogers and Skinner did not question the basic, mistaken, assumption that they had in common: that determinism invariably implies predictability. It was only with the advent of the modern computer that mathematicians and physicists could investigate nonlinear dynamic systems thoroughly, and knowledge about nonlinear dynamic systems did not become common knowledge until the two last decades of the past century. At the time of the dialogues between Rogers and Skinner, thinking in terms of predictable, linear dynamic systems was still the way of thinking that dominated the sciences.

The individual level and the general level

Rogers and Skinner, though, were not altogether wrong about their assumption that determinism implies predictability. They were only wrong on the level of the individual human organism's interaction with the environment, and they were not very careful about distinguishing between the individual and the general level in their dialogues. Their failure to do so may have contributed to their thinking of themselves as being in disagreement. On the general level, that is, on the level of people, at large, or of sufficiently large groups of people, or on the level of "the average," they were right. And it may be this level Rogers had in mind when he spoke of the "scientific perspective" in the quotation above. On this level Skinner's laws of conditioning can be applied to predict which environments will produce a certain kind of behavior among the majority of people exposed to this environment. And on this level Rogers and Skinner were actually in rather close agreement about the general kind of environment that would produce the most constructive behaviors in the majority of people or, vice versa, they were in agreement about the kind of environment that hampered the actualization of these behaviors, that is, they were in agreement about a general theory of disturbed psychological development or, in Skinner's terminology, disturbed behavior. For Rogers, psychological disturbance came about as a result of the individual having been excessively exposed to conditional regard. For Skinner, disturbed behavior came about as a result of the individual having been excessively exposed to punishing consequences.

Skinner's theory operates with four reinforcers, two positive (increasing the likelihood of recurrence of a certain set of behaviors) and two negative (decreasing the likelihood of recurrence of a certain set of behaviors). The two positive reinforcers are (1) finding rewarding consequences, and (2) escaping from punishing consequences; and the two negative reinforcers are (3) finding punishing consequences, and (4) finding that rewarding consequences have disappeared (O'Donohue & Ferguson, 2001, p. 92).

For Rogers conditional positive regard is actually implicitly punishing of behaviors that are not regarded positively: "I love you when you . . .," implies that "I do not love you when . . . " and withdrawal of love can be as painful to humans as electric shock to rats.

Thus, it is hard to see any essential difference between "conditional regard" and "punishing consequences."

Rogers, however, employed the concept "unconditional positive regard" that has no counterpart in Skinner's work, and this concept of Rogers', although he didn't explicitly say so anywhere, embraces the unpredictability of human behavior that went beyond Skinner's theory. (See further, below.) Rogers, more than Skinner, had a "sense" of nonlinear dynamics, before knowledge of these dynamics became accessible.

In any case, they were in agreement about the essential point: that disturbed psychological development or disturbed behavior is the result of unfortunate environments, that is, it is conditioned and thus controlled by the environment, even if it is impossible to tell, on the individual level, what the specific conditioning contingencies or reinforcement relationships of any given set of disturbed behaviors might have been and may be. The logical conclusion is that they were also in agreement that any behavior, not only disturbed behavior, is controlled by the environment. The

evidence is Rogers' statement, in the debate with Skinner, that "I am in thorough agreement with Dr. Skinner that, viewed from the external, scientific, objective perspective, man is determined by genetic and cultural influences" (1962/1989, p. 132; see the full quotation above). In another context Rogers wrote:

> The question is often raised: But what about the therapist's attitude toward his client's asocial or antisocial behavior? Is he to accept this without evaluation? Sometimes this question is answered by saying that the effective therapist prizes the person, but not necessarily his behavior. Yet it is doubtful if this is an adequate or true answer. To be sure, the therapist may feel that a particular behavior is socially unacceptable or socially bad, something he could not approve of in himself, and a way of behaving which is inimical to the welfare of the social group. But the effective therapist may feel acceptant of this behavior in his client, not as desirable behavior, but as a *natural consequence* of the circumstances, experiences, and feelings of this client. Thus the therapist's acceptance may be based upon this kind of feeling: "If I had had the same background, the same circumstances, the same experiences, it would be inevitable in me, as it is in this client, that I would act in this fashion." (Rogers, Gendlin, Keisler, & Truax, 1967, p. 103)

Here Rogers is fully on the same wavelength as Skinner.

Rogers' dilemma

As we have seen, Rogers, however, had problems with this and he also often spoke about the self-governing, internally controlled individual. For example, in his definition of the actualizing tendency (Rogers, 1959, p. 196) he spoke of development "toward autonomy and away from heteronomy, or control by external forces." It seems to be this apparent contradiction in Rogers' view that he tried to resolve with reference to the "paradoxical" relationship between the individual experience of freedom and the notion that human behavior is determined.

Rogers also expressed his efforts to integrate the idea of individual freedom on the one hand and determinism on the other in the following quote:

> The fully functioning person ... not only experiences, but utilizes, the most absolute freedom when he spontaneously, freely, and voluntarily chooses and wills that which is also absolutely determined. (Rogers, 1961, p. 193)

It should be evident from these quotations that Rogers struggled hard with the free will/determinism issue without resolving it. The author wonders why Rogers seemingly did not pay attention to the everyday occurrence that subjective experience and objective knowledge can be held simultaneously in mind: We can enjoy the beautiful sunset while knowing full well that the sun is not at all "setting"; instead the earth is revolving around it. Likewise, we can hold in mind, simultaneously, the enjoyment of freedom while knowing full well that the experience of freedom is a subjective experience that is as determined as everything else and certainly not an experience enjoyed by all.

Skinner's mistake

It is undoubtedly the impossibility of making sufficiently precise predictions about the individual conditioning contingencies of a given client's behavior that is at the heart of the problems of behavior analytic therapy, that is, the therapeutic

application of Skinner's laws of conditioning on the level of individual clients. The engineering of specific reinforcement strategies to change a given "target behavior" demands isolation of the target behavior and prediction about the reinforcement strategies to be applied to change the target behavior, and this is, more often than not, impossible with the complex kind of behaviors and problems ordinary clients present to ordinary practicing therapists. The rare client may present with sufficiently simple and well-delineated kinds of symptom/problem/target behavior for effective reinforcement strategies to be successfully predicted and applied. In these cases, it is possible to approach the client's "presenting problem" to a linear dynamic system and to work with it accordingly. To remain with the bullet in the funnel analogy, this would roughly correspond to a whole in the bottom that was sufficiently large to make it obvious that the bullet would disappear directly into it independently of any differential influences on its way to the bottom. As early as the middle of the last century numerous studies, referred to by Patterson (1963), showed that carefully chosen, well-delineated and obvious "bits" of behavior can be changed by predictive operant conditioning. Ordinarily, though, this kind of approximation to a linear dynamic system is not possible in therapy without disregarding essential features of the relatively complex problems that clients typically see therapists about. These problems are part of a unique nonlinear dynamic system that is as unpredictable as it is determined. With ordinary clients, the diameter of the hole in the bottom is not much larger than the diameter of the bullet, the side of the funnel is very irregular and there are an infinite number of variations in the ways different bullets were started on their unique trajectories toward the bottom.

Therefore, Chaos Theory and the basic unpredictability of nonlinear dynamic systems falsify Skinner's assumption of the predictability of the behavior of the individual human organism. Rogers' opposition to this point of Skinner's ideas seems to have been, primarily, ethically motivated, but later developments in mathematics and physics came to his support: Rogers' idea that a therapist should not try to control the behavior of any individual client gains momentum by the idea that the therapist, quite simply, *cannot* control the behavior of any given client.

A third dialogue

Thus, if Rogers and Skinner could have had a third dialogue, today, Rogers, in the light of the basic unpredictability of nonlinear dynamic systems, might not have been as uncomfortable about Skinner's deterministic position as he was in 1957 and 1962. Rogers might more unequivocally have agreed with Skinner that the human organism is determined and Skinner might have agreed that Rogers was right that the complex sets of interaction of a client with his or her environment should not be exposed to efforts of direction or control in therapy; not, primarily, because such efforts were ethically wrong, but because such efforts were, more likely than not, doomed to failure, since they would imply therapist capacity to make appropriate predictions about the client at odds with the trajectory of client changes being best modeled by a nonlinear dynamic system.

Classical client-centered therapy: Nonpredictive rather than nondirective

Classical client-centered therapy is regarded by most practitioners as being utterly nondirective (see, e.g., Bozarth, 1998, p. 57; Grant, 1990; Levitt & Brodley, 2005). It

is the therapy of the "early Rogers" or "Rogers-1" (Frankel & Sommerbeck, 2005, 2007) that Rogers described in *Counseling and Psychotherapy* (1942), where he distinguished between "directive" approaches and his own "nondirective" approach (1942, pp. 115–128). He articulated the philosophical foundation of this therapy in more detail in *Client-Centered Therapy* (1951). Nevertheless, most would probably agree that classical-client centered therapists do direct, in the sense of influence, their clients, just as clients influence their therapists, and just as we all influence each other all the time. Skinner would say that control or conditioning is mutual; it goes both ways. Therefore, the essence of nondirectivity in classical client-centered therapy is, more precisely, that the therapist does not *systematically* try to influence or direct the client. The therapist has no preconceived, that is, *predictive* idea, based on diagnostic assessment, a particular theory of personality, or any other perspective of their own, of how and in which direction they wish to influence the client, and the therapist entertains no such ideas at any moment during the course of therapy. In short, the therapist makes no effort to predict what will be helpful to a given client, but engages instead in continuous empathic understanding of the client's perspective.

This does not mean that expert predictions do not exist. However, expert predictions are made on the basis of experts' knowledge of what characterizes some average of a group of people or some sufficiently large group. Such predictions can be made with satisfying precision when the average process and the actual process are virtually the same, which is the case with linear dynamic systems like, for example, the trajectory of the planets around the sun. But for nonlinear dynamic systems, where small differences in initial conditions can develop into very large differences at a later point in time, it is impossible to know how widely the known average and the actual system of interest at this precise moment in time differ from each other, except in very crude and banal ways. Psychologists and psychiatrists, for example, are experts at how a sufficiently large group of people with a certain diagnosis responds to this or that intervention, or how the "average" person with this diagnosis responds, but have no way of knowing how close any given client with the given diagnosis is to this average (Sommerbeck, 2004, pp. 295–297). Therefore:

> Any intervention with the process of another, which is made with the intention of being helpful, as will most often be the case in therapy, is made on the basis of a prediction that this intervention will actually, more likely than not, be helpful for the other. Otherwise, the intervention would not be made. In making such predictions, the other is treated as a linear dynamic system, as "an average person" or as "an average client" on the basis of theory, research, or the therapist's professional or personal experience. The other is not treated as this particular, unique, nonlinear dynamic system, John, who is different from any average, or as this other, different and equally unique, nonlinear dynamic system, Jenny, whose future interactions with her environment it is impossible to make predictions about. (Sommerbeck, 2004, p. 296)

To this author, Rogers' fourth condition of the six necessary and sufficient conditions for therapeutic change (Rogers, 1959, p. 213), unconditional positive regard for the client, is, first and foremost, respect for the uniqueness of each client, or respect for the client being a nonlinear dynamic system that it is impossible to make anything but banal and crude, that is, more often than not therapeutically irrelevant, predictions about. It is also respect for the importance of clients' subjective *experience* of freedom, while knowing that objectively the client is as

determined as the therapist or anything/anyone else, and that the therapy, as a whole, is but another determining or conditioning influence, albeit a nonpredicted influence, in the life of the client and in the life of the therapist. The only prediction made by the classical client-centered therapist is that the client will, more likely than not, be better off at the conclusion of the therapy than at the start of it, but what this means, in any detail, is left blowing in the wind.

A personal note

This paper represents the way the author, for herself, has tried to resolve Rogers' "paradox" regarding freedom and determinism, and the way she can effortlessly and satisfyingly contain the wisdom of Rogers' client-centered therapy and the wisdom of Skinner's principle of operant conditioning at the same time. It is the way for the author to be dedicated to Rogers' theory of therapy, while being critical of Rogers' theory of personality, particularly the concept of the actualizing tendency (Frankel, Sommerbeck, & Rachlin, 2010) and instead embracing Skinner's behaviorist theory of personality.

Conclusion

Hopefully, this paper has demonstrated that if Rogers and Skinner could have had a third dialogue, today, informed by knowledge of nonlinear dynamic systems, they could have bridged the apparent gap between their respective positions. They could have agreed that, rather ironically, the very point they used to agree about, that determinism implies predictability, was the very point they were both wrong about. They would have known that most aspects of a human being's trajectory through life are a nonlinear dynamic system, determined and unpredictable, and in the light of this knowledge Rogers could have agreed unequivocally with Skinner about operant conditioning and determinism without fearing the consequences of his agreement for the relevance of his therapeutic approach. And in this same light, Skinner could have agreed about the wisdom of nonpredictive client-centered therapy for the ordinary complexity of client problems that demand unconditional respect for client uniqueness rather than respect for client freedom in some nonscientific "dimension."

A corollary of this conclusion is the dispensability of a belief in the existence of the actualizing tendency as the foundation for practicing client-centered therapy. A belief in, and respect for client uniqueness suffices.

Notes

1. This explanation of nonlinear dynamic systems is a slightly revised and shortened excerpt of Sommerbeck (2004). The author is grateful to the editors of the PCEP journal for their permission to insert this excerpt in the present article.
2. It would be more correct to say that *the trajectory of* the weather is *best modeled by* a nonlinear dynamic system, since the concept of a nonlinear dynamic system is a mathematical concept that consists of mathematical variables and their interrelationships, which the weather, of course, does not consist of. For easier readability, though, the author has chosen the less mathematically correct formulation that this or that is a nonlinear dynamic system rather than the more circumstantial, but also more correct, formulation that the trajectory of this or that is best modeled by a nonlinear dynamic system.

References

Bozarth, J. (1998). *Person-centered therapy: A revolutionary paradigm.* Ross-on-Wye, UK: PCCS Books.

Frankel, M., & Sommerbeck, L. (2005). Two Rogers and congruence: The emergence of therapist-centred therapy and the demise of client-centred therapy. In B. Levitt (Ed.), *Embracing non-directivity* (pp. 40–61). Ross-on-Wye, UK: PCCS Books.

Frankel, M., & Sommerbeck, L. (2007). Two Rogers: Congruence and the change from client-centered therapy to we-centered therapy. *Person-Centered & Experiential Psychotherapies, 4,* 286–295.

Frankel, M., Sommerbeck, L., & Rachlin, H. (2010). Rogers' concept of the actualizing tendency in relation to Darwinian theory. *Person-Centered & Experiential Psychotherapies, 9,* 69–80.

Gleick, J. (1987). *Chaos: Making a new science.* New York: Viking Penguin.

Grant, B. (1990). Principled and instrumental non-directiveness in person-centered and client-centered therapy. *Person-Centered Review, 5,* 77–88.

Levitt, B., & Brodley, B.T. (2005). "It enlightens everything you do": Observing non-directivity in a client-centred therapy demonstration session. In B. Levitt (Ed.), *Embracing non-directivity* (pp. 96–112). Ross-on-Wye, UK: PCCS Books.

Lorenz, E. (1993). *The essence of chaos.* Seattle, WA: University of Washington Press.

O'Donohue, W., & Ferguson, K. (2001). *The psychology of B. F. Skinner.* London: Sage.

Patterson, C. (1963). Control, conditioning and counselling. *Personnel and Guidance Journal, 41,* 680–686.

Peitgen, H., Jürgens, H., & Saupe, D. (1992). *Chaos and fractals.* New York: Springer-Verlag.

Rogers, C.R. (1942). *Counseling and psychotherapy.* Boston: Houghton Mifflin.

Rogers, C.R. (1951). *Client-centered therapy.* Boston: Houghton Mifflin.

Rogers, C.R. (1959). A theory of therapy, personality, and interpersonal relationships as developed in the client-centered framework. In S. Koch (Ed.), *Psychology: A study of a science. Vol. 3: Formulations of the person and the social context.* New York: McGraw-Hill.

Rogers, C.R. (1961). *On becoming a person.* London: Constable.

Rogers, C.R., & Skinner, B. (1956). Some issues concerning the control of human behavior. *Science, 124*(3231), 1057–1066.

Rogers, C.R., & Skinner, B.F. (1989). Dialogue. In H. Kirschenbaum & V. L. Henderson (Eds.), *Carl Rogers: Dialogues* (pp. 79–152). Boston: Houghton Mifflin. (Original work published 1962).

Rogers, C.R., Gendlin, E., Kiesler, D., & Truax, C. (1967). *The therapeutic relationship with schizophrenics.* Madison, WI: The University of Wisconsin Press.

Skinner, B.F. (1948). *Walden Two.* Indianapolis, IN: Hackett Publishing Company.

Sommerbeck, L. (2004). Non-linear dynamic systems and the non-directive attitude in client-centered therapy. *Person-Centered & Experiential Psychotherapies, 3,* 291–299.

(Source 5)

Journal of Experimental Psychology:
Animal Behavior Processes
2009, Vol. 35, No. 4, 447–472

0097-7403/09/$12.00 DOI: 10.1037/a0015626

The Dynamics of Conditioning and Extinction

Peter R. Killeen, Federico Sanabria, and Igor Dolgov
Arizona State University

Pigeons responded to intermittently reinforced classical conditioning trials with erratic bouts of responding to the conditioned stimulus. Responding depended on whether the prior trial contained a peck, food, or both. A linear persistence–learning model moved pigeons into and out of a response state, and a Weibull distribution for number of within-trial responses governed in-state pecking. Variations of trial and intertrial durations caused correlated changes in rate and probability of responding and in model parameters. A novel prediction—in the protracted absence of food, response rates can plateau above zero—was validated. The model predicted smooth acquisition functions when instantiated with the probability of food but a more accurate jagged learning curve when instantiated with trial-to-trial records of reinforcement. The Skinnerian parameter was dominant only when food could be accelerated or delayed by pecking. These experiments provide a framework for trial-by-trial accounts of conditioning and extinction that increases the information available from the data, permitting such accounts to comment more definitively on complex contemporary models of momentum and conditioning.

Keywords: autoshaping, behavioral momentum, classical conditioning, dynamic analyses, instrumental conditioning

Estes's stimulus sampling theory provided the first approximation to a general quantitative theory of learning; by adding a hypothetical attentional mechanism to conditioning, it carried analysis one step beyond extant linear learning models into the realm of theory (Atkinson & Estes, 1962; Bower, 1994; Estes, 1950, 1962; Healy, Kosslyn, & Shiffrin, 1992). Rescorla and Wagner (1972) added the important nuance that the asymptotic level of conditioning might be partitioned among stimuli that are associated with reinforcers as a function of their reliability as predictors of reinforcement; that refinement has had tremendous and widespread impact (Siegel & Allan, 1996). The attempt to couch the theory in ways that account for increasing amounts of the variance in behavior has been one of the main engines driving modern learning theory. Models have been the agents of progress, the go-betweens that reshaped both our theoretical inferences about the conditioning processes and our modes of data analysis. In this theoretical–empirical dialogue, the Rescorla–Wagner (R-W) model has been a paragon.

Despite the elegant mathematical form of their arguments, the predictions of recent learning models are almost always qualitative—a particular constellation of cues is predicted to block or enhance conditioning more than others because of their differential associability or their history of association, and those effects are measured by differences in speed of acquisition or extinction or as a response rate in test trials. Individual differences, and the brevity of learning and extinction processes, make convergence on meaningful parametric values difficult: There are nothing like the basic constants of physics and chemistry to be found in psychology. To this is the added difficulty of a general analytic solution of the R-W model (Danks, 2003; Yamaguchi, 2006). As Bitterman (2006) astutely noted, the residue of these difficulties leaves predictions that are at best ordinal and dependent on simplifying assumptions concerning the map from reinforcers to associations and from associations to responses:

> The only thing we have now that begins to approximate a general theory of conditioning was introduced more than 30 years ago by Rescorla and Wagner (1972). . . . . An especially attractive feature of the theory is its statement in equational form, the old linear equation of Bush and Mosteller (1951) in a different and now familiar notation, which opens the door to quantitative prediction. That door, unfortunately, remains unentered. Without values for the several parameters of the equation, associative strength cannot be computed, which means that predictions from the theory can be no more than ordinal, and even then those predictions are made on the naïve assumption of a one-to-one relation between associative strength and performance. (p. 367)

To pass through the doorway that these pioneers have opened requires techniques for estimating parameters in which we can have some confidence, and to achieve that requires a database of more than a few score learning and testing trials. But most regnant paradigms get only a few conditioning sessions out of an organism (see, e.g., Mackintosh, 1974), whereupon the subject is no longer naive. To reduce error variance, therefore, data must be averaged over many animals. This is inefficient in terms of data utilization and also confounds the variability of learning parameters as a function of conditions with the variability of performance across subjects (Loftus & Masson, 1994). The pooled data may not yield parameters representative of individual animals; when functions are nonlinear, as are most learning models, the average of param-

Peter R. Killeen, Federico Sanabria, and Igor Dolgov, Department of Psychology, Arizona State University.

This work was supported by National Institute of Mental Health Grant R01MH066860 and some of the workers by National Science Foundation IBN 0236821.

Correspondence concerning this article should be addressed to Peter R. Killeen, Department of Psychology, Arizona State University, Box 871104, Tempe, AZ 85287-1104. E-mail: killeen@asu.edu

eters of individual animals may deviate from the parameters of pooled data (Estes, 1956; Killeen, 2001). Averaging the output of large-*N* studies is therefore an expensive and nonoptimal way to narrow the confidence intervals on parameters (Ashby & O'Brien, 2008).

Most learning is not, in any case, the learning of novel responses to novel stimuli. It is refining, retuning, reinstating, or remembering sequences of action that may have had a checkered history of association with reinforcement. In this article, we make a virtue of the necessity of working with non-naïve animals, to explore ways to compile adequate data for convergence on parameters, and prediction of data on an instance-by-instance basis. Our strategy was to use voluminous data sets to choose among learning processes that permit both Pavlovian and Skinnerian associations. Our tactic was to develop and deploy general versions of the linear learning equation—an error-correction equation, in modern parlance—to characterize repeated acquisition, extinction, and reacquisition of conditioned responding.

Perhaps the most important problem with the traditional paradigm is its ecological validity: Conditioning and extinction acting in isolation may occur at different rates than when occurring in mélange (Rescorla, 2000a, 2000b). This limits the generalizability of acquisition–extinction analyses to newly acquired associations. A seldom-explored alternative approach consists of setting up reinforcement contingencies that engender continual sequences of acquisition and extinction. This would allow the estimation of within-subject learning parameters on the basis of large data sets, thus increasing the efficiency of data use and disentangling between-subjects variability in parameter estimates from variability in performance. Against the possibility that animals will just stop learning at some point in extended probabilistic training, Colwill and Rescorla (1988; Colwill & Triola, 2002) have shown that if anything, associations increase throughout such training.

One of Skinner's many innovations was to examine the effects of mixtures of extinction and conditioning in a systematic manner. He originally studied fixed-interval schedules under the rubric "periodic reconditioning" (Skinner, 1938). But, absent computers to aggregate the masses of data his operant techniques generated, he studied the temporal patterns drawn by cumulative recorders (Skinner, 1976). Cumulative records are artful and sometimes elegant, but difficult to translate into that common currency of science, numbers (Killeen, 1985). With a few notable exceptions (e.g., Davison & Baum, 2000; Shull, 1991; Shull, Gaynor, & Grimes, 2001), subsequent generations of operant conditioners tended to aggregate data and report summary statistics, even though computers had made a plethora of analyses possible. Limited implementations of conditional reconditioning have begun to provide critical insights on learning (e.g., Davison & Baum, 2006).

Recent contributions to the study of continual reconditioning are found in Reboreda and Kacelnik (1993), Killeen (2003), and Shull and Grimes (2006). The first two studies exploited the natural tendency of animals to approach signs of impending reinforcement, known as *sign tracking* (Hearst & Jenkins, 1974; Janssen, Farley, & Hearst, 1995). Sign tracking has been extensively studied as Pavlovian conditioned behavior (Hearst, 1975; Locurto, Terrace, & Gibbon, 1981; Vogel, Castro, & Saavedra, 2006). It is frequently elicited in birds using a positive automaintenance procedure (e.g., Perkins, Beavers, Hancock, Hemmendinger, & Ricci, 1975), in which the illumination of a response key is followed by food, regardless of the bird's behavior. Reboreda and Kacelnik and Killeen recorded pecks to the illuminated key as indicators of an acquired key–food association. In both studies, a negative contingency between key pecking and food, known as *negative automaintenance* (Williams & Williams, 1969), was imposed. In negative automaintenance, an omission contingency is superimposed such that key pecks cancel forthcoming food deliveries, whereas absent key pecks, food follows key illuminations. Key–food pairing elicits key pecking (conditioning), which, in turn, eliminates the key–food pairings, reducing key pecking (extinction), which reestablishes key–food pairings (conditioning), and so on. This generates alternating epochs of responding and nonresponding, in which responding eventually moves off key or lever (Myerson, 1974; Sanabria, Sitomer, & Killeen, 2006) and, to a naive recorder, "extinguishes." Presenting food whether or not the animal responds provides a more enduring, but no less stochastic, record of conditioning (Perkins et al., 1975). The data look similar to those shown in Figure 1; a self-similar random walk ranging from epochs of nonresponding to epochs of responding with high probabilities. Such data are paragons of what we wish to understand: How does one make scientific sense of such an unstable dynamic process? A simple average rate certainly will not do. Killeen (2003) showed that data like these had fractal properties, with Hurst exponents in the "pink noise" range. However, other than alerting us to control over multiple time scales, this throws no new light on the data in terms of psychological processes.

To generate a database in which pecking is being continually conditioned and extinguished, we instituted probabilistic classical conditioning, with the unconditioned stimulus (US) generally presented independently of responding. Using this paradigm, we examined the effect of duration of intertrial interval (ITI; Experiment 1), duration of conditioned stimulus (CS; Experiment 2), and peck–US contingency (Experiment 3) on the dynamics of key peck conditioning and extinction.

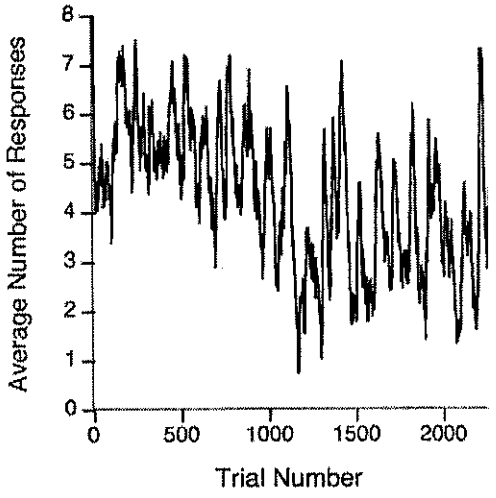

Figure 1. Moving averages of the number of responses per 5-s trial over 25 trials from 1 representative subject and condition (Pigeon 98, first condition, 40-s intertrial interval).

Experiment 1: Effects of ITI Duration and US Probability

Method

Subjects

Six experienced adult homing pigeons (*Columba livia*) were housed in a room with a 12-hr light–dark cycle, with lights on at 6:00 a.m. They had free access to water and grit in their home cages. Running weights were maintained just above their 80% ad libitum weight; a pigeon was excluded from a session if its weight exceeded its running weight by more than 7%. When required, supplementary feeding of Ace-Hi pigeon pellets (Star Milling Co., Perris, CA) was given at the end of each day, no fewer than 12 hr before experimental sessions were conducted. Supplementary feeding amounts were based equally on current deviation and on a moving average of supplements over the past 15 sessions.

Apparatus

Experimental sessions were conducted in three MED Associates (St. Albans, VT) test chambers (305 mm long × 241 mm wide × 292 mm high), enclosed in sound- and light-attenuating boxes equipped with a ventilating fan. The sidewalls and ceiling of the experimental chambers were clear plastic. The floor consisted of thin metal bars above a catch pan. A plastic, translucent response key 25 mm in diameter was located 70 mm from the ceiling, centered horizontally on the front of the chamber. The key could be illuminated by green, white, or red light emitted from diodes behind the keys. A square opening 77 mm across was located 20 mm above the floor on the front panel and could provide access to milo grain when the food hopper (part H14-10R, Coulbourne Instruments, Allentown, PA) was activated. A house light was mounted 12 mm from the ceiling on the back wall. The ventilation fan on the rear wall of the enclosing chamber provided masking noise of 60 dB. Experimental events were arranged and recorded via a Med-PC interface connected to a PC computer controlled by Med-PC IV software.

Procedure

Each session started with the illumination of the house light, which remained on for the duration of the session. Sessions started with a 40-s ITI, followed by a 5-s trial, for a total cycle duration of 45 s. During the ITI, only the house light was lit; during the trial, the center response key was illuminated white. After completing a cycle, the keylight was turned off for 2.5 s, during which food could be delivered. Two and a half seconds after the end of a cycle, a new cycle started, or the session ended and the house light was turned off. Food was always provided at the end of the first trial of every session. Pecking the center key during a trial had no programmed effect.

Initially, food was accessible for 2.5 s with reinforcement $p = .1$ at the end of every trial after the first, regardless of the pigeon's behavior. In subsequent conditions, the ITI was changed from 40 s to 20 s and then to 80 s for 3 pigeons; for the other 3 pigeons, the ITI was changed to 80 s first and then to 20 s. ITIs for all pigeons were then returned to 40 s. Each session lasted for 200 cycles when the ITI was 20 s, 100 cycles when the ITI was 40 s, and 50 cycles when the ITI was 80 s. In the last condition, the probability of reinforcement was reduced to .05 at the 40-s ITI. One pigeon (113) had ceased responding by the end of the .1 series and was not run in the .05 condition. Table 1 arrays these conditions and the number of sessions at each.

Results

The first dozen trials of each condition were discarded, and the responses in the remaining trials, averaging 2,500 per condition, are presented in the top panel of Figure 2 as mean number of responses per 5-s trial. The high-rate subject at the top of the graph is Pigeon 106 (cf. Figure 3 below). There appears to be a slight decrease in average response rates as the ITI increased and a larger decrease when the probability of food decreased from .1 to .05. Rates in the second exposure to the 40-s condition were lower than the first. These changes are echoed in the lower panel, which gives the relative frequency of at least one response on a trial. The interposition of other ITIs between the first and second exposure to the 40-s ITI caused a slight decrease in rate and probability of responding in 5 of the 6 birds, although the spread in rates in the top panel and the error bars in the bottom indicate that that trend would not achieve significance.

These data seem inconsistent with the many studies that have shown faster acquisition of the key-peck response at longer ITIs. But these data were probabilistically maintained responses over the course of many sessions. Only one other report, that of Perkins et al. (1975), constitutes a relatively close prequel to this one. These authors maintained responding on schedules of noncontingent partial reinforcement after CSs associated with different delays, probabilities, and ITIs. They used five different key colors associated with different conditions within each study. Those that come closest to those of the present experiment are shown as open symbols in Figure 2. The circles represent the average response rate of 4 pigeons on 4-s trials (converted to this 5-s base) receiving reinforcement on one of six (~16.7%) of the trials, at ITIs of 30 s (first circle) and 120 s (second circle). These data also indicate a slight decrease in rates with increasing ITIs. Perkins et al. also reported a condition with 8-s trials and 60-s ITIs involving probabilistic reinforcement. The first square in Figure 2 shows the average rate (per 5 s) of 4 pigeons at a probability of 3 of 27 (~11.1%); the second square, at a probability of 1 of 27 (~3.7%). Their subjects, like ours (and like a few other studies reported by these authors) showed a decrease in responding with a decrease in probability of reinforcement.

Any inferences one may wish to draw concerning these data are chastened by a glance at the intersubject variability of Figure 2 and of

Table 1
Conditions of Experiment 1

Order	ITI (seconds)	p[a]	Sessions
1	40	.1	20–21
2	20, 80	.1	21–23
3	80, 20	.1	20–22
4	40	.1	21–23
5	40	.05	24–29

Note. Half the subjects experienced the extreme intertrial intervals (ITIs) in the order 20 s, 80 s, and half experienced them in the other order.
[a] p is the probability of the trial ending with food.

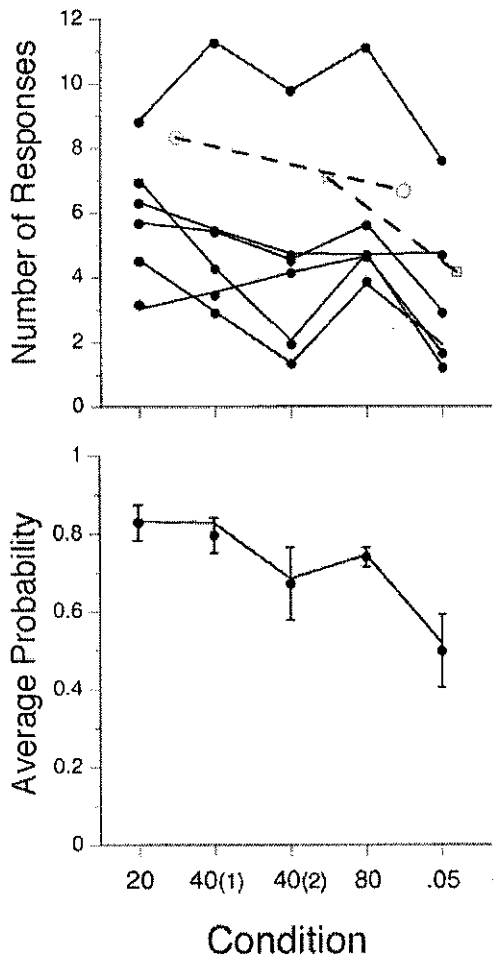

Figure 2. Data from Experiment 1. Top: average number of responses per trial (dots) for each subject, ranging from Pigeon 106 (top curve) to Pigeon 105 (bottom in Condition 20). Open symbols represent data from Perkins et al. (1975). Bottom: Average probability of making at least one response on a trial averaged over pigeons; bars give standard errors. Unbroken lines in both panels are from the Momentum/Pavlovian model, described later in the text.

Perkins et al.'s (1975) data. The effect size is small given that variability, and in fact some authors such as Gibbon, Baldock, Locurto, Gold, and Terrace (1977) have reported no effect of ITI on response rate in sustained automaintenance conditions; others (e.g., Terrace, Gibbon, Farrell, & Baldock, 1975) have reported some effect. Representing intertrial variability visually is no simpler than characterizing intersubject variability; Figure 1 gives an approximation for 1 subject (Pigeon 98) under the first 40-s ITI condition, with data averaged in running windows of 25 trials. There is an early rise in rates to around six responses per trial, then slow drift down over the first 1,000 trials, with rates stabilizing thereafter at around four responses per trial. There may be within-session warm-up and cool-down effects not obvious in this figure. We may proceed with similar displays and characterizations of them for each of the subjects in each of the conditions—all different. Or we may average performance over the whole of the experimental condition, as we did to generate the vanilla Figure 2. Or we may average data over the last 5 or 10 sessions as is the traditional modus operandi for such data. But such averages reduce a performance yielding thousands of bits of data to a report conveying only a few bits of information. As is apparent from the (smoothed) trace of Figure 1, the averages do not tell the whole story. How do we pick a path between the oversimplification of Figure 2 and the overwhelming complexity of figures such as Figure 1? And how do we tell a story of psychological processes rather than of procedural results? Models help, assayed next.

Analysis: The Models

Response Output Model

The goal of this research is to develop a procedure that can provide a more informative characterization of the dynamics of conditioning. To do this, we begin analysis with the simplest and oldest of learning models, a linear learning model of associative strength. These analyses have been in play for more than half a century (Bower, 1994; Burke & Estes, 1956; Bush & Mosteller, 1951; Couvillon & Bitterman, 1985; Levine & Burke, 1972), with the R-W model a modern avatar (Miller, Barnet, & Grahame, 1995; Wasserman & Miller, 1997). Because associative strengths are asymptotically bounded by the unit interval, it is seductive to think that they can be directly mapped to probabilities of responding or to probabilities of being in a conditioned state. Probabilities can be estimated by taking the number of trials containing at least one response within some epoch, say, 25 trials, and dividing that by the number of trials in that epoch (cf. Figure 1). There are three problems with this approach:

1. Twenty-five trials is an arbitrary epoch that may or may not coincide with a meaningful theoretical–behavioral window.

2. Information about the contingencies that were operative within that epoch are lost, along with the blurring of responses to them.

3. Parsing trials into those with and without a response discards information. Response probability makes no distinction between trials containing 1 response and trials containing 10 responses, even though they may convey different information about response strength.

4. As Bitterman (2006) noted, associative strengths are not necessarily isomorphic with probability (Rescorla, 2001).

The map between response rates and inferred strength must be the first problem attacked. The place to start is by looking at, and characterizing, the distribution of responses during a CS. Figure 3 displays the relative frequency of 0, 1, 2, . . ., 20 responses during a trial in the first condition of Experiment 1 for each of its participants.

The curves through the distributions are linear functions of Weibull densities:

$$p(n = 0) = s_i \cdot w(n, \alpha, c) + 1 - s_i,$$

$$p(n > 0) = s_i \cdot w(n, \alpha, c). \quad (1)$$

The variable s_i is the probability that the pigeon is in the response state on the ith trial. For the data in Figure 3, this is averaged over all trials. The w function is the Weibull density with index n for the actual

number of responses during the CS, the shape parameter α, and the scale parameter c, which is proportional to the mean number of responses on a trial. The first line of Equation 1 gives the probability of no responses on a trial: It is the probability that the animal is in the response state (s_i) and makes no responses [$w(n, \alpha, c)$], plus the probability that it is out of the response state ($1 - s_i$). The second line gives the probability of all nonzero responses.

The Weibull distribution is a generalization of the exponential/Poisson distribution that was recommended by Killeen, Hall, Reilly, and Kettle (2002) as a map from response rate to response probability. That recommendation was made for free operant responding during brief observational epochs. The Poisson also provides an approximate account of the response distributions shown in Figure 1. It is inferior to the Weibull, however, even when the additional shape parameter is taken into account using the Akaike information criterion (AIC). The Weibull distribution[1] is

$$W(n, \alpha, c) = 1 - e^{-(n/c)^{\alpha}}. \quad (2)$$

According to this model, when the pigeon is in a response state, it begins responding after trial onset and emits n responses during the course of that trial. It is obvious that when $\alpha = 1$, the Weibull reduces to the exponential distribution recommended by Killeen et al. (2002). In that case, there is a constant probability $1/c$ of terminating the response state from one response to the next, and the cumulative distribution is the concave asymptotic form we might associate with learning curves. Pigeon 105 exemplifies such a shape parameter, as witnessed by the almost-exponential shape of its density shown in Figure 3. Just below Pigeon 105, Pigeon 107 has a more representative shape parameter, around 2. (Whenever $\alpha > 1$, as was generally found here, there is an increasing probability of terminating responding as the trial elapses—the hazard function increases.) When α is slightly greater than 3, the function most closely approximates the normal distribution, as seen in the data for Pigeon 119. Pigeon 106, familiar from the top of Figure 2, has the most extreme shape parameter seen anywhere in these experiments, $\alpha \approx 5$. The poor fit of the function to this animal is due to its "running through" many trials, which were not long enough for its distribution to come to its natural end.

It is the Weibull density, the derivative of Equation 2, that drew the curves through the data in Figure 3. The density is easily called as a function in Excel as *=Weibull(n, α, c, false)*. It is readily interpreted as an extreme value distribution, one complementary to that shown to hold for latencies (Killeen et al., 2002). In this article, we do not use the Weibull as part of a theory of behavior but rather as a convenient interface between response rates and the conditioning machinery. Conditioning is assumed to act on s, the probability of being in the response state, a mode of activation (Timberlake, 2000, 2003) that supports key pecking.

Does the Weibull continue to act as an adequate model of the response distribution after tens of thousands of trials? For a different, and more succinct, picture of the distributions, in Figure 4 we plot the cumulative probability of emitting n responses on a trial, along with linear functions of the Weibull distribution. As before, the y-intercept of the distribution is the average probability of not making a response; the corresponding theoretical value is the probability of being out of the state, plus the (small) probability of being in the state but still not making a response. Thereafter, the probability of being in the state multiplies the cumulative Weibull distribution. The fits to the data are generally excellent, except, once again, for Pigeon 106, who did not have time for a graceful wind-down. This subject continued to run through the end of the trial; a good fit requires the Weibull distribution to be "censored," involving another parameter, which was not deemed worthwhile for its present purposes.

Changes in Response State Probability: Momentum and Pavlovian Conditioning

In his analysis of the dynamics of responding under negative automaintenance schedules, Killeen (2003) found that the best first-order predictor was the probability that the pigeon was in a response state, as given by a linear average of its probability of being in that state on the last trial and the behavior on the last trial. In the case of a trial in which a response occurred, the probability of being in the response state is incremented toward its ceiling ($\theta = 1$) using the classic (Killeen, 1981) linear average:

$$s_i' = s_i + \pi_R(\theta - s_i), \quad (3)$$

where pi (π) is a rate parameter. Pi will take different values depending on the contingencies: π_R subscripts the response, being instantiated as π_P on trials containing a peck and as π_Q on quiet trials. Theta (θ) is 1 on trials that predict future responding and 0 on trials that predict quiescence. Thus, after a trial on which the animal responded, the probability of being in the response state on the next trial will increase as

$$s_i' = s_i + \pi_P(1 - s_i),$$

whereas after a trial that contained no peck, it will decrease as

$$s_i' = s_i + \pi_Q(0 - s_i).$$

After these intermediate values of strength are computed, they are perturbed by the delivery or nondelivery of food. For that we use a version of the same exponentially weighted moving average of Equation 3:

$$s_{i+1}' = s_i' + \pi_O(\theta - s_i'). \quad (4)$$

Now the learning parameter π_O subscripts the outcome (food or empty). All of these pi parameters tell us how quickly probability approaches its ceiling or floor and thus how quickly the state on the prior trial is washed out of control (Tonneau, 2005). For geometric progressions such as these, the mean distance back is

[1] Whereas the Weibull is a continuous function, it approximates a proper distribution function on the integers, as

$$\sum_n w(n, \alpha, c) \approx 1$$

over the range of all parameters studied here. The approximation is significantly improved by adding a continuity correction of $\varepsilon = 0.5$ to all response counts. Epsilon may be thought of as a threshold for emitting the first response but is treated here merely as an ad hoc statistical correction applied to all data (except not to the pedagogic example given below). A better estimate is given by evaluating the distribution function between $n + (1/2)$ and $n - (1/2)$, with the latter taking 0 as a minimum. However, that extra computation does not add enough precision in the current situation to be useful. The Weibull should be right censored because there are time constraints on responding. This causes the deviation between predicted and obtained for Pigeon 106 in Figures 3 and 4. That refinement is not engaged here.

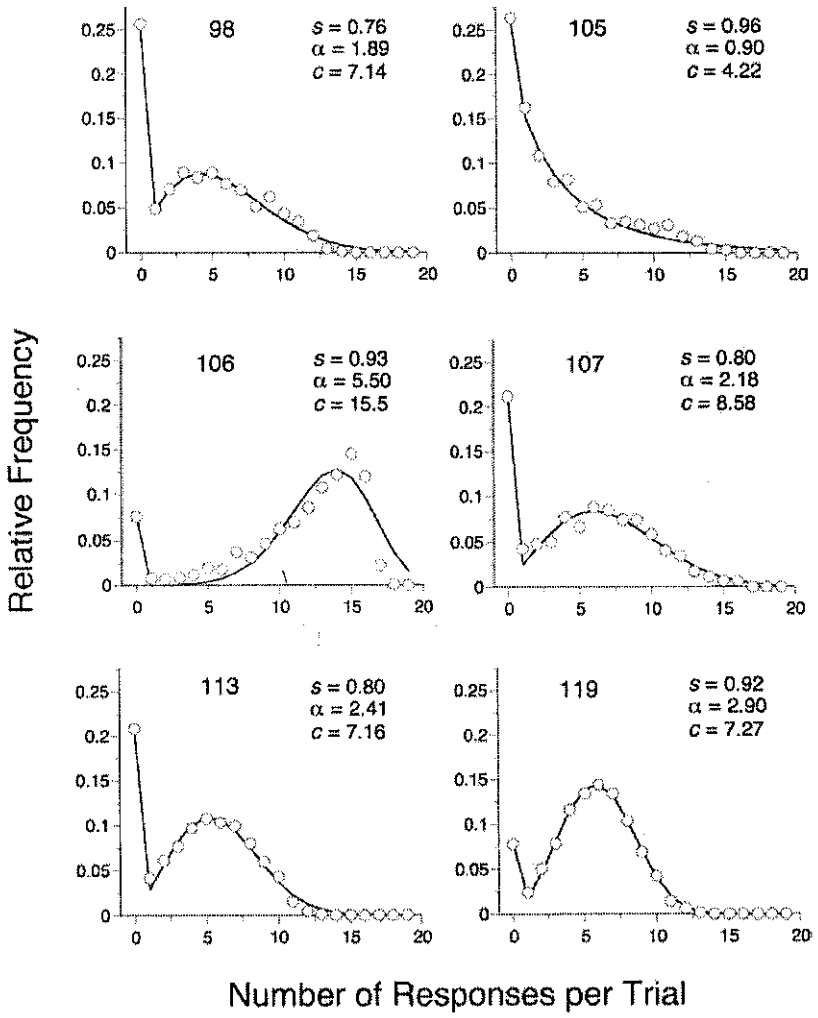

Figure 3. The relative frequency of trials containing 0, 1, 2, . . . responses. The data are from all trials of the first condition of Experiment 1. The curves are drawn by the Weibull response rate model (Equation 1). The parameter s is the probability of being in the response state; the complement of this probability accounts for most of the variance in the first data point. The parameter α dictates the shape, from exponential ($\alpha = 1$) to approximately normal ($\alpha \approx 3$) to increasingly peaked ($\alpha \approx 5$). The parameter c is proportional to the mean number of responses on trials in the response state and gives the rank order of the curves in Figure 2 at Condition 20.

$(1 - \pi)/\pi$, whenever $\pi > 0$. One might say that this is the size of the window on the past when the window is half open. As before, theta (θ) is 1 on trials that strengthen responding and 0 on trials that weaken it. Thus, after a trial on which food was delivered, we might expect to see the probability of being in the response state on the next trial (s_{i+1}) increase as

$$s_{i+1} = s_i' + \pi_F(1 - s_i'),$$

whereas after a trial that contained no food, it might decrease as

$$s_{i+1} = s_i' + \pi_E(0 - s_i').$$

These steps may be combined in a single expression, as noted in the Appendix. Although shamefully simple compared with more recent theoretical treatments, such linear operator models can acquit themselves well in mapping performance (e.g., Grace, 2002).

There are four performance parameters in this model corresponding to the four operative contingencies, each with an associated ceiling or floor. We list them in Table 2, where parenthetical signs indicate whether behavior is being strengthened (positive entails that $\theta = 1$) or weakened (negative entails that $\theta = 0$).[2] The values assumed by these parameters, as a function of the conditions of reinforcement, are the key objects of our study.

[2] In our analysis programs, we let the learning variables go negative to indicate decrementing ($\theta = 0$), extract the sign of the parameters to set their direction toward floor (when $\pi < 0$, $\theta = 0$) or ceiling (when π 0, $\theta = 1$), and use their absolute value $|\pi|$ to adjust the distance traveled toward those limits, as in Equation 4. Thus, we refrain from imposing our expectations about what the directions of events should be on behavior.

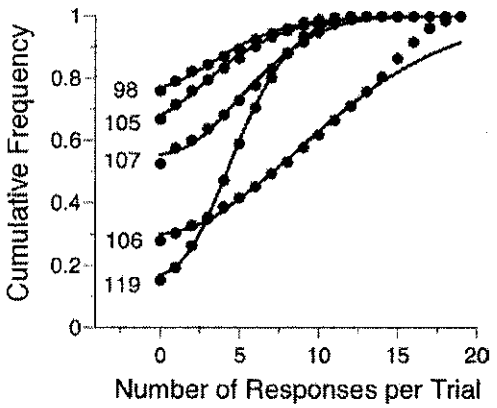

Figure 4. The cumulative frequency of trials containing 0, 1, 2, ... responses. The data are from all trials of the last condition of Experiment 1. The curves are drawn by the Weibull response rate model (Equation 1), using the distribution function rather than the density.

Notice that this model makes no special provision for whether a response and food co-occurred on a trial. It is a model of persistence, or behavioral momentum, and Pavlovian conditioning of the CS. Because these factors may always be operative, it is presented first, and the role of Skinnerian response–outcome associations is subsequently evaluated. The model also takes no account of warm-up or cool-down effects that may occur as each session progresses. Covarying these out could only help the fit of the models to the residuals, but it would also put one more layer of parameters between the data and the reader's eye.

The matrix of Table 2 is referred to as the Momentum/Pavlov model, or MP model. By calling it a model of momentum, we do not mean that a new hypothetical construct is invoked to explain the data. It is simply a way of recognizing that response strength will not in general change maximally on receipt of food or extinction. Just how quickly it will change is given by the parameters π_P and π_Q. If these are 1, there will be no lag in responsiveness and no need for the construct; if they equal 0, the pigeon will persist at the current probability indefinitely, and there will be no need for the construct of conditioning. In early models without momentum (i.e., where these parameters were de facto 1), goodness of fit was at least e^{10} worse than in the model as developed here, and typically worse than the comparison model, described later.

Implementation

To fit the model to the data, we use Equation 1 to calculate the probability of the observed data given the model. Two hypothetical cases illustrate the computation of this probability:

1. Assume the following: no key pecks on trial i, the predicted probability of being in the response state $s_i = 2/3$, and the Weibull parameters were $\alpha = 2$, $c = 6$. Then the probability of the data (0 responses) given the model $p(d_i|m)$ is the probability of being
   (a) out of the response state, $1 - s_i$, times the probability of no response when out of the state, 1.0: $(1 - 2/3) \cdot 1 = 1/3$; to that, add the probability of being
   (b) in the state, times the probability of no responses in the state: $2/3w(n, 2, 6) = 2/3 \cdot 0$;
   (c) the sum of which equals $p(d_i = 0|m) \approx .333 + 0 \approx 0.333$.
2. If four pecks were made on trial i, given the same model parameters, then the probability would be $p(d_i = 4|m) = 0 + 2/3w(n, 4, 6), \approx 0.142$.

The natural logarithm of these conditional probabilities gives the index of merit of the model for this trial: That is, it gives the log-likelihood (LL_i) of the data (given the model) on trial i. These logarithms are summed over the thousands of trials in each condition to give a total index of merit LL (Myung, 2003). Case 1 added $\ln(1/3) \approx -1.1$ to the index, whereas Case 2 added $\ln(.142) \approx -1.9$, its smaller value reflecting the poorer performance of the model in predicting the data on that trial. The parameters are adjusted iteratively to maximize this sum and thus to maximize the likelihood of the data given the model. The LL is a sufficient statistic, so that it contains all information in the sample relevant to making any inference between the models in question (Cox & Hinkley, 1974).

A Base (Comparison) Model

Log-likelihoods are less familiar to this audience than are coefficients of determination—the proportion of variance accounted for by the model. The coefficient of determination compares the residual error (the mean square error) with that available from a simple default model, the mean (whose error term is the variance); if a candidate model can do no better than the mean, it is said to account for 0% of the variance around the mean. In like manner, the maximum likelihood analysis becomes more interpretable if it is compared with a default, or base, model. The base model we adopt has a structure similar to our candidate model: It uses Equation 1 and updates the probability of being in the response state as a moving average of the recent probability of a response on a trial:

$$s_{i+1} = \gamma P_i + (1 - \gamma)s_i,\ 0 < \gamma < 1, \tag{5}$$

where gamma (γ) is the weight given to the most recent event and P takes a value of 1 if there was a response on the prior trial and 0 otherwise. Equation 5 is an exponentially weighted moving average, and can be written as $s_{i+1} = s_i + \gamma(P_i - s_i)$, which reveals its similarity to the Momentum/Pavlovian model, with the one parameter γ replacing the four contingency parameters of that model. The base model attempts to do the best possible job of predicting future behavior from past behavior, with its handicap being ignorance as to whether food or extinction occurred on a

Table 2
Momentum/Pavlovian Model With Events Mapped Onto Direction and Rate Parameters

Event	Representation
Peck	P: $(\pm)\pi_P$
No peck (quiet)	Q: $(\pm)\pi_Q$
Food	F: $(\pm)\pi_F$
Empty/extinction	E: $(\pm)\pi_E$

Note. The parentheticals indicate whether the learning process is driving behavior up (positive entails that $\theta = 1$) or down (negative entails that $\theta = 0$); the rate parameters themselves are always positive.

trial. It is a model of perseveration, or momentum, pure and simple. It invokes three explicit parameters: γ, α, and c. Other details are covered in the Appendix.

An Index of Merit for the Models

The log-likelihood does not take into account the number of free parameters used in the model. Therefore, we use a transformation of the log-likelihood that takes model parsimony into account. The AIC (Burnham & Anderson, 2002) corrects the log-likelihood of the model for the number of free parameters in the model to provide an unbiased estimate of the information-theoretic distance between model and data:

$$\mathrm{AIC} = 2(n_P - LL), \tag{6}$$

where n_P is the number of free parameters and LL is the total log-likelihood of the data given the model. (We do not require the secondary correction for small sample size, AIC_C.)

We compare the models under analysis with the simple perseveration model, the base model, characterized by Equations 1 and 5. This comparison is done by subtraction of their AICs. The smaller the AIC, the better the adjusted fit to the data. There are $n_P = 3$ parameters in the base model (hereinafter *base*), and 6 parameters (8 in later versions) in the candidate model (hereinafter *model*), so the relative AIC is

$$\begin{aligned}\mathrm{Merit} = \mathrm{Relative\ AIC} &= \mathrm{AIC(Base)} - \mathrm{AIC(Model)}\\ &= 2(3 - \mathrm{LL_B}) - 2(6 - LL_M)\\ &= 2(LL_M - LL_B) - 6.\end{aligned} \tag{7}$$

Because logarithms of probabilities are negative, the actual log-likelihoods are negative. However, our index of merit subtracts the model AIC from the base AIC so that it is generally positive and is larger because the model under purview is better than the base model. The relative AIC is a linear function of the log-likelihood ratio of model to base (LLR = log[(likelihood of model)/(likelihood of base)]). Because of the additional free parameters of the model, it must account for e^3 as much variance as the base model just to break even. An index of merit of 4 for a model means that under that model, the data are e^4—approximately 50 times—as probable as under the base model, after taking into account the difference in number of free parameters. A net merit of 4 is our criterion for claiming strong support for one model over another. If the prior probabilities of the model under consideration and the base (or other comparison) model are deemed equal, Bayes's theorem tells us that when the index of merit is greater than 4 (after handicapping for excess parameters), then the posterior odds of the candidate model compared with the comparison is at least 50/1.

The base model is nested in the Pavlovian/Momentum model: Setting $\pi_Q = -\pi_P = \gamma$, and $\pi_F = \pi_E = 0$ reduces it to the base model. For summary data, we also display the Bayesian information criterion (BIC; Schwarz, 1978), which sets a higher standard for the admission of free parameters in large data sets such as ours: $\mathrm{BIC} \approx -2\mathrm{LL} + k\ln(n)$. We now apply this modeling framework to the results of the first experiment.

The index of merit is relative to the default base model, just as the proportion of variance accounted for in quotidian use is relative to a default model (the mean). If the default model is very bad, the candidate model looks very good by comparison. If, for instance, we had used the mean response rate or probability over all sessions in a condition as the default model, the candidate would be on the order of e^{400} better in most of the experiments. A tougher test would be to contrast the present linear operator model with the more sophisticated models in the literature, but that is not, per reviewers' advice, included here.

Applying the Models

The AIC advantage of the Pavlovian model over the base model averaged 43 AIC points for the first four conditions, in which only 2 of the 24 Subject × Condition comparisons did not exceed our criterion for strong evidence (improvement over the base model by 4 points). For the last, $p = .05$, condition, the average merit jumped to 183 points. Figure 5 shows that the Weibull response rate parameters were little affected by the varied conditions. The average value of c, 8.2, corresponded to a mean of 7.3 responses per trial on trials on which a response was made (the mean is primarily a function of c, but also of α). The average value of the shape parameter α was 2.4: The modal response distribution looked like that of Pigeon 113 in Figure 3. The values of these Weibull parameters were always essentially identical for the base and MP models and were therefore shared by them.

The values of gamma (γ), the perseveration constant in the base model, averaged .038 in the first four conditions and increased to .100 in the $p = .05$ condition. This indicates that there was a greater amount of character—more local variance—in this last condition for the moving average to take advantage of, a feature that was also exploited by the MP model. There was no change in the rate of responding—given that the pigeon is in a response state—as indicated by the constancy of c. All of the decrease seen in Figure 2 was the result of changes in the probability of entering a response state, as given by the model and seen in the model's predictions, traced by the lines in the bottom panel of Figure 2.

The weighted average parameters of the MP model are shown in the bottom panel of Figure 5 (the values for each subject were weighted by the variance accounted for by the model for that subject). Just as autoshaping is fastest with longer ITIs, the impact of the π_F and π_P parameters increases markedly with ITI. The increase in π_F indicates that at long ITIs, the delivery of food, independent of pigeons' behavior, increases the probability of a response on the next trial. It increases 11% of its distance toward 1.0 in the 20-s ITI condition, up to 28% in the 80-s ITI condition. Also notice that π_F is everywhere of greater absolute magnitude than π_E, a finding consistent with that of Rescorla (2002a, 2002b).

The increase in π_P indicates that pecking acquires more behavioral momentum as the ITI is increased. The parameter π_Q remains around −7% over conditions (although a drop from −5% to −10% in the first and second replication of the 40-s conditions accounts for the decrease in probability of responding in the second exposure). A trial without a response decreases the probability of a response on the next by 7%. The parameter π_E hovers at zero for the short and intermediate ITIs: Extinction trials add no new information about the pigeons' state on the next trial and do not change behavior from the *status quo ante*. Under these conditions, extinction does not discourage responding. The law of disuse, rather than extinction, is operative: If a pigeon does not

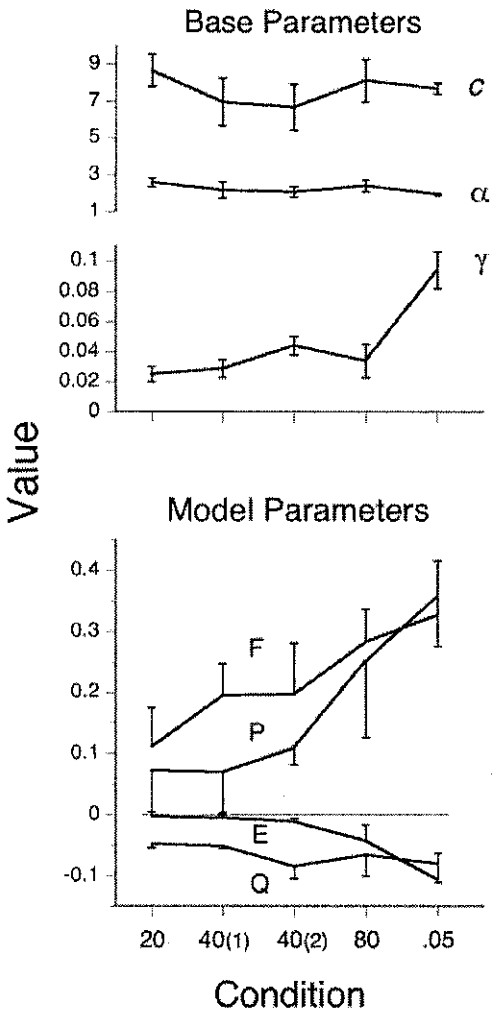

Figure 5. The average parameters of the base and Momentum/Pavlovian models for Experiment 1. The first four conditions are identified by their intertrial interval (ITI), with the first and second exposure to the 40-s ITI noted parenthetically. The same Weibull parameters, c and α, were used for both models. In the last condition, the probability of hopper activation on a trial was reduced from .1 to .05, with ITI = 40. The error bars delimit the standard error of the mean. F = food; P = response; E = no food; Q = no response.

respond, momentum in not responding (measured by π_Q) carries response probability lower and lower. At the longest ITI and in the p = .05 condition, extinction trials decrease the probability of being in a response state on the next trial by 4% and 10%, respectively. When reinforcement is scarce, both food and extinction matter more, as indicated by increased values of π_F and π_E, but the somewhat surprising effect on π_E is modest compared with the former. The importance of food when it is scarce is substantial—with π_F increasing more than 30% in the p = .05 condition. The fall toward extinction of responding, driven by π_Q and π_E, is arrested only by delivery of food, a strong tonic to responding (π_F), or an increasingly improbable peck, which, as reflected in π_P, is associated with substantially enhanced response probabilities on the next trial.

We may see how close the simulations look to the real performance, such as that shown in Figure 1. We did this by replacing the pigeon with a random number generator, using the average parameters from the first condition, shown in Figure 5. The probability of the generator's entering a response state was adjusted using the MP model, and when in the response state, it emitted responses according to a Weibull distribution with the parameters shown in the top of Figure 5. Figure 6 plots the resulting data in a fashion similar to that shown in Figure 1 (a running average of 25 trials). Comparison of the three panels cautions how different a profile can result from a system operating according to the same fixed parameters once a random element enters. Analyses are wanted that can deal with such vagaries without recourse to averaging over a dozen pigeons. By analysis on a trial-by-trial basis, the present models attempt to take a step in that direction.

These graphs have a similar character to those generated by the pigeons (although they lack the change in levels shown by Pigeon 98 in Figure 1, a change not clearly shown by most of the other subjects). The challenge is how to measure "similar" in a fashion other than impressionistically. Killeen (2003) showed that responding had a fractal structure, and given the self-similar aspect of these curves, that is likely to be the case here. However, the indices yielded by fractal analysis throw little new light on the psychological processes. The AIC values returned by the model provide another guide for those comfortable with likelihood analyses; they tell us how good the candidate model is relative to a plausible contender.

The variance accounted for in the probability of responding will look pathetic to those used to fitting averaged data: It averages around 10% in Experiment 1 and around 15% in the remaining experiments. But even when the probability of a response on the next trial is known exactly, there is probabilistic variance associate with Bernoulli processes such as these, in particular, a variance of $p(1-p)$. The parameters were not selected to maximize variance accounted for, and in aggregates of data much of the sampling error that is inevitable in single-trial predictions is averaged out. When the average rate over the next 10 trials, rather than the single next trial, is the prediction, the variance accounted for by the matrix models doubles. At the same time, the ability to speak to the trial-by-trial adjustment of the parameters is blunted. Other analyses, educing predictions from the model and testing them against the data, follow.

Hazard Functions

That π_Q and π_E are negative in the p = .05 condition makes a strong prediction about sojourns away from the key: When a pigeon does not respond on a trial, there is a greater likelihood that it will not respond on the next, and yet greater on the next, and so on. Only free food (or the unlikely peck despite the odds) saves it. The probability of food is 5%, but the cumulative probability is continually increasing, reaching 50% after 15 trials since the first nonresponse. The probability of returning to the key should decrease at first, flatten, and then eventually increase. A simple test of this prediction is possible: Plot the probability of returning to the key after various numbers of quiet trials. In making these plots, each point has to be corrected for the number of opportunities left for the next quiet trial. Such plots of marginal probabilities are called *hazard functions*. If there is a constant probability of return-

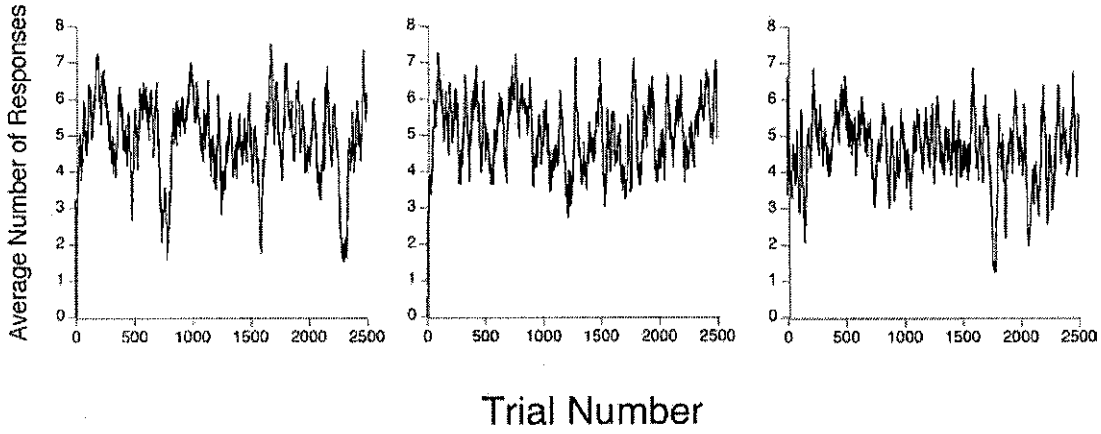

Figure 6. Moving averages of the number of responses per 5-s trial over 25 trials from three representative "statrats," characterized by the average parameters of real pigeons in the first condition, 40-s ITI. The only difference among these three panels is the random number seed for Trial 1. Compare with Figure 1.

ing to the key, as would be the case if returns were at random, the hazard function would be flat. The earlier analysis predicts hazard functions that decrease under the pressure of the negative parameters and eventually increase as the cumulative probability of the arrival of food increases.

Figure 7 shows the functions for individual pigeons (truncated when the residual response probabilities fell to 1%). They show the predicted form. The filled squares show the averaged results of running three "statrats" in the program, with parameters taken from the .05 condition of Figure 5. If the model controls behavior the way it is claimed, the output of the statrats should resemble that of the pigeons. There is indeed a family resemblance, although the statrats' hazard function was more elevated than the average of the pigeons, indicating a greater eagerness to return to the operandum than was the case for the birds. Note also that the predicted

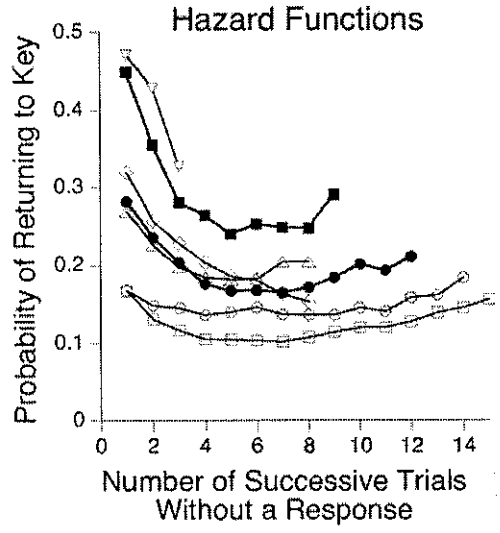

Figure 7. The marginal probability of ending a run of quiet trials. The unfilled symbols are for individual pigeons, and the filled circles represents their average performance. The hazard function represented by filled squares comes from simulations of the model.

decrease—first 8% of the distance to 0 from π_Q and then another 11% from π_E—predicts a decrease to 82% of the initial value after the first quiet—that is, from about 0.45 to 0.37 for the statrats and from about .28 to about .23 for the average pigeon. These are right in line with the functions of Figure 7. The eventual flattening and slow rise in the functions is due to the cumulative effects of π_E.

Is Momentum Necessary?

In the parameters π_P and π_Q, the MP model invokes a trait of persistence or momentum, which may appear supererogatory to some readers. However, the base model, the linear average of the recent probability of responding, actually proves a strong contender to the MP model. It embodies the adage "The best predictor of what you will do tomorrow is what you did today." It is the simplest model of persistence, or momentum. We may contrast it with a MP-minus-M model: That is, adjust the probability of responding on the next trial as a function of food or extinction on the current trial, while holding the momentum parameters at zero. Even though the base model has one fewer parameter, it easily trumps the MP-minus-M model. For example, for Pigeon 98, the median advantage of the MP model over the base model was 14 AIC points in the .1 condition and 58 points in the .05 condition. But without the momentum aspect, the MP-minus-M model tumbles to a median of 106 points below the base model in the .1 conditions and 540 points below it in the .05 condition. However one characterizes the action of the π_P and π_Q parameters, their presence in the model is absolutely necessary. This analysis carries the within-session measurement of resistance to change reported by Tonneau, Ríos, and Cabrera (2006) to the next level of contact with data.

Operant Conditioning

What is the role of response–reinforcer pairing in controlling this performance? The first analysis of these data (unreported here) consisted of a model involving all interaction terms, and those alone: PF, PE, QF, and QE. Although this interaction model was substantially better than the base model (18 AIC units over all

conditions, 73 in the $p = .05$ condition), it was always trumped by the MP model (51 AIC units over all conditions, 183 in the $p = .05$ condition).

In search of evidence of Skinnerian conditioning, we asked whether there was a correlation between the number of responses on a trial and the probability of responding on the next trial. Any simple correlation could be just due to persistence; however, if response–reinforcer contiguity is a factor in strengthening responding, then that correlation should be larger for trials that end with food (r_F) than for trials that end without food (r_E). When many responses occur on a reinforced trial, (a) there are more responses in close contiguity with the reinforcer and (b) the last of them is likely to be closer in time to the reinforcer than the case of trials with only a few responses. Therefore, there should be a positive correlation between number of responses on trials ending with food and number of responses on the next trial. It is different for trials that end without food: When many responses occur on a nonreinforced trial, there are many more instances of the response subject to extinction; this should not only undermine a positive correlation, it could drive it negative. We can therefore test for Skinnerian conditioning by correlating the number of responses on F and E trials that had at least one response (the predictors) with the presence or absence of a response on the next trial (the criterion). If contiguity of multiple responses with food strengthens behavior more than contiguity of one response to food, the correlation with subsequent responding should be larger when the trial was followed by food than when it was not. That is, we would expect $r_F > r_E$. We restrict the analysis to trials with at least one response so that the correlation is not simply driven by the information that the pigeon is in a response state, which we know from π_P has good predictive value.

We analyzed the data for all subjects from all conditions and found no evidence for value added by multiple response–reinforcer contiguity. For no pigeon was the average correlation between predictor and criterion greater when the predictor was followed by food than when it was not. The averages over all subjects and conditions were $r_F = 0.035$ and $r_E = 0.081$. With an average n of 150 for r_F and of 1,470 for r_E for each of the 29 pairs of correlations, the conclusion is unavoidable: Reinforcement on trials with multiple responses did not increase the probability of a response on the next trial any more than did extinction on trials with multiple responses.

Perhaps fitting a delay-of-reinforcement model from each response to an eventual reinforcer would show evidence of operant conditioning? This was our first model of these data, not reported here. We found no value added by the extra parameter (the slope of the delay-of-reinforcement gradient).

Convinced that there must be some way to adduce evidence of (adventitious) operant conditioning, we turned to the next analysis. It remains possible that reinforcement increases the probability of staying in the response state on the next trial: Possibly the commitment to a behavioral module (Timberlake, 1994), rather than the details of actions within the module, is what gets strengthened by reinforcement. To test this hypothesis, we added conditioning factors, π_{PF} and π_{PE}, to the model. If response–reinforcer contiguity added strengthening–prediction beyond that afforded by the independent actions of persistence and of food delivery, one or both of these parameters should take values above zero and should add significantly to predictive accuracy when it does. We measure accuracy with the AIC score; any increase (after handicapping for the added parameter) lends credibility, and increases by at least 4 constitute strong evidence.

The average value of π_{PF} across the 29 cases was 0.064: That is, the probability of a response on the next trial increased by 6% beyond that predicted by momentum and mere delivery of food (independent of the presence or absence of a peck). For 2 birds, 107 and 119, there was no advantage, and π_{PF} remained close to zero, as often negative as positive. Of the 19 remaining Pigeon × Condition cases, 11 showed an AIC advantage for the added parameter, 5 of them meeting our criterion for strong evidence. Of the 4 birds that showed evidence of Skinnerian conditioning, the average value of π_{PF} was 8%, which may be compared with 16% for π_P and 14% for π_F. Examining the data on a condition-by-condition basis, all 4 of these pigeons showed evidence of Skinnerian conditioning in the 20-s ITI condition (3 of them, strong evidence), and in all cases but one π_{PF} was larger than either π_P or π_F. Across all 6 pigeons, the advantage of adding the contiguity parameter was 2.6 AIC points at the 20-s ITI 20, 0.8 at the 40-s ITI, and −1.5 at the 80-s ITI. (The negative value indicates that the cost of the extra parameter in Equation 7 is not repaid by increased predictive ability.) In the $p = .05$ condition, the total advantage conferred by the π_{PF} parameter increased to 6.4. (When the Skinnerian parameter comes into play, there is typically a readjustment of the other parameters that had been tasked with picking up the slack.) The Skinnerian extinction parameter π_{PE} was almost never called into play and exerted negligible improvement in the predictions. Parameter values for each pigeon are listed in Table 3; indices of merit, in Table 4.

These results indicate that Skinnerian conditioning was strongest where Pavlovian conditioning was weakest—whether that weakness was the result of a small ITI-to-trial ratio (20-s ITI) or to a less reliable CS ($p = .05$). This is consistent with the findings of Woodruff, Conner, Gamzu, and Williams (1977). We retain π_{PF} and π_{PE} in subsequent analyses, in which we call the full model the Momentum/Pavlovian/Skinnerian (MPS) model.

Implications for Acquisition and Extinction

On the basis of Equation 4 and the parameters shown in Figure 5, we may predict the courses of acquisition and extinction in similar contexts—it is given by Equation A5 in the Appendix. For the parameters in Figure 5, the MPS model predicts faster acquisition at longer ITIs—the trial spacing effect, along with an increasing dependence on the original starting strength (derived from hopper training) as trial spacing decreases. Pretraining plays a critical role in determining the speed of acquisition (Davol, Steinhauer, & Lee, 2002; Downing & Neuringer, 2003); the current analysis suggests that this is in part because of elevation of the initial probability of a response, s_0, possibly through generalization of hopper stimuli and key stimuli (Sperling, Perkins, & Duncan, 1977; Steinhauer, 1982). Conditioning of the context proceeds rapidly, however, so that more than a few pretraining trials in the same context will slow the speed of subsequent key conditioning (Balsam & Schwartz, 2004).

The predicted number of trials to criterion show an approximate power-law relation between trials to acquisition and the ITI (Gibbon et al., 1977). Those researchers, along with Terrace, Gibbon, Farrell, and Baldock (1975), found that both acquisition and re-

Table 3
Parameter Values of the Base and Momentum/Pavlovian/Skinnerian Models for the Data of Experiment 1

Bird no.	Parameter	20	40_1	40_2	80	.05
98	γ	.02	.03	.05	.02	.08
	c	8.78	6.93	5.31	7.77	5.90
	α	3.00	2.08	1.55	2.30	1.76
	P	.00	.02	.04	−.01	.27
	Q	−.02	−.03	−.05	−.01	−.19
	F	.00	.05	.03	.22	.00
	E	.00	.00	.00	.00	.01
	PF	.20	.00	.13	.00	.00
	PE	.00	.00	.00	.00	−.08
105	γ	.05	.04	.11	.10	.12
	c	5.69	5.12	7.18	7.49	5.74
	α	1.56	1.31	2.13	2.33	1.62
	P	.04	.05	.19	.61	.32
	Q	−.04	−.04	−.24	−.15	.02
	F	.03	.00	.56	.33	.34
	E	.00	.00	.03	−.10	−.30
	PF	.14	.04	.34	.00	.00
	PE	.00	.02	−.04	.00	.28
106	γ	.05	.02	.07	.02	.12
	c	12.71	13.88	13.29	14.35	12.40
	α	3.54	4.49	3.55	4.25	2.36
	P	.05	.10	.18	.17	.30
	Q	−.06	−.07	−.14	−.07	−.16
	F	.00	.00	.27	.27	.54
	E	.01	.03	.00	−.02	−.03
	PF	.19	.43	.16	.10	.00
	PE	.00	−.01	−.01	.00	−.01
107	γ	.02	.05	.05	.01	.04
	c	8.56	8.20	7.47	8.69	8.00
	α	2.57	2.20	2.11	2.22	2.00
	P	.06	.00	.04	.00	.06
	Q	−.03	−.04	−.06	−.01	−.05
	F	.29	.27	.12	.30	.30
	E	−.02	.00	.00	−.01	.00
	PF	.00	.00	.00	.00	.00
	PE	.00	.00	.00	.00	−.04
113	γ	.02	.05	.04	.03	
	c	6.90	5.45	4.79	6.45	
	α	2.51	2.22	1.76	2.46	
	P	.02	.06	.06	.68	
	Q	−.02	−.06	−.04	.12	
	F	.00	.14	.00	−.06	
	E	.00	−.01	.00	−.16	
	PF	.05	.00	.00	.00	
	PE	.00	.00	−.01	.00	
119	γ	.01	.01	.03	.02	.06
	c	8.12	7.17	6.41	7.78	6.76
	α	3.17	2.92	2.49	2.30	2.44
	P	.21	.28	.04	−.01	.48
	Q	−.05	−.07	−.04	−.01	−.17
	F	.90	.65	.31	.23	.50
	E	−.02	−.03	.00	.00	.00
	PF	−.02	.00	−.01	.00	.00
	PE	.09	.17	.00	.00	−.04

Note. γ = the rate constant for the comparison base model; c = the Weibull rate constant; α = the Weibull shape constant; the remaining letters indicate the rate constants brought into play on trials with (P) or without (Q) a response; with (F) or without (E) food; and the Skinnerian interaction terms PF and PE.

sponse probability in steady-state performance after acquisition covaried with the ratio of trial duration to ITI. Gibbon, Farrell, Locurto, Duncan, and Terrace (1980) found the permutation that partial reinforcement during acquisition had no effect on trials to acquisition, when those were measured as reinforced trials to acquisition. This is consistent with the acquisition equations in the Appendix. Despite these tantalizing similarities, however, the obvious difference in the parameters for the $p = .1$ and $p = .05$ conditions seen in Figure 5 undermines confidence in extrapolations to typical acquisition, where $p = 1.0$.

It is possible to test the predictions for extinction within the context of the present experiments, where parameter change is not so central an issue, for there were long stretches (especially in the $p = .05$ condition) without food. The relevant equation, transplanted from the Appendix (Equation A6), is

$$s_{i+1} = s_i(1 - \pi_E)[1 + \pi_{P-Q}(1 - s_i)], \tag{8}$$

where the strength s_{i+1} gives the probability of entering a response state on that trial. All parameters are positive, with asymptotes of 0 or 1 used as appropriate to the signs shown in Figure 5. Neither π_F nor π_{PF} appear because there are no food trials in a series of extinction trials, and π_{PE} is typically small and its work can be adequately handled by π_E. The probability of responding on a trial decreases with π_E as expected (note the element $-\pi_E s_i$)—substantially when s_i is large, not much at all when s_i is small. Only the difference in the two momentum parameters, $\pi_P - \pi_Q$, affects the prediction; for parsimony, we collapse those into a single parameter representing their difference, $\pi_{P-Q} = \pi_P - \pi_Q$. Equation 8 makes an apparently counterfactual prediction.

A Surprising Prediction

Inspection of Figure 5 shows that π_{P-Q} is generally positive. Because it multiplies the probability of not responding (Equation 8 contains the element $\pi_{P-Q}[1 - s_i]$), on average π_{P-Q} increases the probability of responding on each trial and does so more as s_i gets small. Depending on the specific value of the parameters, this restorative force may be sufficient to forestall extinction. To show this more clearly, we solve Equation 8 for its fixed point, or steady state, which occurs when $s_{i+1} = s_i$:

$$s_\infty = 1 - \frac{\pi_E}{\pi_{P-Q}(1 - \pi_E)}, \tag{9}$$

where $0 < \pi_{P-Q}(1 - \pi_E) \leq \pi_E$; this is the level at which responding is predicted to stabilize after a long string of extinction trials.

If response probability fluctuates below the level of s_i, the next response (if and when it occurs, which it does with probability s_i) will drive probability up, and if it fluctuates above this level, the next trial will drive it down. For responding to extinguish, it is necessary that the force of extinction be greater than the restoring force:

$$\pi_E \geq \frac{\pi_{P-Q}}{1 + \pi_{P-Q}}. \tag{9}$$

This is automatically satisfied whenever momentum in quiescence, π_Q, is greater than momentum in pecking π_P—whenever π_{P-Q} is negative. That is especially likely to be the case in rich

Table 4
Indices of Merit for the Model Comparison of Experiment 1

Bird no.	Metric[a]	20	40_1	40_2	80	.05
98	CD	0.03	0.06	0.17	0.06	0.19
	AIC	9	−1	32	29	115
	BIC	−17	−18	15	20	97
105	CD	0.07	0.02	0.17	0.13	0.16
	AIC	57	72	162	105	375
	BIC	38	55	134	91	352
106	CD	0.17	0.03	0.18	0.05	0.19
	AIC	47	40	69	5	217
	BIC	22	17	46	−14	193
107	CD	0.04	0.09	0.14	0.05	0.11
	AIC	101	40	13	18	88
	BIC	82	34	2	8	70
113	CD	0.04	0.07	0.30	0.03	
	AIC	12	27	38	24	
	BIC	−7	10	19	9	
119	CD	0.02	0.01	0.02	0.07	0.07
	AIC	57	40	8	29	87
	BIC	25	17	−14	19	69
Group	CD	*0.06*	*0.05*	*0.16*	*0.06*	*0.14*
	AIC	*47*	*36*	*54*	*35*	*176*
	BIC	*24*	*19*	*34*	*22*	*156*

Note. Italics indicate averages over the group.
[a] The metrics of goodness of fit for the models are the coefficient of determination (CD), the Akaike information criterion (AIC), and the Bayesian information criterion (BIC). Values of the last two greater than 4 constitute strong evidence for the Momentum/Pavlovian/Skinnerian model.

contexts where quiescence on the target key may be associated with foraging in another patch or responding on a concurrent schedule. For the parameters in Figure 5 under $p = .05$, however, this is never the case; indeed, the more general inequality of Equation 10 is never satisfied. Therefore Equations 8 and 9 make the egregious prediction that the probability of responding will fall (with a speed dictated by π_E) to a nonzero equilibrium dictated by Equation 9. We may directly test this derivation by plotting the course of extinction within the context of dynamic reconditioning of these experiments. The best data come from the $p = .05$ condition, which contained long strings of nonreinforced responding. The courses of extinction, along with the locus of Equation 8, are shown in Figure 8.

Do Equations 8–10 condemn the birds to an endless Sisyphean repetition of unreinforced responding? If not, what then saves them? Those equations are continuous approximations of a finite process. Because the right-hand side of Equation 8 is multiplied by s_i, if that probability ever does get close enough to 0 through a low-probability series of quiescent trials, it may never recover. It is also likely that after hundreds of extinction trials, the governing parameters would change, as they did across the conditions of this experiment, releasing the pigeons to seek more profitable employment. The maximum number of consecutive trials without food in this condition averaged around 120. Surely over unreinforced strings of length 95 through 120, the probability of responding would be decreasing toward zero. Such was the case for 2 pigeons, 98 and 107, whose response probability decreased significantly (using a binomial test) to around 5% (the drift for 107 is already visible in Figure 8). The predicted fixed points and obtained probabilities for another 2, 105 and 119, were invariant, .20→.19 and .77→.78, respectively; Pigeon 106 showed a decrease in probability, .61→.54, that was not significant by the binomial test. The substantial momentum shown in Figure 8, and extended in some cases by the binomial analysis, resonates with the data of Killeen (2003; cf. Sanabria, Sitomer, & Killeen, 2006), where some pigeons persisted in responding over many thousands of trials of negative automaintenance.

The validation of this unlikely prediction should, by some accounts of how science works, lend credence to the model. But it certainly could also be viewed as a fault of the model, in that it predicts the flatlines of Figure 8, when few pigeons, except perhaps those subjected to learned helplessness training, will persist in unreinforced responding indefinitely. On that basis we could reject the MPS model because it does not specify when the pigeons will abandon a response mode (as reflected in changes in the persistence parameters). Conversely, the data of Figure 8 indict models that do not predict the plateaus that are clearly manifest there. On that same basis, we could therefore reject all of the remaining models. But perhaps the most profitable path is to reject Popper in favor of MPS, which permits tracking of parameters over an indefinite number of trials, to see when, under extended dashing of expectations, those begin to change.

Equation 8 contains the element $s_i(1 - s_i)$: The product of the probability of a response and its complement enters the prediction of response probability on the next trial. This element is the core of the "logistic map." Depending on the coefficient of this term, the pattern of behavior it governs is complex and may become chaotic. This, along with the multiple timescales associated with the rate parameters, is the origin of the chaos that Killeen (2003) found in the signatures of pigeons responding over many trials of automaintenance and the factor that gives the displays in Figures 1 and 6 their self-similar character.

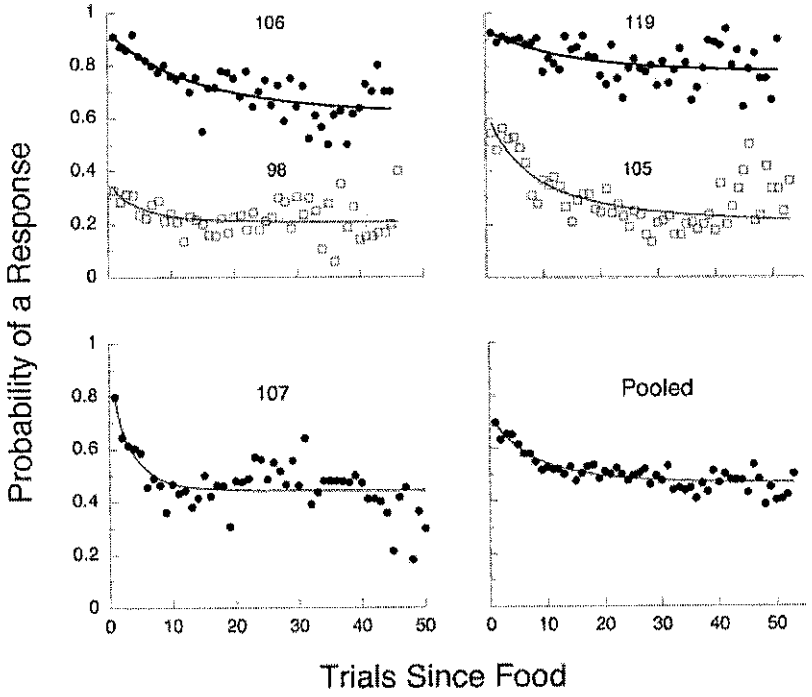

Figure 8. The average probability of responding as a function of the number of trials since reinforcement, from the $p = .05$ condition. The number of observations decreases by 5% from one trial to the next, from hundreds for the first points to 10 for the last displayed. The curve comes from Equation 8, using parameters π_{P-Q} and π_E fit to these data.

Experiment 2: Trial Duration

The trial-spacing effect depends on the duration of both the ITI and the trial; arrangements that keep that ratio constant often yield about the same speed of acquisition of responding. Therefore, to test the generalizability of both the response rate model and the MPS model, we systematically varied trial duration in this experiment.

Method

Subjects and Apparatus

Six experienced adult homing pigeons, housed in similar conditions as in Experiment 1, served. Pigeons 105, 106, 107, 113, and 119, who had participated in Experiment 1, were joined by 108, who replaced 98. The apparatus remained the same.

Procedure

Seven sessions of extinction were conducted before beginning this experiment. In extinction, stimulus conditions were similar to those of Experiment 1, but the ITI was 35 s and trial duration was 10 s; no food was delivered ($p = 0$). In experimental conditions, food was delivered with $p = .05$, the ITI remained 35 s, and trial duration varied, starting at 10 s for 13 sessions. Then half the subjects went to the 5-s CS condition, and half went to the 20-s CS condition. Finally, the 10-s CS was recovered. All sessions lasted 150 trials; Table 5 reports the number of sessions per condition.

Results

In the last session of extinction, the typical pigeon pecked on 3% of the trials. This is a lower percentage than shown in Figure 8 because it follows six sessions of extinction. Extinction happens. On moving to the first experimental condition, this proportion increased to an average of 75%. The average response rates and probabilities of responding are shown in Figure 9. Both rates and probabilities decreased as CS duration increased. Also shown are average rates from 4 pigeons studied by Perkins et al. (1975) for CS durations of 4, 8, 16, and 32 s for pigeons maintained on probabilistic ($p = 1/6$) Pavlovian conditioning schedules, with an ITI of 30 s. (The average rate at 32 s was 0.2 responses per second). The higher rates for Perkins et al.'s subjects are probably due to their higher rates of reinforcement (1/6 trials compared with our 1/20). The decrease in response rate with CS duration is consistent with the data of Gibbon et al. (1977), who found

Table 5
Conditions of Experiment 2

Order	Trial duration (seconds)	Sessions
1	10	13
2	5, 20	13
3	20, 5	13
4	10	14

Note. Half the subjects experienced the extreme trial durations in the order 5 s, 20 s; half experienced them in the other order.

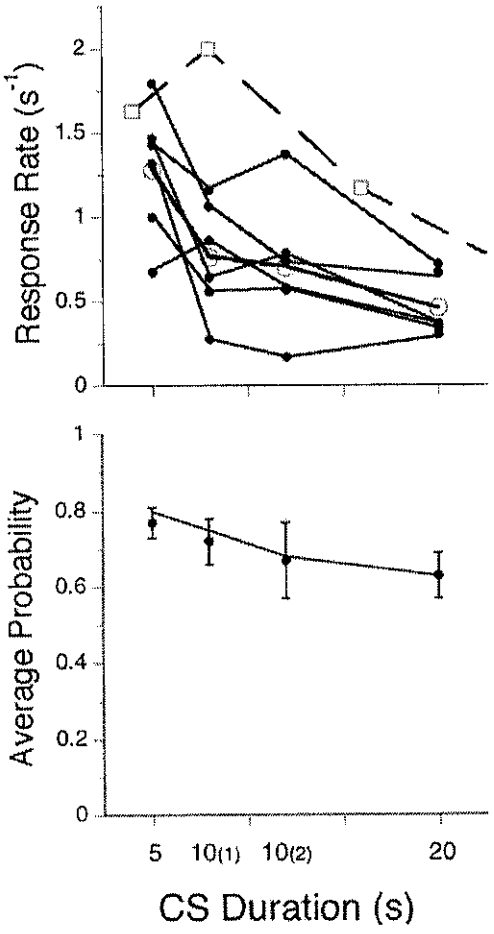

Figure 9. Data from Experiment 2. Top: average response rate (dots) for each subject. Open circles gives average rate and squares represent data from Perkins et al. (1975). Bottom: Average probability of making at least one response on a trial averaged over pigeons; bars give standard errors. Unbroken lines in both panels are from the Momentum/Pavlovian/Skinnerian model. CS = conditioned stimulus.

that rate decreased as a power function of trial duration, with exponent −0.75. A power function also described rates in these experiments, accounting for 99% of the variance in the average data, with exponent −0.74.

The MPS model continued to outperform the base momentum model, with an average advantage of 130 AIC units, giving it an advantage in likelihood of e^{130}. The parameters were larger than those found in the last condition of Experiment 1 (see Figure 10 and Tables 6 and 7) and on average did not show major changes among conditions, although the impact of a trial with food was greatest in the first condition studied, 10(1), and there were slight decreases in π_{PF} and π_Q as a function of trial duration. There was a moderate increase in the average number of responses emitted (c, top panel of Figure 10) as trial duration increased from 5 s to 20 s; the birds adjusted to having longer to peck before the chance of reinforcement carried them to the hopper.

Despite the importance of trial duration for acquisition of autoshaped responding, the changes in the conditioning parameters as a function of that variable were modest. They did, however, work in unison to decrease response rates as the CS duration increased. The only moderate changes may be due to the very short ITI in this series. The biggest effect was the transition into the first condition of the experiment, the first 10-s CS, after several sessions of extinction, where the Pavlovian and Skinnerian learning parameters π_F and π_{PF} were as large or larger than in any other conditions. Empty trials, although common, had little effect on behavior because π_E was generally very close to 0. In general, the dominance of π_F over π_{PF} (and the other parameters), especially at the longest CS duration, may have been due to the extended opportunity for nonreinforced pecking in that long CS condition.

In interpreting these parameters, and those of Figure 5, it is important to keep in mind that π_E was in play on 95% of the trials,

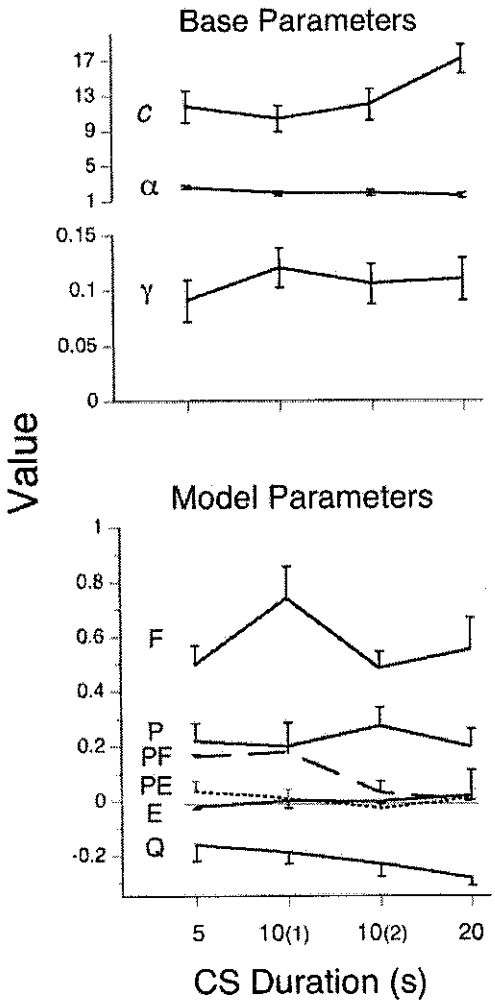

Figure 10. The average parameters of the base and Momentum/Pavlovian/Skinnerian models for Experiment 2. The conditions are identified by their trial duration, with the first and second exposure to the 10-s intertrial interval noted parenthetically. The same Weibull parameters, c and α, were used for both models. The error bars delimit the standard errors of the mean. π_{PF} is traced by a dashed line, and π_{PE} by a dotted line. F = food; P = response; E = no food; Q = no response; PF and PE = Skinnerian interaction terms.

Table 6
Indices of Merit for the Model Comparison of Experiment 2

Bird no.	Metric[a]	5	10_1	10_2	20
105	CD	0.01	0.32	0.26	0.29
	AIC	5	347	365	360
	BIC	0	319	342	338
106	CD	0.27	0.07	0.26	0.15
	AIC	169	106	285	163
	BIC	152	79	262	141
107	CD	0.25	0.12	0.22	0.11
	AIC	193	141	172	119
	BIC	170	118	155	97
108	CD	0.10	0.14	0.08	0.05
	AIC	79	127	49	37
	BIC	62	104	32	20
113	CD	0.23	0.15	0.04	0.05
	AIC	183	214	40	34
	BIC	155	197	12	17
119	CD	0.12	0.11	0.12	0.10
	AIC	75	56	81	54
	BIC	53	45	53	32
Group	CD	0.18	0.12	0.22	0.10
	AIC	124	134	127	87
	BIC	107	111	104	64

[a] The metrics of goodness of fit for the models are the coefficient of determination (CD), the Akaike information criterion (AIC), and the Bayesian information criterion (BIC). Values of the last two greater than 4 constitute strong evidence for the Momentum/Pavlovian/Skinnerian model.

either π_P or π_Q on every trial, π_F on 5% of the trials, and π_{PF} on fewer than 5% of the trials. Thus, a trial with food in this experiment would move response strength a very substantial 60% of the way to maximum—but this happened only rarely.

Once again, the quiescence parameter π_Q was the primary force driving the probability of entry into the response state toward 0, having a mean value of −.215. This value, so close to that for π_P (.222), indicates that the momenta of pecking and quiescence were, on average, essentially identical. This situation, $\pi_{P-Q} \approx 0$, will not sustain asymptotic responding above zero (see Equation 10); with so short an ITI, that is perhaps not surprising. The success of this prediction is illustrated in Figure 11 for the 5-s CS condition, which showed no evidence of a plateau. The slight negative acceleration is due to the dominance in the pooled data of profiles from pigeons whose π_{P-Q} was negative. This analysis may throw additional light on within-session partial reinforcement extinction effects (Rescorla, 1999) because different animals or paradigms may have quite different values of π_{P-Q}.

Because these conditions were preceded by seven sessions of extinction, the opportunity arises to trace the course of reacquisition for these birds and compare it with the model's profiles. The probability of a response on each of the first 100 trials, averaged over all pigeons and over a 7-trial moving window, is drawn as circles in Figure 12. The MPS model provides a closed-form solution to the acquisition curve. The equation is shown in the Appendix; the smooth acquisition curve is shown in Figure 12. The curve provides—at best—an idealized picture of the process because it assumes that response probability is dependent on the programmed probability of food, p, which is uniform over trials. MPS can do better than that by using the real thing—whether food was delivered or not—to inform its predictions. Replacing p with the trial-to-trial relative frequency across pigeons, represented by the hatch marks in the figure, and keeping all parameters otherwise the same, gives the jagged curve, a better characterization of the process. Figure 12 draws a graphic reminder of a point made by Benedict and Ayres (1972): Nonlinear dynamic processes, such as the course of learning, can be extremely sensitive to the particulars of stochastic processes. Generic models with asymptotic parameters, such as limiting values for p or even for s, will provide at best an idealization; the dynamics is in the details. The textbook-smooth curve shown in Figure 12 does not represent the character of the data. Over the full course of the experimental conditions, MPS easily supports its burden of parameters, as attested by its AICs, and carries us from milquetoast descriptions to the jagged profiles of Figure 12—to predictions with teeth.

All of the manipulations so far have been classic Pavlovian kinds, varying experimental parameters that did not interact with behavior, and those only modestly (see, e.g., Schachtman, 2004, for some modern developments). Although noncontingent food presentation can leave response–outcome associations intact (Colwill, 2001; Rescorla, 1992), all of the response–outcome associations up to this point were adventitious. We conducted the last series of experiments to complement those open-loop Pavlovian operations with closed-loop instrumental operations having more consistent contingencies.

Experiment 3: Fixed Ratio and Differential Reinforcement of Other Behavior Contingencies

Method

Subjects and Apparatus

Eight experienced adult homing pigeons, half having served in other experiments reported in this article, were used. They were maintained under the same conditions as the prior experiments. The apparatus was the same as used before.

Table 7
Parameter Values of the Base and Momentum/Pavlovian/Skinnerian Models for the Data of Experiment 2

Bird no.	Parameter	5	10_1	10_2	20
105	γ	0.01	0.17	0.13	0.14
	c	8.47	7.07	6.45	15.67
	α	3.03	1.53	1.58	1.73
	P	0.00	0.16	0.21	0.34
	Q	−0.01	−0.27	−0.33	−0.45
	F	0.13	0.83	0.55	0.71
	E	0.00	0.02	0.01	0.04
	E	0.00	0.02	0.01	0.04
	E	0.00	0.02	0.01	0.04
106	γ	0.12	0.09	0.15	0.18
	c	12.87	16.53	16.87	22.21
	α	3.32	2.00	1.78	1.30
	P	0.31	0.08	0.45	0.30
	Q	−0.19	−0.21	−0.30	−0.29
	F	0.68	0.35	0.44	0.60
	E	0.00	0.10	−0.14	0.02
	PF	0.00	0.62	0.00	0.00
	PE	−0.03	−0.02	−0.08	−0.03
107	γ	0.14	0.14	0.15	0.11
	c	8.95	9.26	11.73	17.65
	α	2.68	1.60	1.73	1.42
	P	0.00	0.00	0.19	−0.04
	Q	−0.14	−0.14	−0.16	−0.19
	F	0.69	0.69	0.48	0.38
	E	−0.03	−0.02	−0.02	0.01
	PF	0.00	0.00	0.00	0.00
	PE	0.17	0.18	0.00	0.15
108	γ	0.11	0.14	0.08	0.05
	c	5.70	119.00	41.00	29.00
	α	1.45	0.15	0.06	0.08
	P	0.10	0.29	0.02	0.00
	Q	−0.15	−0.24	−0.03	−0.08
	F	0.55	1.00	0.20	0.30
	E	0.04	0.03	0.00	−0.01
	PF	0.00	0.00	0.00	0.00
	PE	0.00	−0.02	0.00	0.10
113	γ	0.13	0.12	0.07	0.08
	c	17.22	10.77	10.08	11.40
	α	2.01	2.12	2.60	1.21
	P	0.39	0.54	0.12	0.06
	Q	−0.19	−0.10	−0.11	−0.09
	F	0.34	0.87	0.39	0.27
	E	−0.06	−0.10	0.02	0.02
	PF	0.34	0.00	0.02	0.00
	PE	0.00	0.00	0.00	0.00
119	γ	0.08	0.05	0.08	0.06
	c	10.04	15.25	16.99	19.68
	α	2.96	3.18	3.61	2.35
	P	0.26	0.00	0.41	0.05
	Q	−0.10	−0.02	−0.15	−0.09
	F	0.00	0.32	0.64	0.46
	E	−0.02	0.00	−0.02	−0.01
	PF	0.67	0.00	0.25	0.00
	PE	0.00	0.00	0.00	0.12

Note. γ = the rate constant for the comparison base model; *c* = the Weibull rate constant; α = the Weibull shape constant; the remaining letters indicate the rate constants brought into play on trials with (P) or without (Q) a response; with (F) or without (E) food; and the Skinnerian interaction terms PF and PE.

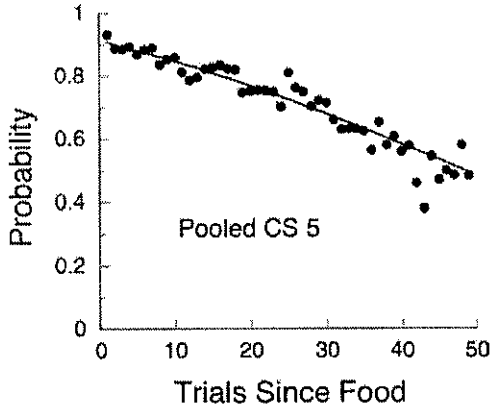

Figure 11. The average probability of responding as a function of the number of trials since reinforcement, from the 5-s conditioned stimulus (CS) condition of Experiment 2, pooled over subjects. The number of observations decrease by 5% from one trial to the next, from 485 for the first point to 29 for the last displayed. The curve comes from Equation 8, using parameters π_{P-Q} and π_E fit to these data.

Procedure

Before the experiment proper, six to seven sessions of extinction were conducted with a 35-s ITI and 10-s trial duration; the probability of food delivery was zero ($p = 0$). A preliminary series of experiments was conducted with $p = .05$. In these conditions, which we call fixed-ratio 3 (FR 3) and differential reinforcement of other behavior (DRO), reinforcement contingencies were intended to vary response–reinforcement contiguity in opposite directions. However, the low probability of exposure to those contingencies—5% of the trials at most, usually less—gave animals insufficient exposure to the Skinnerian contingencies: In a number

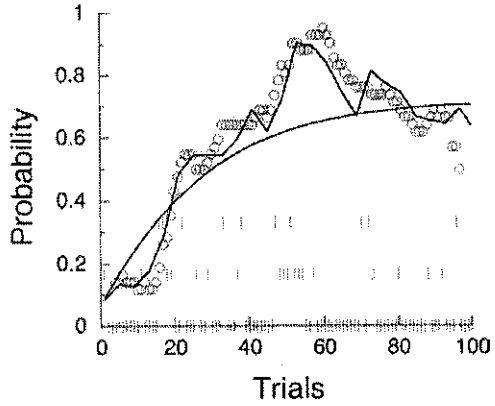

Figure 12. The average probability of responding as a function of the number of trials since the start of the 10-s conditioned stimulus condition of Experiment 2, pooled over subjects, and represented as a seven-trial moving average (circles). The hatch marks indicate trials on which 1 (plotted at $p = 1/6$) or 2 (plotted at $p = 2/6$) pigeons happened to have received food. In no cases did the same trial end with food for more than 2 pigeons. The smooth curve comes from the Momentum/Pavlovian/Skinnerian model, setting $p = .06$, with all other parameters fit to these data. The jagged curve comes from the same equation with the same parameters but uses the obtained relative frequency of food as given by the hatch marks.

of cases, there was no change consistent with the direction in which the contingencies were pushing. The probability of food was increased to $p = .1$, and the series replicated.

DRO. A DRO schedule was operative concurrently with baseline automaintenance contingencies, but only when food was programmed, which happened with a probability of $p = .10$. If an animal pecked during the 2 s preceding the delivery of food in a DRO trial, the trial was extended for an additional 2 s from the peck, until the pigeon had not pecked for 2 s, when food was finally delivered. All pigeons received 18 sessions of baseline training before being moved to the experimental conditions.

FR. A FR 3 schedule of reinforcement was operative concurrently with baseline automaintenance contingencies, but only when food was programmed. Thus, a trial in the FR 3 condition in which food was programmed (10% of the trials) would be terminated immediately by food delivery as soon as three key pecks were emitted. If three pecks were not emitted, the trial ended with noncontingent food presentation.

The order of experimental conditions was determined by the mean response rate during the last five sessions of baseline: Low responders were assigned first to DRO and high responders to FR 3. Table 8 shows the order of presentation of conditions and the number of sessions in each condition. In analyzing the data, all trials with a reinforcer were excluded from measurement of goodness of fit. This is because responding could have extended or shortened the trial duration, undermining comparison. This reduced the database by 10%.

Results

The contingencies, even though present on only 10% of the trials (DRO) or fewer (FR, which would end with food after 10 s if the FR contingency had not been met), were effective with most of the pigeons. This is consistent with the results of Locurto, Duncan, Terrace, and Gibbon (1980). The requirements were satisfied on 79% of the trials (median, with interquartile range from 68% to 81%). The effects of the contingencies on response rates and probabilities are displayed in Figure 13. Reinforcement contingencies clearly matter, by affecting both the probability of entering a response state and the number of responses emitted in that state.

Of the 24 Subject × Condition analyses, in 18 the MPS model exceeded our criterion for strong evidence (see Table 9). Averaged over all subjects, the AIC advantage for the MPS model over the base model was 38 points. Figure 14 displays the weighted average parameters for the base model (simple persistence) and the MPS model for the key DRO–FR comparisons. Table 9 lists the individual parameters. Note that as the contingencies went from DRO to FR, all parameters in the top panel increased in value. The increase in alpha indicates that the distribution of number of responses moved from one that looked like a gamma distribution ($\alpha = 1.46$) to one that looked like a skewed normal distribution ($\alpha = 2.23$; see Figure 3). The doubling of c reflects a large increase in the mean number of responses emitted in the response state in the FR condition. The increase in gamma indicates that pecking tended to occur more often in alternative strings of responding or quiescence, making it advantageous for the simple moving average of the base model to place more weight on the recent history of responding in the FR conditions.

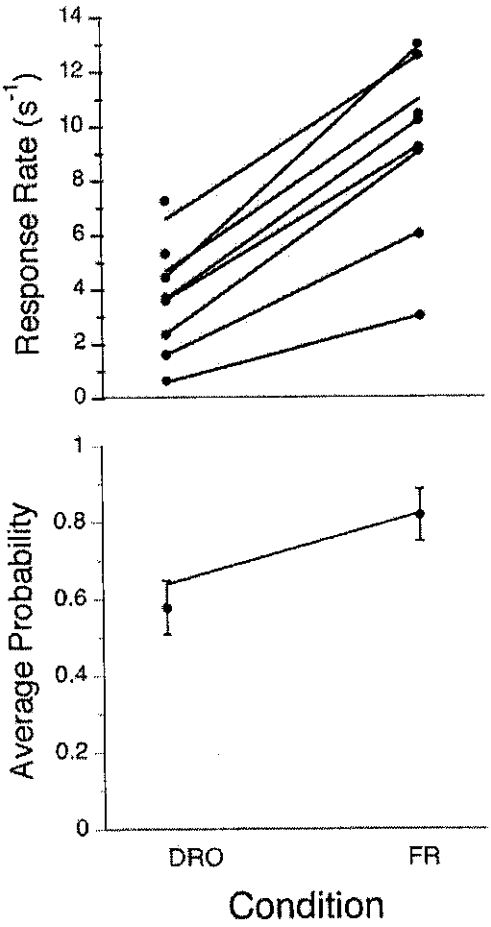

Figure 13. Data from Experiment 3. Top: average response rate (dots) for each subject. Bottom: Average probability of making at least one response on a trial averaged over pigeons; bars give standard errors. Lines in both panels are from the Momentum/Pavlovian/Skinnerian model. FR = fixed ratio; DRO = differential reinforcement of other behavior.

Table 8
Order (and Number of Sessions) in Each Condition of Experiment 3

Condition	P43	P86	P87	P89	P90	P105	P106	P107
Training	1 (18)	1 (18)	1 (18)	1 (18)	1 (18)	1 (18)	1 (18)	1 (18)
FR 3	2 (17)	3 (7)	3 (7)	2 (17)	2 (17)	3 (17)	2 (17)	3 (7)
DRO 2 s	3 (7)	2 (17)	2 (17)	3 (7)	3 (7)	2 (7)	3 (7)	2 (17)

Note. P = pigeon; FR = fixed ratio; DRO = differential reinforcement of other behavior.

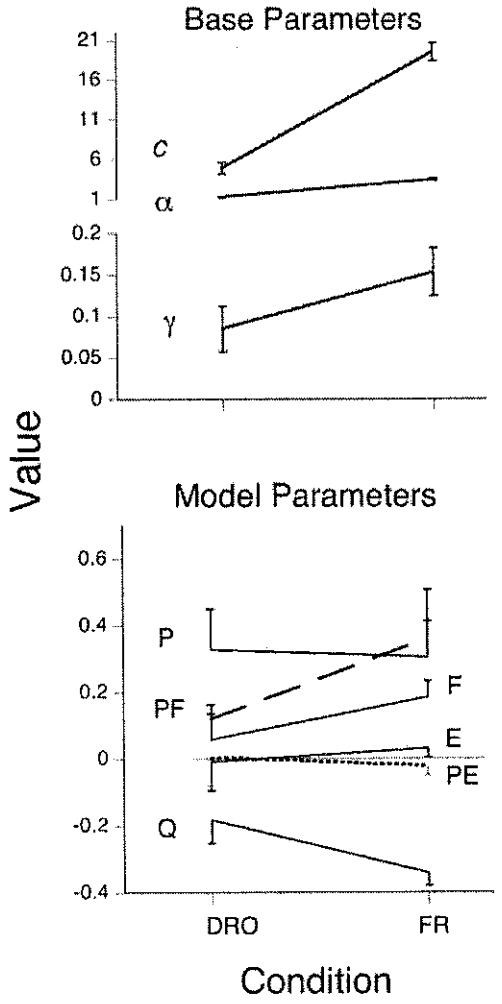

Figure 14. The average parameters of the base and Momentum/Pavlovian/Skinnerian models for the differential reinforcement of other behavior (DRO) and fixed-ratio (FR) contingencies of Experiment 3. The same Weibull parameters, c and α, were used for the response distributions of both models. The error bars delimit the standard errors of the mean.

The main purpose of this experiment was to test the sensitivity of the MPS model to changes in behavior brought about by the manipulation of contingencies of reinforcement, and in particular to monitor changes in the instrumental learning parameter, π_{PF}. Figure 14 shows that there was a large increase in π_{PF} under the FR contingencies and smaller changes in some of the other parameters (see Table 9). Trials without a reinforcer had, on average, no effect, because π_E was very close to zero for most animals in most conditions, as was π_{PE}. The momentum parameter for the base model (γ) was larger under the FR condition, suggesting greater movement into and out of response states, whereas those for the MPS model (π_P and π_Q) were in line with those found in Experiment 2. The decrease in the latter under FR suggests a kind of ratio strain: Absence from the key on one trial became a better predictor of absence on the next.

The smaller value of π_{PF} under DRO should not be taken as an indication that the pigeons were learning less; they were learning to do other things than key pecking. A smaller value of π_{PF} indicates that they were less likely to peck on an ensuing trial. If they received food on a trial with a peck, the peck was removed from the reinforcer by at least 2 s and was followed by non–key-peck behavior. This latter was successfully reinforced, yielding the smaller tendency to peck on the next trial than found under the FR contingency.

The impression from the first two experiments, that the power of instrumental contingencies was weak compared with Pavlovian contingencies, now stands corrected. Where there is no instrumental contingency, but only adventitious pairing of responding as in the first two experiments, control by that pairing can be weak or nil. This may be due to the many instances of pecking without presentation of food, causing the pigeon to place little weight on pecking as a predictor of food. In Experiment 3, FR contingencies trebled the Skinnerian parameter π_{PF} from .12 to .36. Under the FR contingencies, each contingent presentation of food moved the typical pigeon a third of the way to certain responding on the next trial, with the persistence and Pavlovian parameters together halving the remaining distance. The increase in response rates seen in the top panel of Figure 13 for FR thus arises from two factors: An increased probability of entering a response state in that condition because of the action of these parameters and a higher rate of responding once in that state, reflected by the increase in c. The first set of conditioning factors are substantial and consistent with the theoretical position of Donahoe, Palmer, and Burgos (1997a), yet they are inadequate to completely explain the large differences in rate. Differential proximity between responses and reinforcement in these two conditions is further affecting the behavior within the response state, much as it does in free operant schedules (e.g., Killeen, 1969). The parameter c reflects the operation of instrumental conditioning in the response state, moving more of the conditioned behavior onto the key.

The joint role of respondent and operant conditioning demonstrated here was presaged by Wasserman, Hunter, Gutowski, and Bader (1975) in their study of automaintained responding in chicks, with warmth as the US/S^R. Locurto et al. (1980) found similar interactions and suggested adopting "an 'interactivist' position wherein Pavlovian and instrumental relations are seen as independent variables which conjointly determine the outcome of any conditioning procedure" (p. 42). It was manifest in an experiment by Osborne and Killeen (1977), who superimposed CSs ranging from 7.5 s to 120 s on a variable-interval schedule that only reinforced responses spaced by 3 s (TAND[VT60, DRL3]). Even though the CS signaled noncontingent food, it enhanced median response rates from a baseline of 25 per minute to 170 per minute at the shortest CS, decreasing monotonically to 45 per minute at the longest. They successfully analyzed within the CS with an extreme value function in the same family as the Weibull used here. Such within-CS analysis begins to fill one of the silences of the R-W model (Hanson, 1977; Miller & Barnet, 1993).

General Discussion

Momentum

The analysis of momentum, or durability of responding, has a long history marked by two changes of paradigm. The first was the discovery of the partial reinforcement extinction effect by Hum-

Table 9
Indices of Merit, and Parameter Values of the Base and Momentum/Pavlovian/Skinnerian Models for the Data of Experiment 3

	Schedule							
Parameter	FR	DRO	FR	DRO	FR	DRO	FR	DRO
Bird no.	43		86		87		89	
CD	0.11	0.16	0.21	0.07	0.09	0.05	0.12	0.02
AIC	77	19	−5	84	−2	−2	63	19
BIC	59	4	−19	66	−14	−17	39	−1
γ	0.15	0.08	0.12	0.08	0.02	0.06	0.08	0.04
α	11.66	3.62	16.95	6.93	12.90	10.75	7.99	4.39
c	2.45	1.57	2.36	1.01	1.49	1.84	1.85	1.69
P	0.31	0.16	0.09	0.22	0.04	0.02	0.11	0.83
Q	−0.20	−0.10	−0.26	−0.12	−0.03	−0.03	−0.33	0.41
F	0.31	0.10	0.14	−0.13	0.01	0.00	0.18	−0.47
E	−0.01	0.00	0.07	0.02	0.00	0.00	0.09	−0.74
PF	0.00	0.42	0.00	0.00	0.00	0.07	0.00	0.00
PE	0.00	0.00	0.00	0.00	0.00	0.00	−0.03	0.13
Bird no.	90		105		106		107	
CD	0.53	0.31	0.13	0.06	0.20	0.07	0.18	0.42
AIC	435	176	10	17	2	84	71	9
BIC	406	151	0	5	−8	66	54	−2
γ	0.31	0.26	0.22	0.03	0.11	0.08	0.18	0.09
α	12.64	4.64	15.76	10.15	16.52	6.93	14.31	5.82
c	2.53	1.42	2.11	1.52	2.34	1.01	2.00	1.39
P	0.66	0.82	0.24	0.02	0.00	0.22	0.12	0.14
Q	−0.55	−0.46	−0.18	−0.03	−0.27	−0.12	−0.25	−0.10
F	0.18	0.16	0.07	0.13	0.01	−0.13	0.52	0.03
E	0.02	0.01	0.00	0.00	0.15	0.02	0.00	0.00
PF	1.00	0.57	0.00	0.00	0.00	0.00	0.14	0.00
PE	−0.05	−0.09	0.00	0.00	0.00	0.00	0.00	0.00

Note. γ = the rate constant for the comparison base model; c = the Weibull rate constant; α = the Weibull shape constant; the remaining letters indicate the rate constants brought into play on trials with (P) or without (Q) a response; with (F) or without (E) food; and the Skinnerian interaction terms PF and PE. FR = fixed ratio; DRO = differential reinforcement of other behavior.

phreys (1939)—the paradoxical result that probabilistic reinforcement generates more responses in extinction than does continuous reinforcement. It generated a tremendous and continuing amount of research (Mackintosh, 1974). The second was the renewed call of attention to momentum by Nevin and his students (Nevin & Grace, 2001; Nevin, Mandell, & Atak, 1983; Nevin, Tota, Torquato, & Shull, 1990) under the rubric *behavioral momentum.* As is the case for the partial reinforcement extinction effect, which it helps to explicate (Nevin, 1988), the study of behavioral momentum has applications well beyond the animal behavior laboratory (Nevin, 1996; Plaud & Gaither, 1996). It is most closely associated with the opposing forces of π_P and π_Q and, in extinction, with their simple difference π_{P-Q}.

This work has shown that behavioral momentum is most closely associated with Pavlovian forces, such as the relative densities of food in CS and background, and less so with instrumental contingencies and rates of responding. Consistent with Nevin and associates' results (Nevin & Grace, 2001; Nevin, Mandell, & Atak, 1983; Nevin, Tota, Torquato, & Shull, 1990), Figure 5 shows that when ITI was varied, persistence in both pecking π_P and quiescence π_Q, and their difference, π_{P-Q}, increased with the Pavlovian variable of ITI-to-trial (ITI/T) ratio; Figures 2 and 9 show that when trial duration was varied, persistence in both pecking and quiescence decreased with decreases in ITI/T, and Figure 14 shows that despite radically different responding under DRO and FR contingencies, π_{P-Q} was about the same in those experimental conditions, indicating that momentum would also be about the same, echoing Nevin and associates' conclusions. The influence of prior behavior on current behavior has been demonstrated in a different paradigm by de la Piedad, Field, and Rachlin (2006), who underscored the importance of the persistence they demonstrated for issues of rationality and self-control, a theme most beautifully introduced to our field by James (1890a, 1890b). The current paradigm and analysis provides a new set of operations for testing and developing behavioral momentum theory and other more general theories of momentum and choice (e.g., Killeen, 1992; Roe, Busemeyer, & Townsend, 2001).

Conditioning

"Today, most contemporary theories of acquired behavior are predicated on observations initially made to assess the Rescorla-Wagner model" (Miller et al., 1995, p. 381). The MPS model developed here is in that tradition; it is an "error-correction" model, like the R-W model and its linear-learning model forebears. Deviation from complete momentum or quiescence and deviation from complete conditioning or extinction both proceed as a function of distance from asymptote. This aperçu, however, may reflect more a limitation of imagination on our part than on the organisms'. Only a few of the infinite number of possible models of conditioning have been evaluated.

Does the MPS model capture learning or performance effects? It predicts response strength, s, the probability that the pigeon will be in a response state, with the rate of responding in that state given by the Weibull distribution. What the pigeon learns in this context is relative frequencies of food given keylight and given both light and peck. The scheduled probabilities of these is constant (at .1 or .05 or 0) in all these experiments, but the random sampling of trials by responses makes the observed frequencies a continually varying estimator of those probabilities. The strengths of the context, key, and peck state could be continuously varying, each in their own way, with our reduction to a net strength (s, probability of entering the response state) a synopsis of more nuanced three-way tugs of war among these factors. Models that keep separate accounts of these components of learning might easily trump MPS in rich data sets such as these, despite their extra parameters, or in others in which the forces are put into strong opposition. By treating instrumental responses as stimuli to be approached in the same manner as a lit key (Bindra, 1978), SOCR (Stout & Miller, 2007), attentional models (Frey & Sears, 1978; Mackintosh, 1975), RET (Gallistel & Gibbon, 2000) and its refinement by Kakade and Dayan (2002), SOP (Brandon, Vogel, & Wagner, 2003), WILL (Dayan, Niv, Seymour, & Daw, 2006), and the artificial neural net genre (e.g., Burgos, 1997; Donahoe, Palmer, & Dorsel, 1994) may be evaluated against these data. This paradigm also provides an ideal environment to analyze the potential progression of "learned irrelevance" (Baker, Murphy, & Mehta, 2003).

A limitation of the current analysis is its focus on one well-prepared response, appetitive key pecking in the pigeon. The relative importance of operant and respondent control will vary substantially depending on the response system studied (Donahoe, Palmer, & Burgos, 1997b; Jenkins, 1977; Timberlake, 1999). Another is that we have fit only a limited number of models to the data—albeit more than mentioned here, including versions of SOCR (Stout & Miller, 2007), attentional models (Frey & Sears, 1978; Mackintosh, 1975), and RET (Gallistel & Gibbon, 2000) and its improvement by Kakade and Dayan (2002). The models we present in this article were the best of the lot. But other models might have done better, in particular ones with attention (Mackintosh, 1975) or memory (Bouton, 1993; Wagner, 1981) as latent states. All theories, successful and otherwise, are at best sufficient accounts of the phenomena that they cover (Mazur, 2006), as Poincaré (1905/1952) noted long ago.

The dependent variable was a standard operant response. Holland (1979) has shown that omission contingencies have differential effects on various components of Pavlovian conditioned responding in rats. It may be that the difference is merely greater associability of different responses (Killeen, Hanson, & Osborne, 1978; Seligman, 1970), manifested as differences in the pi parameters. Indeed, it maybe that in some configurations, the Pavlovian parameter goes negative, with delivery of food increasing goal approach (Timberlake, 1994) on the next trial, competing with the measured operant. Such possibilities have yet to be demonstrated.

Another limitation is that MPS does not address the key contribution of the R-W model and its successors, cue competition and the partitioning of attention in the conditioning process. It did not need to here because changes in the predictive value of key or peck change the probability of entering the response state in the same direction: The conditionals coordinate rather than compete. Independent bookkeeping for cue, context, and peck conditioning were assayed in preliminary evaluation of the models presented here, but the experimental paradigm did not generate enough leverage where those models might contribute their strong suits. The partitioning out of momentum that the MPS model permits may, for the right experimental paradigm, provide a much clearer signal for how the Pavlovian and Skinnerian factors—or Pavlovian and Pavlovian factors—compete, or where one differentially sets the occasion for the other (Colwill & Rescorla, 1986; Nadel & Willner, 1980; Schmajuk, Lamoureux, & Holland, 1998). Such qualitative tests work hand in hand with quantitative ones (Roberts & Pashler, 2000) to converge on models that are powerful, parsimonious, and in register with the complexity of evolved processes such as learning. Although we strive for a unified theory of behavior, the best way to achieve it may be by perfecting modules that can account for their domain, while exchanging information with modules of other domains (Guilhardi, Yi, & Church, 2007).

In their penetrating assessment of the R-W model, Miller et al. (1995) noted 18 theoretical successes and about as many failures. They went on to observe that newer models are "highly complex or have their own list of failures at least as extensive as the R-W model" (p. 381) but that each of the new models has its strengths in fixing some of the failures of the R-W model. It is our hope that by embedding contemporary models in the present framework, which permits variance due to momentum to be partitioned out and permits ad libitum degrees of freedom in the data to counterpoise those required for modern complex models of conditioning (see, e.g., Hall, 2002, for an overview), that the models themselves may compete on a higher playing field. Dynamic analysis may also permit the refinement of experiments and permit reduction of the number of subjects required to answer behavioral or pharmacological questions (Corrado, Sugrue, Seung, & Newsome, 2005; Smith et al., 2004). The MPS model is but a second step through the door opened by Bush and Mosteller (1951) so many years ago.

References

Ashby, F. G., & O'Brien, J. B. (2008). The p_{rep} statistic as a measure of confidence in model fitting. *Psychonomic Bulletin & Review, 15,* 16–27.

Atkinson, R. C., & Estes, W. K. (1962). *Stimulus sampling theory.* Palo Alto, CA: Institute for Mathematical Studies in the Social Science, Applied Mathematics and Statistics Laboratories, Stanford University.

Baker, A. G., Murphy, R. A., & Mehta, R. (2003). Learned irrelevance and retrospective correlation learning. *Quarterly Journal of Experimental Psychology: Journal of Comparative and Physiological Psychology, 56*(B), 90–101.

Balsam, P. D., & Schwartz, A. L. (2004). Rapid contextual conditioning in autoshaping. *Journal of Experimental Psychology: Animal Behavior Processes, 7,* 382–393.

Benedict, J. O., & Ayres, J. J. (1972). Factors affecting conditioning in the truly random control procedure in the rat. *Journal of Comparative and Physiological Psychology, 78,* 323–330.

Bindra, D. (1978). How adaptive behavior is produced: A perceptual-motivational alternative to response-reinforcement. *Behavioral Brain Sciences, 1,* 41–91.

Bitterman, M. E. (2006). Classical conditioning since Pavlov. *Review of General Psychology, 10,* 365–376.

Bouton, M. E. (1993). Context, time, and memory retrieval in the interference paradigms of Pavlovian learning. *Psychological Bulletin, 114,* 80–99.

Bower, G. H. (1994). A turning point in mathematical learning theory. *Psychological Review, 101,* 290–300.

Brandon, S. E., Vogel, E. H., & Wagner, A. R. (2003). Stimulus representation in SOP: I. Theoretical rationalization and some implications. *Behavioral Processes, 62,* 5–25.

Burgos, J. E. (1997). Evolving artificial networks in Pavlovian environments. In J. W. Donahoe & V. Packard-Dorsel (Eds.), *Neural-network models of cognition: Biobehavioral foundations* (pp. 58–79). New York: Elsevier.

Burke, C. J., & Estes, W. K. (1956). A component model for stimulus variables in discrimination learning. *Psychometrika, 22,* 133–145.

Burnham, K. P., & Anderson, D. R. (2002). *Model selection and multimodel inference: A practical information-theoretic approach* (2nd ed.). New York: Springer-Verlag.

Bush, R. R., & Mosteller, F. (1951). A mathematical model for simple learning. *Psychological Review, 58,* 313–323.

Colwill, R. M. (2001). The effect of noncontingent outcomes on extinction of the response-outcome association. *Animal Learning & Behavior, 29,* 153–164.

Colwill, R. M., & Rescorla, R. A. (1986). Associative structures in instrumental learning. In G. H. Bower (Ed.), *The psychology of learning and motivation* (Vol. 20, pp. 55–103). New York: Academic Press.

Colwill, R. M., & Rescorla, R. A. (1988). The role of response-reinforcer associations increases throughout extended instrumental training. *Animal Learning & Behavior, 16,* 105–111.

Colwill, R. M., & Triola, S. M. (2002). Instrumental responding remains under the control of the consequent outcome after extended training. *Behavioural Processes, 57,* 51–64.

Corrado, G. S., Sugrue, L. P., Seung, H. S., & Newsome, W. T. (2005). Linear-nonlinear-Poisson models of primate choice dynamics. *Journal of the Experimental Analysis of Behavior, 84,* 581–617.

Couvillon, P. A., & Bitterman, M. E. (1985). Analysis of choice in honeybees. *Animal Learning & Behavior, 13,* 246–252.

Cox, D. R., & Hinkley, D. V. (1974). *Theoretical statistics.* London: Chapman & Hall.

Danks, D. (2003). Equilibria of the Rescorla–Wagner model. *Journal of Mathematical Psychology, 47,* 109–121.

Davison, M., & Baum, W. M. (2000). Choice in a variable environment: Every reinforcer counts. *Journal of the Experimental Analysis of Behavior, 74,* 1–24.

Davison, M., & Baum, W. M. (2006). Do conditional reinforcers count? *Journal of the Experimental Analysis of Behavior, 86,* 269–283.

Davol, G., Steinhauer, G., & Lee, A. (2002). The role of preliminary magazine training in acquisition of the autoshaped key peck. *Journal of the Experimental Analysis of Behavior, 1977,* 99–106.

Dayan, P., Niv, Y., Seymour, B., & Daw, N. D. (2006). The misbehavior of value and the discipline of the will. *Neural Networks, 19,* 1153–1160.

de la Piedad, X., Field, D., & Rachlin, H. (2006). The influence of prior choices on current choice. *Journal of the Experimental Analysis of Behavior, 85,* 3–21.

Donahoe, J. W., Palmer, D. C., & Burgos, J. E. (1997a). The S-R issue: Its status in behavior analysis and in Donahoe and Palmer's learning and complex behavior. *Journal of the Experimental Analysis of Behavior, 67,* 193–211.

Donahoe, J. W., Palmer, D. C., & Burgos, J. E. (1997b). The unit of selection: What do reinforcers reinforce? *Journal of the Experimental Analysis of Behavior, 67,* 259–273.

Donahoe, J. W., Palmer, D. C., & Dorsel, V. P. (1994). *Learning and complex behavior.* Boston: Allyn & Bacon.

Downing, K., & Neuringer, A. (2003). Autoshaping as a function of prior food presentations. *Journal of the Experimental Analysis of Behavior, 26,* 463–469.

Estes, W. K. (1950). Toward a statistical theory of learning. *Psychological Review, 57,* 94–107.

Estes, W. K. (1956). The problem of inference from curves based on group data. *Psychological Bulletin, 53,* 134–140.

Estes, W. K. (1962). Learning theory. *Annual Review of Psychology, 13,* 107–144.

Frey, P. W., & Sears, R. J. (1978). Model of conditioning incorporating the Rescorla-Wagner associative axiom, a dynamic attention process, and a catastrophe rule. *Psychological Review, 85,* 321–340.

Gallistel, C. R., & Gibbon, J. (2000). Time, rate, and conditioning. *Psychological Review, 107,* 289–344.

Gibbon, J., Baldock, M. D., Locurto, C. M., Gold, L., & Terrace, H. S. (1977). Trial and intertrial durations in autoshaping. *Journal of Experimental Psychology: Animal Behavior Processes, 3,* 264–284.

Gibbon, J., Farrell, L., Locurto, C. M., Duncan, H. J., & Terrace, H. S. (1980). Partial reinforcement in autoshaping with pigeons. *Animal Learning & Behavior, 8,* 45–59.

Grace, R. C. (2002). Acquisition of preference in concurrent chains: Comparing linear-operator and memory-representational models. *Journal of Experimental Psychology: Animal Behavior Processes, 28,* 257–276.

Guilhardi, P., Yi, L., & Church, R. M. (2007). A modular theory of learning and performance. *Psychonomic Bulletin & Review, 14,* 543–559.

Hall, G. (2002). Associative structures in Pavlovian and instrumental conditioning. In R. Gallistel (Ed.), *Steven's handbook of experimental psychology* (pp. 1–46). New York: Wiley.

Hanson, S. J. (1977). *The Rescorla-Wagner model and the temporal control of behavior.* Unpublished master's thesis, Arizona State University, Tempe.

Healy, A. F., Kosslyn, S. M., & Shiffrin, R. M. (Eds.). (1992). *From learning theory to connectionist theory: Essays in honor of William K. Estes.* Hillsdale, NJ: Erlbaum.

Hearst, E. (1975). The classical-instrumental distinction: Reflexes, voluntary behavior, and categories of associative learning. In W. K. Estes (Ed.), *Handbook of learning and cognitive processes* (Vol. 2, pp. 181–223). Mahwah, NJ: Erlbaum.

Hearst, E., & Jenkins, H. M. (1974). *Sign-tracking: The stimulus-reinforcer relation and directed action.* Austin, TX: Psychonomic Society.

Holland, P. C. (1979). Differential effects of omission contingencies on various components of Pavlovian appetitive conditioned responding in rats. *Journal of Experimental Psychology: Animal Behavior Processes, 5,* 178–193.

Humphreys, L. G. (1939). The effect of random alternation of reinforcement on the acquisition and extinction of conditioned eyelid reactions. *Journal of Experimental Psychology, 25,* 141–158.

James, W. (1890a). *Classics in the history of psychology: The principles of psychology.* Retrieved March 25, 2009, from http://psychclassics.yorku.ca/James/Principles

James, W. (1890b). *Principles of psychology.* New York: Holt.

Janssen, M., Farley, J., & Hearst, E. (1995). Temporal location of unsignaled food deliveries: Effects on conditioned withdrawal (inhibition) in pigeon signtracking. *Journal of Experimental Psychology: Animal Behavior Processes, 21,* 116–128.

Jenkins, H. M. (1977). Sensitivity of different response systems to stimulus–reinforcer and response–reinforcer relations. In H. Davis & H. Hurwitz (Eds.), *Operant-Pavlovian interactions* (pp. 47–66). Mahwah, NJ: Erlbaum.

Kakade, S., & Dayan, P. (2002). Acquisition and extinction in autoshaping. *Psychological Review, 109,* 533–544.

Killeen, P. R. (1969). Reinforcement frequency and contingency as factors in fixed-ratio behavior. *Journal of the Experimental Analysis of Behavior, 12,* 391–395.

Killeen, P. R. (1981). Averaging theory. In C. M. Bradshaw, E. Szabadi, & C. F. Lowe (Eds.), *Quantification of steady-state operant behaviour* (pp. 21–34). Amsterdam: Elsevier.

Killeen, P. R. (1985). Reflections on a cumulative record. *Behavior Analyst, 8,* 177–183.

Killeen, P. R. (1992). Mechanics of the animate. *Journal of the Experimental Analysis of Behavior, 57,* 429–463.

Killeen, P. R. (2001). Writing and overwriting short-term memory. *Psychonomic Bulletin & Review, 8,* 18–43.

Killeen, P. R. (2003). Complex dynamic processes in sign-tracking with an omission contingency (negative automaintenance). *Journal of Experimental Psychology: Animal Behavior Processes, 29,* 49–61.

Killeen, P. R., Hall, S. S., Reilly, M. P., & Kettle, L. C. (2002). Molecular analyses of the principal components of response strength. *Journal of the Experimental Analysis of Behavior, 78,* 127–160.

Killeen, P. R., Hanson, S. J., & Osborne, S. R. (1978). Arousal: Its genesis and manifestation as response rate. *Psychological Review, 85,* 571–581.

Levine, G., & Burke, C. J. (1972). *Mathematical model techniques for learning theories.* New York: Academic Press.

Locurto, C. M., Duncan, H., Terrace, H. S., & Gibbon, J. (1980). Autoshaping in the rat: Interposing delays between responses and food. *Animal Learning & Behavior, 8,* 37–44.

Locurto, C. M., Terrace, H. S., & Gibbon, J. (Eds.). (1981). *Autoshaping and conditioning theory.* New York: Academic Press.

Loftus, G. R., & Masson, M. E. J. (1994). Using confidence intervals in within-subject designs. *Psychonomic Bulletin & Review, 1,* 476–490.

Mackintosh, N. J. (1974). *The psychology of animal learning.* New York: Academic Press.

Mackintosh, N. J. (1975). A theory of attention: Variations in the associability of stimuli with reinforcement. *Psychological Review, 82,* 276–298.

Mazur, J. E. (2006). Mathematical models and the experimental analysis of behavior. *Journal of the Experimental Analysis of Behavior, 85,* 275.

Miller, R. R., & Barnet, R. C. (1993). The role of time in elementary associations. *Current Directions in Psychological Science, 2,* 106–111.

Miller, R. R., Barnet, R. C., & Grahame, N. J. (1995). Assessment of the Rescorla-Wagner model. *Psychological Bulletin, 117,* 363–386.

Myerson, J. (1974). Leverpecking elicited by signaled presentation of grain. *Bulletin of the Psychonomic Society, 4,* 499–500.

Myung, I. J. (2003). Tutorial on maximum likelihood estimation. *Journal of Mathematical Psychology, 47,* 90–100.

Myung, I. J., & Pitt, M. A. (1997). Applying Occam's razor in modeling cognition: A Bayesian approach. *Psychonomic Bulletin & Review, 4,* 79–95.

Nadel, L., & Willner, J. (1980). Context and conditioning: A place for space. *Physiological Psychology, 8,* 218–228.

Nevin, J. A. (1988). Behavioral momentum and the partial reinforcement effect. *Psychological Bulletin, 103,* 44–56.

Nevin, J. A. (1996). The momentum of compliance. *Journal of Applied Behavior Analysis, 29,* 535–547.

Nevin, J. A., & Grace, R. C. (2001). Behavioral momentum and the law of effect. *Behavioral and Brain Sciences, 23,* 73–90.

Nevin, J. A., Mandell, C., & Atak, J. R. (1983). The analysis of behavioral momentum. *Journal of the Experimental Analysis of Behavior, 39,* 49–59.

Nevin, J. A., Tota, M., Torquato, R. D., & Shull, R. L. (1990). Alternative reinforcement increases resistance to change: Pavlovian or operant contingencies? *Journal of the Experimental Analysis of Behavior, 53,* 359–379.

Osborne, S., & Killeen, P. R. (1977). Temporal properties of responding during stimuli that precede response-independent food. *Learning and Motivation, 8,* 533–550.

Perkins, C. C., Beavers, W. O., Hancock, R. A., Hemmendinger, D., & Ricci, J. A. (1975). Some variables affecting rate of key pecking during response-independent procedures (autoshaping). *Journal of the Experimental Analysis of Behavior, 24,* 59–72.

Plaud, J. J., & Gaither, G. A. (1996). Human behavioral momentum: Implications for applied behavior analysis and therapy. *Journal of Behavior Therapy and Experimental Psychiatry, 27,* 139–148.

Poincaré, H. (1952). *Science and hypothesis.* New York: Dover. (Original work published 1905)

Popper, K. R. (2002). *The logic of scientific discovery.* London: Routledge.

Reboreda, J. C., & Kacelnik, A. (1993). The role of autoshaping in cooperative two-player games between starlings. *Journal of the Experimental Analysis of Behavior, 60,* 67–83.

Rescorla, R. A. (1992). Response-independent outcome presentation can leave instrumental R-O associations intact. *Animal Learning & Behavior,* 104–111.

Rescorla, R. A. (1999). Within-subject partial reinforcement extinction effect in autoshaping. *Quarterly Journal of Experimental Psychology: Journal of Comparative and Physiological Psychology,* 52(B), 75–87.

Rescorla, R. A. (2000a). Associative changes in excitors and inhibitors differ when they are conditioned in compound. *Journal of Experimental Psychology: Animal Behavior Processes, 26,* 428–438.

Rescorla, R. A. (2000b). Extinction can be enhanced by a concurrent excitor. *Journal of Experimental Psychology: Animal Behavior Processes, 26,* 251–260.

Rescorla, R. A. (2001). Are associative changes in acquisition and extinction negatively accelerated? *Journal of Experimental Psychology: Animal Behavior Processes, 27,* 307–315.

Rescorla, R. A. (2002a). Comparison of the rates of associative change during acquisition and extinction. *Journal of Experimental Psychology: Animal Behavior Processes, 28,* 406–415.

Rescorla, R. A. (2002b). Savings tests: Separating differences in rate of learning from differences in initial levels. *Journal of Experimental Psychology: Animal Behavior Processes, 28,* 369–377.

Rescorla, R. A., & Wagner, A. R. (1972). A theory of Pavlovian conditioning: Variations in the effectiveness of reinforcement and nonreinforcement. In A. H. Black & W. F. Prokasy (Eds.), *Classical conditioning II: Current research and theory* (pp. 64–99). New York: Appleton-Century-Crofts.

Roberts, S., & Pashler, H. (2000). How persuasive is a good fit? A comment on theory testing. *Psychological Review, 107,* 358–367.

Roe, R. M., Busemeyer, J. R., & Townsend, J. T. (2001). Multialternative decision field theory: A dynamic connectionist model of decision making. *Psychological Review, 108,* 370–392.

Sanabria, F., Sitomer, M. T., & Killeen, P. R. (2006). Negative automaintenance omission training is effective. *Journal of the Experimental Analysis of Behavior, 86,* 1–10.

Schachtman, T. (Ed.). (2004). *Pavlovian conditioning: Basic associative processes* (Vol. 17, pp. 3–5). : International Society for Comparative Psychology.

Schmajuk, N. A., Lamoureux, J. A., & Holland, P. C. (1998). Occasion setting: A neural network approach. *Psychological Review, 105,* 3–32.

Schwarz, G. (1978). Estimating the dimension of a model. *Annals of Statistics, 6,* 461–464.

Seligman, M. E. P. (1970). On the generality of the laws of learning. *Psychological Review, 77,* 406–418.

Shull, R. L. (1991). Mathematical description of operant behavior: An introduction. In I. H. Iversen & K. A. Lattal (Eds.), *Experimental analysis of behavior* (Vol. 2, pp. 243–282). New York: Elsevier.

Shull, R. L., Gaynor, S. T., & Grimes, J. A. (2001). Response rate viewed as engagement bouts: Effects of relative reinforcement and schedule type. *Journal of the Experimental Analysis of Behavior, 75,* 247–274.

Shull, R. L., & Grimes, J. A. (2006). Resistance to extinction following variable-interval reinforcement: Reinforcer rate and amount. *Journal of the Experimental Analysis of Behavior, 85,* 23–39.

Siegel, S., & Allan, L. G. (1996). The widespread influence of the Rescorla-Wagner model. *Psychonomic Bulletin & Review, 3,* 314–321.

Skinner, B. F. (1938). *The behavior of organisms.* New York: Appleton-Century-Crofts.

Skinner, B. F. (1976). Farewell, my lovely. *Journal of the Experimental Analysis of Behavior, 25,* 218.

Smith, A. C., Frank, L. M., Wirth, S., Yanike, M., Hu, D., Kubota, Y., et

al. (2004). Dynamic analysis of learning in behavioral experiments. *Journal of Neuroscience, 24,* 447.

Sperling, S. E., Perkins, M. E., & Duncan, H. J. (1977). Stimulus generalization from feeder to response key in the acquisition of autoshaped pecking. *Journal of the Experimental Analysis of Behavior, 27,* 469–478.

Steinhauer, G. D. (1982). Acquisition and maintenance of autoshaped key pecking as a function of food stimulus and key stimulus similarity. *Journal of the Experimental Analysis of Behavior, 38,* 281–289.

Stout, S. C., & Miller, R. R. (2007). Sometimes-competing retrieval (SOCR): A formalization of the comparator hypothesis. *Psychological Review, 114,* 759–783.

Terrace, H. S., Gibbon, J., Farrell, L., & Baldock, M. D. (1975). Temporal factors influencing the acquisition and maintenance of an autoshaped keypeck. *Animal Learning & Behavior, 3,* 53–62.

Timberlake, W. (1994). Behavior systems, associationism, and Pavlovian conditioning. *Psychonomic Bulletin & Review, 1,* 405–420.

Timberlake, W. (1999). Biological behaviorism. In W. O'Donahue & R. Kitchener (Eds.), *Handbook of behaviorism* (pp. 243–284). New York: Academic Press.

Timberlake, W. (2000). Motivational modes in behavior systems. In R. R. Mowrer & S. B. Klein (Eds.), *Handbook of contemporary learning theories* (pp. 155–209). Mahwah, NJ: Erlbaum.

Timberlake, W. (2003). Is the operant contingency enough for a science of purposive behavior? *Behavior and Philosophy, 32,* 197–229.

Tonneau, F. (2005). Windows. *Behavioural Processes, 69,* 237–247.

Tonneau, F., Ríos, A., & Cabrera, F. (2006). Measuring resistance to change at the within-session level. *Journal of the Experimental Analysis of Behavior, 86,* 109–121.

Vogel, E. H., Castro, M. E., & Saavedra, M. A. (2006). Quantitative models of Pavlovian conditioning. *Brain Research Bulletin, 63,* 173–202.

Wagner, A. R. (1981). SOP: A model of automatic memory processing in animal behavior. In N. E. Spear & R. R. Miller (Eds.), *Information processing in animals: Memory mechanisms* (pp. 5–47). Hillsdale, NJ: Erlbaum.

Wasserman, E. A., Hunter, N., Gutowski, K., & Bader, S. (1975). Autoshaping chicks with heat reinforcement: The role of stimulus-reinforcer and response-reinforcer relations. *Journal of Experimental Psychology: Animal Behavior Processes, 104,* 158–169.

Wasserman, E. A., & Miller, R. R. (1997). What's elementary about associative learning? *Annual Review of Psychology, 48,* 573–607.

Williams, D. R., & Williams, H. (1969). Auto-maintenance in the pigeon: Sustained pecking despite contingent non-reinforcement. *Journal of the Experimental Analysis of Behavior, 12,* 511–520.

Woodruff, G., Conner, N., Gamzu, E., & Williams, D. R. (1977). Associative interaction: Joint control of key pecking by stimulus-reinforcer and response-reinforcer relationships. *Journal of the Experimental Analysis of Behavior, 28,* 133.

Yamaguchi, M. (2006). Complete solution of the Rescorla-Wagner model for relative validity. *Behavioural Processes, 71,* 70–73.

Appendix

Mathematical Details

Framing the Model

There are eight explicit parameters in the Momentum/Pavlovian/Skinnerian (MPS) model: the response parameters α and c, the two momentum parameters, the two Pavlovian parameters, and the Skinnerian parameters. (The Skinnerian parameters are partially redundant with the persistence parameter, but no attempt was made to enforce further parsimony in these already overworked data.) There are also implicit parameters. These involve the structure of the model and how that interacts with the parameters and data (Myung & Pitt, 1997). The directions of conditioning (the nominal signs of the learning parameters specifying their asymptotes θ, here fixed at 0 or 1) are such considerations. Another is the starting value of s, s_0, which is estimated as the average probability of a response over the first dozen trials of each condition, with those trials then excluded from all indices of merit. Because the logarithmic transformation penalizes errors exponentially as they approach maximum (e.g., predicting a response probability close to zero and having a response occur), a floor (of probability of data given the model) of 0.00001 was placed under both the candidate and the default models; it was rare for them to step on that floor except during the iterative process of parameter estimation. All analyses were conducted in Excel using the Solver add-in.

Looking Ahead

In general, the linear learning model was unquestionably better than the base model of momentum—the Akaike information criterion index of merit advantage for the learning model was typically close to 100 units. What does this mean in terms of ability to predict behavior? Most readers unfamiliar with the Akaike information criterion and log-likelihood analysis will appreciate some other indices of merit, such as variance accounted for by the model. However, we are predicting response rates on a trial-by-trial basis, not the typical averages over the last 10 sessions, each consisting of scores of observations. There is no opportunity to average out noise in the present dynamical analysis. In light of this, predictions were not so bad: The MPS model accounted for more than 10% of the variance in response rates on the next trial in Experiment 1, even though the analysis did not optimize goodness of fit for this variable. In the $p = .05$ condition, accuracy increased to 16%.

Whereas these might not seem impressive figures, nor might the advantage of the MPS model seem impressive in that metric, most considerations of variance accounted for—coefficients of determination—are calculated on average data, where noise has been minimized by averaging. None, to our knowledge, reflect accuracy on a moment-to-moment, or at least trial-by-trial, basis. Because conditions are always changing as a function of the behavior of the pigeon in this closed-loop system, there is no obvious larger unit over which we could aggregate data to improve accuracy. But there is a less-than-obvious one, described next.

The conditioning predicated by the learning model has a longer provenance than over just the next trial; a measure of accuracy that is both more informative, and more consistent with traditional

reports of coefficients of determination, can be derived by asking how well the imputed strength, s_i, predicts behavior over the next few trials. Because the learning model posits geometric changes in performance as a function of contingencies, accuracy of prediction should also decrease geometrically with distance into the future. Accuracy decreases because the stochastic processes that might or might not carry the pigeon over a response threshold on a trial throw a multiplicative shadow into the future. Therefore, accuracy should decrease approximately as $(1 - \gamma)^n$, as vicissitudes of responding and reinforcement carry the conditioning process along an increasingly random walk. We may take advantage of this by averaging measured responding over the next score trials, giving the greatest weight to the next trial, less to the trial after that, and so on, and using events on the current trial to project those temporally discounted future response rates. This was accomplished by weighting the accuracy of prediction on the next trial by 20%; adding to that accuracy on the trial after that weighted by 16%; on the trial after that by $.2(.8)^2$, then by $.2(.8)^3$, and so forth. This "forward" exponentially weighted moving average places half the predictive weight on the three trials subsequent to the prediction, trailing off geometrically into the future. Accuracy at predicting this discounted future in the $p = .05$ condition doubled, to 28% of the variance in response rates accounted for by the MPS model. A similar doubling of the coefficient of determination was seen in spot checks of the other conditions.

Asymptotic Responding

In this article, the conditioning process is characterized from trial to trial by a difference equation—the strength on the prior trial plus the probability of a response time by π_P and a nonresponse by π_Q; that is then adjusted by the probability of food times π_F and no food times π_E and finally by the probability of both a response and food times π_{PF} or of a response and no food times π_{PE}. This permits continuous idealizations of acquisition and extinction.

Representation of a stochastic process by its probabilities gives a domesticated version of an intrinsically wild process. For instance, performance is vulnerable to a "gambler's ruin"—a series of nonreinforced trials that leads to extinction. The probabilistic solutions do not take this sudden death into account and do not allow for the recuperative strength provided by spontaneous recovery at the start of new sessions. Nonetheless, they provide some insights into the process. We begin by analysis of the momentum factor and then blend it with the conditioning factors. Here the direction toward ceiling or floor is assumed, in the conventional manner. Conditioning enters after momentum, rather than before, as in the analysis programs. The order of entry makes some difference in accuracy of fit.

Momentum

Letting $p(P_i)$ represent the probability of responding on the ith trial, the momentum of responding is carried forward from the last trial as

$$s_i' = s_i + p(P_i)\pi_P(1 - s_i) + [1 - p(P_i)]\pi_Q(0 - s_i),$$

that is, as the strength coming out of the prior trial, s_i, plus probability of a response, $p(P_i)$, times π_P (the momentum-of-pecking rate parameter), times the distance to the ceiling strength $(1 - s_i)$, plus the probability of not pecking times the momentum-in-quiescence parameter π_Q times the distance to the floor of strength. The probability of pecking is approximately equal to the strength, s_i, so substitute for $p(P_i)$ and simplify to

$$s_i' = s_i[1 + (1 - s_i)\pi_P - \pi_Q]. \tag{A1}$$

The difference in momentum parameters is gated by the distance of strength to its ceiling, to increment response probability. This is slightly off because there is a finite probability of being in the response state and not pecking, but that is negligible. The variable s_i' is the momentum of responding that is carried forward to the next trial.

Pavlovian Conditioning

Food occurs with probability p, so

$$s_i'' = s_i' + p\pi_F(1 - s_i') + (1 - p)\pi_E(0 - s_i');$$

that is, as the status quo ante (s_i'), plus the probability of food times the Pavlovian parameter π_F times the distance to ceiling, plus the probability of no food times the Pavlovian extinction parameter π_E times the distance to floor. Collecting terms yields

$$s_i'' = s_i' + [1 + p(\pi_E - \pi_F) - \pi_E] + p\pi_F. \tag{A2}$$

When the probability of food is $p = 1$, the influence of π_E drops out, leaving strength on a march toward 1; conversely, where $p = 0$, strength decreases geometrically from one trial to the next by the factor $(1 - \pi_E)$.

To predict responding on the next trial, the intervening variable s_i' is removed by substituting from Equation A2 into Equation A1. Because the parameters π_P and π_Q always enter as a difference, some parsimony is achieved by writing $\pi_P - \pi_Q$ as π_{P-Q}. Then Equations A1 and A2 give

$$s_i'' = s_i'[1 + \pi_{P-Q}(1 - s_i)][1 + p(\pi_E - \pi_F) - \pi_E] + p\pi_F. \tag{A3}$$

Skinnerian Conditioning

Remembering that food occurs with probability p and a peck with probability s'', the increment to strength conferred by operant conditioning is

$$s_{i+1} = s_i'' + s_i''[p\pi_{PF}(1 - s'') + (1 - p)\pi_{PE}(1 - s'')(0 - s'')],$$

strength after Pavlovian updating, plus the probability of a response times the large parenthetical. Inside the parenthetical is the probability of food times its rate parameter times the distance to the ceiling, plus the probability of no food times its rate parameter times the probability of no peck times its distance to the floor. This may be simplified to

(*Appendix continues*)

$$s_{i+1} = s_i''\{1 + (1 - s_i'')[p\pi_{PF} - (1 - p)s_i''\pi_{PE}]\}. \quad \text{(A4)}$$

Inserting Equation A3 into this gives the final equation of prediction, too unenlightening to be written out here—even though it is less complicated than the full solution to the R-W model (Yamaguchi, 2006). It is simpler to evaluate Equation A3 and then insert it into Equation A4. A spreadsheet for analyzing data with the present theory on a trial-by-trial basis is available from Peter R. Killeen, Federico Sanabria, and Igor Dolgov.

Special Cases

Acquisition

In the case of acquisition, where few or no responses have yet occurred, Equation A3 provides a good equation of prediction. Because the probability of food p is typically 1.0, it can be further simplified to the acquisition function

$$s_{i+1} = s_i[1 + \pi_{P-Q}(1 - s_i)](1 - \pi_F) + \pi_F. \quad \text{(A5)}$$

Equation A5 can range from a classic exponential-integral learning curve (when π_{P-Q} is of small magnitude) through approximately linear to an S-shaped ogive, depending on the two parameters, the net rate of persistence (π_{P-Q}) and the rate of acquisition (π_F).

Extinction

In extinction, $p = 0$, and Equation A3 simplifies to

$$s_{i+1} = s_i(1 - \pi_E)[1 + \pi_{P-Q}(1 - s_i)], \quad \text{(A6)}$$

which appears as Equation 8 in the text. Equation A6 assumes that π_{PE} is smaller and that extinction decrements can be handled by π_E; this has been the case for all of the data analyzed here, and setting π_E to zero is a parsimonious way to simplify the equation.

Received April 15, 2008
Revision received February 6, 2009
Accepted February 6, 2009 ■