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# Mechanisms of spaced-trial runway extinction in pigeons <sup>☆</sup>

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## Abstract

Three experiments with pigeons examined the mechanisms underlying the partial reinforcement extinction effect (PREE), or greater persistence in extinction after training with partial, rather than continuous, reinforcement. All experiments involved runway performance, food reinforcement, and one trial per day. Experiment 1 provided evidence of the PREE together with evidence that large reward increased persistence relative to small reward. Experiment 2 demonstrated that the random mixture of different reward magnitudes increased persistence. Experiment 3 provided evidence that the PREE was not eliminated by chlordiazepoxide (benzodiazepine anxiolytic) but was eliminated by nicotine (cholinergic stimulant) and haloperidol (dopaminergic antagonist). The results are discussed in relation to comparative studies of learning in situations involving surprising reward downshifts.

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In a changing environment, behaviors that commonly yield a satisfying outcome may occasionally fail to do so. When unexpected omissions occur, an organism may behave in a number of different ways. It may immediately cease its current behavior and try an alternative that has worked in the past, try a novel behavior, continue the

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current behavior in the same environment, or move to a new locale and try the behavior there. The situation that has been studied most extensively is one in which an organism continues to perform a behavior in a particular environment in spite of reward uncertainty. A variety of phenomena, collectively known as the paradoxical learning effects, illustrate the effect of reward uncertainty and reward size earned during acquisition on the degree of behavioral persistence exhibited during extinction (see Amsel, 1992). These phenomena are interesting in part because they violate a common sense view of learning according to which more frequent reinforcement and larger reinforcers should lead to a type of behavior that is stronger, more difficult to modify, and more likely to occur in the face of changing environmental conditions. Yet, reward uncertainty tends to increase, rather than decrease, persistence. The research reported here is particularly concerned with two such extinction phenomena: the partial reinforcement extinction effect (PREE), or greater persistence after partial, rather than continuous, reinforcement training, and the magnitude of reinforcement extinction effect (MREE), or greater persistence after training with small, rather than large, reinforcers.

In addition to challenging common views of learning, the PREE and MREE have played a key role in the development of learning theory. The classic theories of Thorndike, Hull, Guthrie, and others could not deal with these phenomena and, indeed, some underwent changes as new evidence on the paradoxical effects started to accumulate (e.g., Hull, 1952). More recent general theories built on the basis of the classic assumption that reinforcement increases associative strength whereas nonreinforcement weakens it (i.e., strengthening–weakening models) have similar difficulties in dealing with paradoxical effects despite being relatively successful in other areas (e.g., Pearce & Hall, 1980; Rescorla & Wagner, 1972). There is, of course, another set of theories that have been developed precisely to account for the paradoxical effects (see Mackintosh, 1974), of which we single out Capaldi's sequential theory (Capaldi, 1994) and Amsel's frustration theory (Amsel, 1992). Initially developed during the 1960s, these two theories have generated a substantial amount of research and continue to influence research on learning (e.g., Bouton & Sunsay, 2001; Daly & Daly, 1982; Pearce, Redhead, & Aydin, 1997; Rescorla, 1999). A feature common to most accounts of paradoxical learning is that they tend to predict a strict covariation of such phenomena. For example, conditions that generate the PREE should also lead to the MREE and, vice versa, conditions that promote a reversed PREE should also lead to a reversed MREE. Among such conditions is the species used in the experiment.

From a comparative perspective, which is central to the present experiments, the paradoxical effects fit the pattern of strict covariation: they are either both present or both absent. The relevant evidence is provided by spaced-practice experiments in which individual trials are separated by intertrial intervals in the order of hours, during which the animal is located in a place other than the training context. Such a procedure permits a relatively clean assessment of the associative value of the controlling stimuli with little or no influence from carry-over stimuli or memories from the outcomes of previous trials (Weinstock, 1954). Thus, the effects have been discovered and extensively studied in several mammalian species, but they have

failed to appear in nonmammalian vertebrates (see Papini, 2002a). For example, toads (*Bufo arenarum*) receiving partial reinforcement training extinguish faster than toads receiving continuous reinforcement training, that is, a reversed PREE (Muzio, Segura, & Papini, 1992, 1993). Similarly, toads exhibit a reversed MREE and demonstrate a gradual change in performance, rather than an abrupt one, when shifted from a large to a small reward magnitude (Papini, Muzio, & Segura, 1995). Such a pattern of results can be easily accommodated by a strengthening–weakening model according to which partial or small-magnitude reinforcements result in relatively weak control of behavior (relative to continuous or large-magnitude reinforcements) and, consequently, faster extinction.

Evidence from spaced-training experiments with birds suggests that the spaced-trial paradoxical effects can be dissociated. In a series of experiments involving a single trial per day in a Skinner box situation, pigeons demonstrated a reversed MREE under training conditions that also yielded a conventional PREE in experiments involving response–outcome uncertainty (Papini, 1997; Papini & Thomas, 1997; Papini, Thomas, & Mc Vicar, in press). Moreover, the PREE was also reported in a spaced-trial experiment involving runway performance (Roberts, Bullock, & Bitterman, 1963). This sort of dissociation of paradoxical effects had not been reported before in adult animals of any species and merits further attention.

The present experiments were designed with three goals in mind. The first goal was to provide evidence of paradoxical dissociation in the extinction performance of pigeons within the same experiment. Previous evidence (see above) comes from different experiments; while suggestive, such evidence is inconclusive because many factors potentially varying across experiments may explain the presence of contrasting experimental outcomes. The second goal was to test pigeons under conditions more analogous to those used with rats, namely, the runway situation. Evidence of a paradoxical dissociation in pigeons comes entirely from experiments involving key-pecking in a Skinner box situation, and it is plausible that different response systems would respond in different ways under extinction conditions (e.g., Mellgren & Olson, 1983). The third goal was to provide some initial evidence of the physiological mechanisms underlying the PREE in pigeons. Physiological manipulations are here used as a tool to discriminate between two possible evolutionary hypotheses of the PREE. In a comparison between the PREE of rats and pigeons, behavioral similarity may result from the operation of the same mechanisms (i.e., homology) or from the operation of different mechanisms that produce similar learning phenomena (i.e., homoplasy; Papini, 2002a). No single experiment can probably answer this question, so the present results are offered as preliminary observations on the neurochemical basis of the PREE in pigeons.

## Experiment 1

Roberts et al. (1963) and Papini et al. (in press) reported slower extinction following partial reinforcement than following continuous reinforcement in runway and Skinner box situations, respectively. Furthermore, pigeons trained on an instrumen-

tal key-pecking task demonstrated slower extinction following acquisition with a large reward than with a small reward (Papini, 1997; Papini & Thomas, 1997). Taken together, these results suggested dissociation between the effects of reward uncertainty and reward magnitude in pigeons, that is, a conventional PREE combined with a reversed MREE. Two procedural problems complicate the interpretation of these findings. First, key-pecking and running responses may lead to different extinction patterns under otherwise analogous conditions of training (Mellgren & Olson, 1983). Second, the pigeons used in the key-pecking experiments cited above had prior experience in similar situations, whereas Roberts et al. (1963) used naïve birds. Perhaps the key-pecking results were artifacts of training history. Experiment 1 addressed these two concerns by testing naïve pigeons in a runway. Additionally, the design allowed for an assessment of extinction as a function of reward uncertainty and reward magnitude within the same experiment.

Experiment 1 included 4 groups: PR5, CR5/t, CR5/r, and CR1. The capital letters indicate the nature of the reinforcement schedule (continuous or partial reinforcement) and the numbers indicate the amount of grain (1 or 5 g) that was provided on reinforced trials. The lower case letters indicate whether PR5 and CR5 groups were matched in terms of the number of rewards (r) or trials (t) during acquisition.

### *Method*

*Subjects.* The subjects were 20 naïve, sexually mature pigeons of undetermined sex. Pigeons were purchased from Ruthardt Pet and Feed (Fort Worth, Texas) and were housed in the university vivarium in individual cages. Water was available throughout the experiment, but food, in the form of mixed-grain, was restricted so that pigeons were maintained at 80–85% of their ad libitum weight. The colony room was continuously lighted during the course of this experiment.

*Apparatus.* Subjects were conditioned in a wooden runway similar to that used by Roberts et al. (1963). The runway's internal dimensions were 247.0 cm long, 15.5 cm wide, and 25.0 cm high. It was divided into three compartments: a black start box (length: 43.5 cm), a gray alley (length: 160.0 cm), and a white goal box (length: 43.5 cm) separated by opaque doors that opened and closed horizontally. Hinged wooden lids covered each of the three compartments and allowed for easy retrieval of a pigeon at any location within the runway. At the end wall of the goal box, a 5-cm diameter hole opened into an adjacent illuminated chamber where a food cup held mixed-grain on reinforced trials. On nonreinforced trials, the food cup was left empty. Three photocells located 15 cm past the start door and in the alley, 15 cm before the goal door and also in the alley, and 10 cm from the end wall and in the goal box, recorded intercell latencies. These latencies were the primary dependent variable in the experiment. A computer in a separate room controlled the opening and closing of the sliding doors and recorded the latencies in 0.01-s units.

*Procedure.* Pigeons were pretrained to find food in the runway. Pretraining trials were administered at a rate of one per day during the morning. Initially, 5 g of grain were scattered about the floor in the goal box area and in the food cup, and each pigeon was placed directly into the illuminated goal box for 5 min. At the end of

the 5 min, the pigeon was removed and any remaining grain was weighed. Once it became apparent that a pigeon was readily eating in the goal (all grain eaten on 2 consecutive trials, one trial per day), a minimum of 2 trials of reinforced running was administered. On these trials, the bird was placed in the start box and given up to 5 min to traverse the runway. Once the third photocell was activated, the goal box door was automatically closed and the bird was given 3 additional minutes to eat any grain on the floor and in the food cup (5 g of mixed grain). If a pigeon failed to complete the run within 5 min, it was gently and slowly guided by the experimenter to the goal and was then allowed 3 min to eat. Acquisition training began after 2 consecutive trials without guidance. Quadruplets of pigeons matched on the number of pretraining trials were randomly assigned to four groups ( $n = 5$ ).

Acquisition trials consisted of placing a bird in the dark start box with the start door closed. Following a variable 30 s (15–45 s) initial interval, the door was opened allowing the bird to run to the illuminated goal box. Once the third photobeam was crossed, the goal box door was closed and the bird was given 3 min to eat the food (1 g or 5 g of mixed grain placed in the food cup, depending on the group). At the end of 3 min, the experimenter returned the bird to its home cage where it remained until the next trial. The order in which birds were run in any given day was randomized, thus yielding an intertrial interval close to 24 h. On nonreinforced trials, a pigeon was left in the goal for 3 min and was then returned to its home cage until the next trial. Pigeons in Group PR5 received 48 daily trials, half of them reinforced with access to 5 g of grain and the rest ending in nonreinforcement. The sequence of reinforced and nonreinforced trials was arranged according to Gellermann (1933) orders. Each of the CR groups was reinforced in the goal box on every acquisition trial. Groups CR5/t and CR1 received 48 daily trials each ending with access to 5 or 1 g of grain, respectively. Group CR5/r also received 5 g of grain in every acquisition trial, but was only trained on days when Group PR5 received reinforcement; this group thus received 24 acquisition sessions with an average intersession interval of 2 days. Therefore, in comparison with Group PR5, Group CR5/t (matched for trials) received the same number of trials, but twice as much reward, whereas Group CR5/r (matched for reward) received the same total amount of reward, but half as many trials. Acquisition was followed by 24 daily trials of extinction. Extinction trials were like the nonreinforced acquisition trials of Group PR.

This design allows for several key comparisons while simultaneously minimizing the number of groups. A comparison between Groups PR5 and CR5/t assesses the presence of the PREE in a conventional design in which groups are matched in terms of trials. A comparison between Groups PR5 and CR5/r also evaluates the presence of the PREE but, in this case, when groups are matched in terms of the number and temporal distribution of reinforcements during acquisition. A third comparison between Groups CR5/t and CR1 permits an evaluation of the MREE and, together with the first one described previously, allows for an assessment of the potential dissociability of PREE and MREE in pigeons. Finally, because Groups CR5/t and CR5/r differ in the number of acquisition trials (48 and 24, respectively), their extinction performance can be evaluated in terms of another paradoxical phenomenon: the overtraining extinction effect (OEE). The OEE has been studied under relatively

massed conditions of training, with intertrial intervals in the order of seconds to minutes, but there seems to be no information from experiments involving spaced-trial conditions (see Ishida & Papini, 1997, for an example of the massed-trial OEE and a review of available evidence).

*Data analysis.* Total latency, the dependent variable, was defined as the time from the opening of the start door to the breaking of the photobeam located in the goal box. Total latency scores were transformed according to the following procedure in all the experiments reported in this article. First, the score obtained by each pigeon in the last acquisition trial was subtracted from the scores obtained in each of the other trials for both acquisition and extinction (i.e., trial  $x$  – last acquisition trial). This transformation tends to reduce individual differences in runway performance and allows for an evaluation of behavioral change relative to the best performance of each subject in the experiment, namely, the one corresponding to the last acquisition trial. In each experiment, a Kruskal–Wallis analysis was computed for the performance obtained in the last acquisition session to determine whether there were group differences in these scores. Second, every sixth trial was selected for analysis (8 trials in acquisition and 5 in extinction), using non-parametric statistical procedures. Both of these features tend to reduce the possibility of Type I error in the analysis of repeated-measure data that might originate in the violation of such assumptions as homogeneity of variance and sphericity. Latency data are commonly analyzed with parametric statistics (e.g., repeated-measure analysis of variance) and the Group  $\times$  Trial interaction used as evidence that the rate of extinction differed between groups. Unfortunately, latency data are rarely normally distributed (especially late in acquisition and early in extinction) which forces a transformation of the scores (for a recent example see Papini et al., in press). The number of trials selected for analysis seemed to offer a reasonable compromise between information loss and statistical power. Finally, a Kruskal–Wallis analysis was computed for each trial and, when the group effect was significant, pairwise Wilcoxon rank sum tests for independent samples were calculated to determine the source of the effect. Given the use of difference scores, a significant group effect is analogous to a significant interaction in the analysis of variance and it suggests that the groups differ in the rate at which latencies changed relative to terminal acquisition performance. Although all the analyses involved selected trials, the figures illustrate the performance changes in every trial to allow for an assessment of the degree of representativeness of the points chosen for analysis. All analyses were conducted with the SPSS 8.0 software package. An  $\alpha$  value set at 0.05 and two-tailed distributions were used in all the tests reported in this article.

### *Results and discussion*

In order to ensure that the groups were equated prior to acquisition, pigeons were matched on the number of trials to criterion in the pretraining phase. A Kruskal–Wallis analysis confirmed that the groups did not differ before training. Also nonsignificant were the group differences in the last acquisition trial. The mean (range)

latencies were the following: 11.4 s (5.2–29.1), 13.1 s (2.9–44.5), 5.6 s (3.5–9.5), and 5.4 s (3.7–8.2) for Groups CR1, CR5/t, CR5/r, and PR5, respectively.

Acquisition occurred relatively fast in all groups. The first analyses were devoted to the effects of partial reinforcement by comparing Groups PR5, CR5/t, and CR5/r. Kruskal–Wallis tests on each of the target acquisition trials indicated a significant group effect on trial 24,  $H = 6.66$ . Wilcoxon rank sums tests showed that the latencies of Group CR5/r and PR5, which did not differ from one another, were significantly longer than those of Group CR5/t,  $Ts(5) < 19$ . This result is not surprising because the pigeons in Group CR5/t received the greatest amount of reinforcement in acquisition.

The extinction performance of these groups is plotted in the top panel of Fig. 1. The average rates of extinction are different across groups, with partial reinforcement generating a very slow change, if any, compared to both continuous reinforcement conditions. Thus the PREE was observed whether the partial condition is compared against a continuous condition matched in the number of trials, as usually

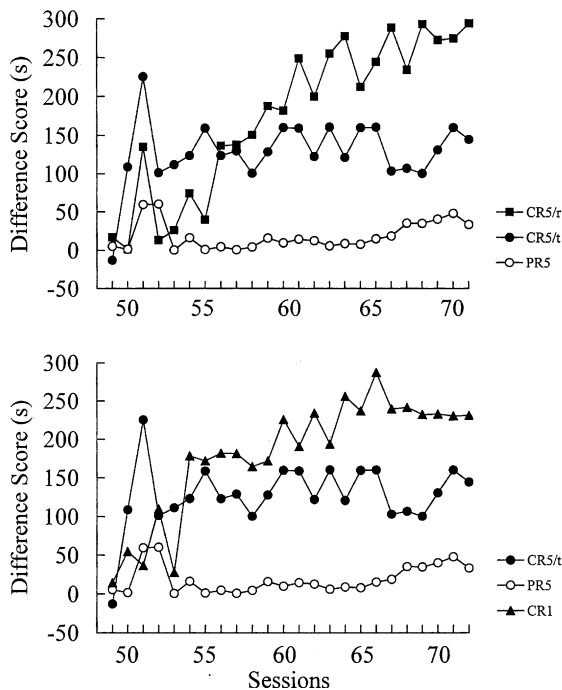


Fig. 1. Extinction performance of groups receiving different acquisition treatments. The top panel shows the PREE, whereas the bottom panel shows both a reversed MREE as well as the conventional PREE. Group PR received 50% partial reinforcement, with 5 g of food in each reinforced trial. Group CR5/t received continuous reinforcement training, 5 g of food per trial, and the same number of trials as Group PR5. Group CR5/r received continuous reinforcement, 5 g of food per trial, but these trials were administered on the days in which the pigeons in Group PR5 received a reinforced trial. Finally, Group CF1 received continuous reinforcement training and 1 g of food per trial.

done in analogous experiments, or against one matched in the number of reinforcements. Kruskal–Wallis tests indicated group differences on trials 66 and 72,  $H_s > 9.06$ . Pairwise comparisons showed that the latencies of Group PR5 were shorter than those of Group CR5/t on trial 66,  $T(5) = 17$ , and shorter than those of Group CR5/r on trials 60, 66, and 72,  $T_s(5) < 19.00$ . Additionally, a comparison of Groups CR5/t and CR5/r in Fig. 1, top panel, indicates a complex extinction pattern as the functions cross-over after about one third of extinction was completed. The terminal pattern reflects a reversed OEE with Group CR5/t performing significantly below Group CR5/r on trial 72,  $T(5) = 15$ .

The second analysis involves the groups trained with different reinforcer magnitudes during acquisition. An examination of Groups CR5/t and CR1 revealed no significant differences in acquisition,  $T_s(5) > 23$ . The extinction performance of these groups is presented in the bottom panel of Fig. 1. Clearly, pigeons trained with the smaller reinforcer extinguished faster than pigeons trained with the larger amount of food, that is, they exhibited a reversed MREE. The latencies of Group CR5/t were significantly shorter than those of Group CR1 on trial 66,  $T(5) = 17$ . This reversed MREE is consistent with the results obtained under operant training conditions (Papini, 1997; Papini & Thomas, 1997).

For the purpose of a further comparison, Fig. 1, bottom panel, includes the extinction performance of Group PR5. Altogether, these results demonstrate a PREE and a reversed MREE in the same experiment, and under spaced-trial conditions, a result that has never been reported in the literature. This finding eliminates some of the concerns raised in connection with previous studies. First, in the key-pecking studies cited above, birds were not naïve at the start of the study. Therefore, it was possible that some aspect of prior experience had contributed to the dissociation. This does not apply to the present results because these pigeons were naïve. Second, most of the work on the paradoxical learning effects has been conducted in runway situations (see Amsel, 1992), thus raising the possibility that the dissociation was a task-specific effect limited to the key-pecking paradigm. The present work in the runway eliminates this possibility.

As mentioned previously, the reversed MREE found in Experiment 1 can be easily understood in terms of a strengthening–weakening model of learning. Large reinforcements strengthen the instrumental response more than do small reinforcements and, as a result, extinction is slower in the former case. However, the PREE is inconsistent with such a model. Vice versa, the PREE is consistent with several models developed to account for the paradoxical effects, but these models are inconsistent with the reversed MREE effect. Therein lies the challenge of the present dissociation of paradoxical effects in pigeons.

## Experiment 2

The aim of Experiment 2 was to determine whether paradoxical extinction depends on the specific presence of surprising nonreward during acquisition training, or on the inconsistency of rewarding outcomes in general. One possibility is that

the complete omission of an expected reinforcer activates a process that remains unaffected by the presentation of small, consistent rewards. Alternatively, it may be that persistence in extinction results from exposure to uncertain reward outcomes, independently of whether such outcomes involve nonreinforcement. These possibilities were tested by training pigeons in a condition involving a random mixture of large and small reinforcers, rather than reinforced and nonreinforced trials. Experiments using such a procedure have traditionally reported a so-called variable magnitude of reinforcement extinction effect (VMREE), that is, greater persistence in extinction after acquisition training with variable magnitudes than after training with continuous, large reinforcer magnitude (e.g., Logan, Beier, & Kincaid, 1956). The first hypothesis outlined above (i.e., persistence requires surprising nonreward) would be supported by a reversed VMREE, whereas the second hypothesis (i.e., persistence requires outcome uncertainty) would be supported by a VMREE.

Three groups were included in Experiment 2. Group VM (variable magnitude) received either 5 or 0.3 g of grain in random order during acquisition trials. Group CL (continuous large; similar to CL5/t in Experiment 1 and, thus, matched for trials with Group VM) was always reinforced with 5 g of food per trial. Finally, Group CA (continuous average; matched for trials and total reward with Group VM) was always reinforced with 2.65 g of grain per trial, that is, the average amount of food received by Group VM. All groups were then given extinction training similar to that of the previous experiment.

### *Method*

*Subjects and apparatus.* The subjects were 18 naïve pigeons, sexually mature, and of undetermined sex. Pigeons were housed and maintained as described in Experiment 1. The apparatus was the same used in Experiment 1.

*Procedure.* Initial magazine and pretraining involved the same procedures described in Experiment 1. Pigeons were assigned to one of three groups ( $n = 6$ ) according to the number of pretraining trials required. Triplets matched in terms of the length of pretraining were assigned to groups randomly. Acquisition trials were like those of Experiment 1 with the following exceptions. Group VM (variable magnitude) received either 5 or 0.3 g of grain per trial in Gellermann (1933) order. Each of the next two groups was trained with a consistent magnitude and thus received the same amount of grain in the goal box on every acquisition trial. Group CL (continuous large) was given 5 g of grain per trial, whereas Group CA (continuous average) was given 2.65 g of grain per trial (the average amount received by pigeons in Group VM). There were 24 acquisition trials followed by 24 extinction trials. A single conditioning trial occurred on each day. Trials in extinction were like those in acquisition except that reward was withheld on every trial for all of the groups. In all trials, pigeons were confined for 3 min in the goal box. Because the order in which birds were run in any given day was randomized, the intertrial interval was close to 24 h on average. The same procedure for data analysis described in Experiment 1 was used in the present experiment.

### Results and discussion

A Kruskal–Wallis analysis confirmed that the groups did not differ in the amount of pretraining, or in their performance in the last trial of acquisition. The mean (range) latency scores in the last acquisition trial were 5.8 s (3.0–9.4), 11.3 s (3.7–42.0), and 11.2 s (3.7–13.6) for Groups VM, CL, and CA, respectively.

Acquisition proceeded relatively fast and nondifferentially. Kruskal–Wallis tests failed to reveal any significant group differences in any of the target trials. The performance during extinction was, however, differential, as shown in Fig. 2. Extinction was considerably retarded in Group VM relative to both controls and then, once it started, it was relatively abrupt. Relatively abrupt behavioral transitions in extinction have been observed in pigeons trained both in the runway (see next experiment) and in the Skinner box (e.g., Papini et al., in press, Experiment 1), although the reasons are not entirely clear. A significant group effect was found on trial 30,  $H = 6.11$ , and a marginal effect occurred on trial 36,  $H = 5.24$ ,  $p = .073$ . Pairwise tests indicated that the latencies of Group VM were significantly lower than those of Group CA on trial 30,  $T(6) = 25$ , and marginally so on trial 36,  $T(6) = 28$ ,  $p = .078$ . The latencies of Group VM were also significantly lower than those of Group CL on trial 36,  $T(6) = 26$ , and marginally so on trial 30,  $T(6) = 27$ ,  $p = .055$ . These results confirm that the VMREE does occur in pigeons and provide the first evidence of such an effect under spaced-practice conditions in any species.

Groups CL and CA did not differ from one another on any of the extinction trials. Based on the results of Experiment 1, it might have been expected that Group CL would show greater persistence than Group CA. However, the smaller difference in reinforcer magnitude (5–2.65 g vs. 5–1 g) and in the number of acquisition trials (24 vs. 48) in this experiment, compared to Experiment 1, may account for the absence of a reversed MREE.

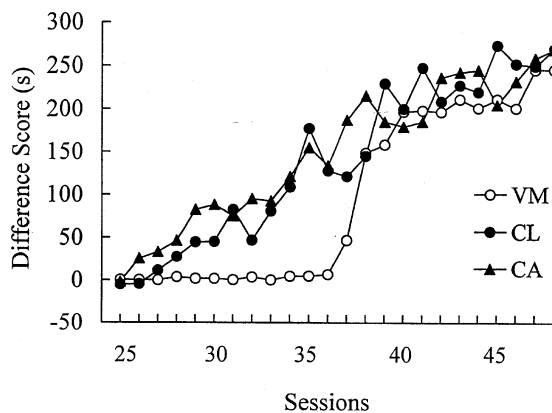


Fig. 2. Extinction performance of groups receiving different acquisition treatment. Group VM received a random mixture of trials ending in either 5 g or 0.3 g of food. Group CL received continuous reinforcement training with 5 g of food in each trial. Group CA received continuous reinforcement treatment with 2.65 g of food per trial.

### **Experiment 3**

The results of Experiments 1 and 2 suggest that response-outcome uncertainty in acquisition increases behavioral persistence in extinction. In this respect, pigeons and rats exhibit similar behavioral adjustments to conditions of unpredictable reinforcement (Amsel, 1992). The present and previous experiments with pigeons (see introduction for references) have explored persistence at the behavioral level. Whereas the behavioral similarities in PREE and VMREE among pigeons and rats suggest the presence of common learning processes, other evidence implies the possibility that such similarity is only superficial. From the evolutionary point of view, the presence of similar phenotypes opens the question of whether such similarity is based on common ancestry (i.e., homology) or on common selective pressures (i.e., homoplasy). Homologous learning mechanisms underlying any learning phenomenon (e.g., the PREE) across species would require that the brain structures, synaptic neurotransmitters, and cellular processes controlling behavior are the same in both species (Papini, 2002a). Differences at any of these mechanistic levels would suggest that the behavioral similarity under comparison is generated by divergent mechanisms. Birds show an impressive series of homoplastic similarities with mammals—and in comparison with extant reptiles—including high metabolic rates, endothermy, relatively large brain size, sleep-wakefulness cycle, complex parental care behavior, and high levels of general activity among others (Papini, 2002b). The potential for homoplastic evolution in paradoxical performance is also suggested by the dissociation between PREE and MREE, which occurs in pigeons but not in rats.

Experiment 3 makes a preliminary test of the homology vs. homoplasy hypotheses of paradoxical extinction in pigeons, relative to rats, by studying the neurochemical basis of the PREE. There is extensive pharmacological research with rats and other mammalian species on the paradoxical effects in general, and the PREE in particular (Amsel, 1992; Flaherty, 1996; Gray & McNaughton, 2000). As a result, it is possible to select drugs that are known to produce specific effects when administered to rats trained under conditions analogous to those used in the present experiments with pigeons. Eight groups of pigeons were used to study the effects of three drugs. The goal was to test pigeons with pharmacological agents known to disrupt, facilitate, or have no effect on the size of the PREE in rats. The three selected drugs that produce these effects were, respectively, chlordiazepoxide, nicotine, and haloperidol.

Chlordiazepoxide (CDP) is an anxiolytic that binds predominantly to a subunit of the GABA<sub>A</sub> ( $\gamma$ -amino butyric acid) receptor, increasing the receptor's affinity for the inhibitory neurotransmitter GABA. The inhibitory effects of GABA result from increased chloride ion conductance across the cell membrane, a process that hyperpolarizes the cell and decreases the likelihood of subsequent action potentials (Haefely, 1991). GABA<sub>A</sub> receptors are widely distributed in the telencephalon and diencephalon of birds and mammals, exhibiting a similar pattern of density suggesting that the evolution of the GABAergic system has been highly conservative (Veenman, Albin, Richfield, & Reiner, 1994). Chronic CDP administration in rats during both acquisition and extinction attenuated the spaced-trial PREE in both running (Feldon & Gray, 1981) and lever-pressing situations (McNaughton, 1984). More specifically,

CDP tends to decrease persistence in partially reinforced subjects but to increase persistence in continuously reinforced subjects. CDP has also been shown to reduce or eliminate other behavioral phenomena based on surprising nonreward in mammals (for reviews, see Flaherty, 1996; Gray & McNaughton, 2000) and thus was selected for study in the present experiment. There is little information on the effects of CDP on performance in situations involving surprising nonreward in pigeons. However, anxiolytic-like effects similar to those observed in rats have been reported in pigeons trained on an approach-avoidance conflict situation (Evenden, 1991; Valentine & Barrett, 1981; Wuttke & Halleher, 1970). In such a situation, key-pecking responses maintained by food reinforcement occasionally lead to punishment with electric shock; CDP increases the frequency of such punished responding thus suggesting a suppression of fear. Because drugs that suppress fear tend to also alleviate performance in situations involving surprising reward downshifts, it was predicted that CDP would reduce or eliminate the PREE in pigeons.

The second drug used in Experiment 3 was nicotine (Nic). Nic is a stimulant that binds selectively to a subclass of cholinergic receptors known as nicotinic receptors. Nicotinic receptors are found in mammalian brain areas in which there is a high number of dopaminergic and serotonergic neurons, such as the substantia nigra and the ventral tegmental area (Jarvik & Schnaider, 1992), where it increases catecholamine synthesis (Mitchell, 1993). Grigoryan and Gray (1996) gave rats a single Nic (or saline) injection on the first day of a runway experiment. Following 3 days of alley exposure, half of the subjects in each injection condition were trained on either partial or continuous reinforcement schedules. Acquisition lasted for 16 trials followed by 10 trials of extinction, all administered at a rate of one trial per day. Whereas the saline condition yielded no evidence of a PREE, a significant PREE was found among the Nic groups.

Although the design of this experiment is somewhat unusual (e.g., a single Nic injection was given at the start of training), the evidence that Nic may enhance the spaced-trial PREE in rats stands as the only evidence of a pharmacological manipulation that increases the size of this effect. Little is known about the effect of Nic on learning in pigeons. Therefore, Nic was chosen for analysis in the present experiment.

The third drug used in Experiment 3 was haloperidol (Hal). Hal is an antipsychotic drug that binds predominantly to dopaminergic receptors, where it has an antagonistic effect (Weiner & Molinoff, 1994). In rats, Hal injections given in acquisition and extinction reduce persistence in extinction after both partial and continuous reinforcement training, without affecting the size of the PREE (Feldon, Katz, & Weiner, 1988; Feldon & Weiner, 1991). Again, little is known about the effects of Hal on learning situations involving surprising nonreward in pigeons. Hal has no effect on punished behavior in the conflict procedure described previously (e.g., Millan, Brocco, Gobert, Schreiber, & Dekeyne, 1999). Other experiments indicate that Hal may have a nonspecific decreasing effect on response rate in a variety of situations; however, Hal does not appear to affect the accuracy of pigeon performance in delayed matching-to-sample situations (Poling, Picker, & Thomas, 1984).

To summarize, then, the drugs selected for an initial analysis of the PREE in pigeons have been shown to reduce (CDP), enhance (Nic), and have no effect (Hal) on the spaced-trial PREE in experiments with rats. In each case, little or nothing is known about the effects of these drugs in learning experiments with pigeons. Assuming that the mechanisms underlying the PREE in rats and pigeons are homologous, then these drugs are expected to have similar effects in pigeons. If, however, as behavioral experiments suggest, the PREE found in pigeons is only superficially similar to that found in rats, then it is expected that these drugs would provide a different set of results compared to the results reported in rats trained under analogous conditions.

### *Method*

*Subjects and apparatus.* The subjects were 40 naïve pigeons, sexually mature at the time of training and of undetermined sex. Housing and maintenance conditions were as described in Experiment 1. The same runway used in the previous experiments was also used here.

*Drug treatments.* The design involved four drug treatments (CDP, Nic, Hal, and an ethanol control), two reinforcement schedules (continuous and partial reinforcement), and a total of 48 daily trials. Pigeons assigned to Groups CR/CDP and PR/CDP received CDP injections, 4.0 mg/kg in 1.0 ml/kg 0.9% NaCl. Pigeons assigned to Groups CR/Nic and PR/Nic received Nic injections, 0.8 mg/kg in 1.0 ml/kg 0.9% NaCl. Pigeons assigned to Groups CR/Hal and PR/Hal received Hal injections, 0.10 mg/kg in 95% ethanol. Finally, pigeons assigned to Groups CR/Eth and PR/Eth received injections of 95% ethanol. Ethanol was the vehicle used for preparing Hal and it was thus selected as a control substance against which to measure the effects of all three drugs. In rats, ethanol may reduce the response to surprising reward downshifts (Becker & Flaherty, 1983) and it is, therefore, a conservative control condition relative to the other vehicles used in this experiment. Any effects of ethanol would tend to make the effect of CDP more difficult to detect and the effect of Nic appear greater than they may actually be. Hence, any conclusions regarding the effects CDP and Nic should be considered tentative; a more appropriate saline control could be included in subsequent experiments if warranted. The dosages for both CDP and Hal were selected on the basis of earlier, unrelated work with pigeons (e.g., Evenden, 1991; Nader & Hannermann, 1993; Poling et al., 1984).

All the drugs were purchased from Sigma Chemicals (St. Louis, MO). Injections were matched in volume (1 ml/kg), intramuscular (i.m.), and administered approximately 30 min before each trial during both acquisition and extinction trials. Daily injections were placed in alternating pectoral muscles to minimize the discomfort that can result from chronic injections.

*Behavioral procedure.* Magazine training and shaping was identical to the previous experiments. Based upon the number of required pretraining trials, pigeons were randomly assigned to one of eight groups ( $n = 5$ ). There were 24 daily trials of acquisition. In each acquisition trial, pigeons in the CR groups received 5 g of grain in the goal box. The pigeons in each of the PR groups received 12 reinforced trials end-

ing with access to 5 g of grain and 12 nonreinforced trials ending with access to an empty goal box. The sequence of reinforced and nonreinforced trials in the PR groups was determined by Gellermann (1933) orders. Acquisition was followed by 24 daily trials of extinction similar to the nonreinforced trials of PR groups. In all trials, pigeons were enclosed in the goal box during 3 min. A single trial per day was administered throughout the experiment, at approximately the same time of the day, resulting in an intertrial interval approaching 24 h. Other procedural details, including data analysis, were as described in Experiment 1.

### *Results and discussion*

A Kruskal–Wallis analysis confirmed that the groups did not differ in the length of pretraining. Furthermore, groups were not different in their performance during the last acquisition trial. The mean (range) latencies for the last acquisition trial were 4.1 s (3–4.8) and 5.5 s (3.9–7.3) for Groups CR/CDP and PR/CDP; 9.6 s (3.1–24.6) and 6.4 s (3.5–9.7) for Groups CR/Nic and PR/Nic; 4.4 s (2.8–6.9) and 5.8 s (3.1–12) for Groups CR/Hal and PR/Hal; and, finally, 4.5 s (1.1–5.4) and 4.7 s (2.9–6.1) for Groups CR/Eth and PR/Eth.

Planned comparisons of two types were performed on the difference scores. First, pairwise comparisons involving groups exposed to CR and PR treatments were conducted for each drug treatment to determine whether a PREE had occurred in each case (e.g., CR/CDP vs. PR/CDP). Second, pairwise comparisons were made against the ethanol controls for the CR groups (e.g., CR/Eth vs. CR/CDP) and PR groups (e.g., PR/Eth vs. PR/CDP). The purpose of these comparisons was to determine how the PREE was modulated by each of the drugs.

*Ethanol controls (Eth).* Wilcoxon rank sums tests indicated that Group CR/Eth and Group PR/Eth did not differ on any of the acquisition trials selected for analysis. In extinction, the latencies of Group CR/Eth were significantly longer than those of Group PR/Eth on trials 30 and 36,  $T_s(5) < 18$ . Hence, a PREE emerged in the first half of the extinction in the ethanol controls.

*Chlordiazepoxide (CDP).* Wilcoxon rank sums tests failed to detect any differences in acquisition between Groups CR/CDP and PR/CDP. The extinction data are presented in Fig. 3 together with the data from the ethanol controls. Extinction was faster in the CR groups than in the PR groups for both CDP and ethanol, thus demonstrating the presence of PREEs. If anything, it would appear that CDP delayed the extinction of runway performance thus retarding the emergence of the PREE. The latencies of Group CR/CDP were significantly longer than those of Group PR/CDP on trials 42 and 48,  $T_s(5) < 18$ , thus confirming the late emergence of the PREE under CDP treatment. Follow-up comparisons with the ethanol controls showed that CDP increased persistence in both PR and CR conditions. Group CR/Eth had a significantly longer latency on trial 36 than Group CR/CDP,  $T(5) = 17$ , and Group PR/Eth's latencies were significantly longer than those of Group PR/CDP on trials 42 and 48,  $T_s(5) = 17$ . In sum, CDP delayed the onset of extinction in both continuous and partial reinforcement conditions, and enhanced persistence after both partial and continuous reinforcement training.

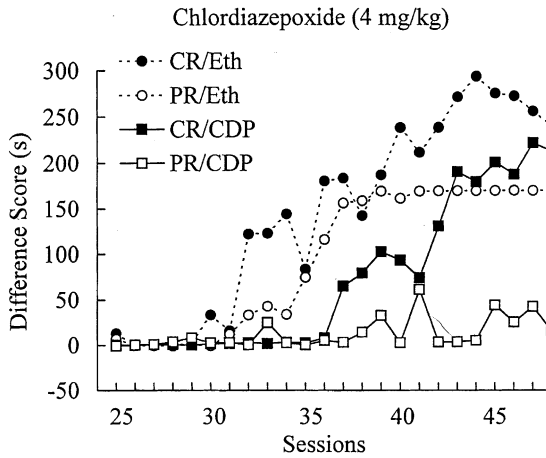


Fig. 3. Extinction performance of groups receiving different acquisition treatment (CR, continuous reinforcement; PR, partial reinforcement) and different drug treatment (Eth, ethanol; CDP, chlordiazepoxide).

*Nicotine (Nic)*. Wilcoxon rank sums tests revealed nonsignificant group differences during acquisition. As shown in Fig. 4, the PREE demonstrated in the ethanol groups failed to appear in the groups treated with Nic. Group CR/Nic did not differ from Group PR/Nic on any of the extinction trials. Follow-up comparisons against the ethanol controls showed that latencies were significantly longer in Group CR/Eth than in Group CR/Nic on trials 30, 36, and 42,  $T_s(5) < 18$ . However, extinction was unaffected by nicotine in the PR condition. This suggests that Nic abolished the PREE because of an enhancement of persistence following conditioning with continuous, but not partial reinforcement.

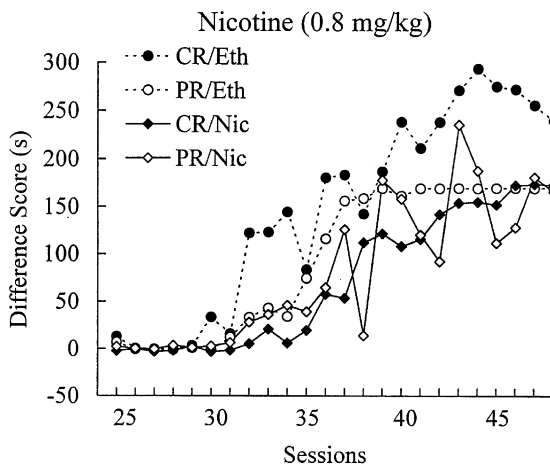


Fig. 4. Extinction performance of groups receiving different acquisition treatment (CR, continuous reinforcement; PR, partial reinforcement) and different drug treatment (Eth, ethanol; Nic, nicotine).

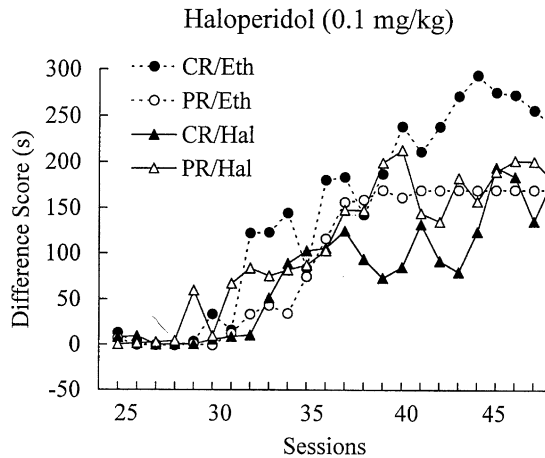


Fig. 5. Extinction performance of groups receiving different acquisition treatment (CR, continuous reinforcement; PR, partial reinforcement) and different drug treatment (Eth, ethanol; Hal, haloperidol).

Table 1  
Predicted and obtained results with drug manipulations in Experiment 3

Drug treatment	Predicted effect on the PREE	Obtained effect on the PREE
Chlordiazepoxide	Eliminated	Delayed its emergence
Nicotine	Enhanced	Eliminated
Haloperidol	No effect	Eliminated

Note. Predictions are based on the results of analogous experiments with rats (see text for references).

*Haloperidol (Hal)*. Wilcoxon rank sums tests failed to detect any differences in acquisition. The extinction results are presented in Fig. 5. As with Nic, Hal eliminated the PREE. None of the target trials yielded significant group differences. Follow-up comparisons in extinction showed that Groups PR/Eth and PR/Hal did not differ on any of the trials, but the latencies of Group CR/Eth were significantly longer than those of Group CR/Hal on trial 42,  $T(5) = 17$ . The PREE was thwarted with Hal because of an increase in persistence after continuous reinforcement training.

Based on previous work with rats, CDP was expected to abolish the PREE, Nic was expected to increase the size of the PREE, and Hal was not expected to affect the PREE. The obtained results were in sharp contrast to these predictions, as summarized in Table 1.

### General discussion

The objectives of this series of experiments were threefold. First, these experiments were designed to provide evidence of dissociation in paradoxical effects in pigeons within the same experiment. Experiment 1 provided such evidence by demonstrating the concurrent presence of a PREE and a reversed MREE in the

spaced-trial extinction performance of pigeons. Second, they were planned to provide evidence of a dissociation in paradoxical extinction using the same runway procedure extensively used in experiments with rats. Third, they were intended to provide initial evidence on the mechanisms underlying the spaced-trial PREE in pigeons. Experiment 2 determined that persistence in pigeons depends upon response-outcome inconsistency. Experiment 3 yielded initial data suggesting that the pigeon PREE, unlike that found in rats, might depend on cholinergic and dopaminergic pathways, but not on GABAergic pathways.

Experiment 1 successfully demonstrated a dissociation of the effects of reward uncertainty and reward magnitude in spaced-trial training (see also Papini et al., in press). Previous evidence indicated that the paradoxical learning effects, when studied with sexually mature animals and with spaced trials, either occur (as in mammals) or do not (as in fish, amphibians, and reptiles). Such an all-or-none pattern of results suggests that the effects of reward uncertainty and reward magnitude depend on a single mechanism. This mechanism might be the one suggested by frustration theory, based on the anticipation of an emotional reaction of frustration induced by surprising reward downshifts (Amsel, 1992), or the one suggested by sequential theory, based on the carry-over or reinstatement of reward and nonreward memories from previous trials (Capaldi, 1994), or some combination of processes depending on the training circumstances (see Mackintosh, 1974). In nonmammalian vertebrates (e.g., fish, amphibians, and reptiles), the absence of paradoxical extinction suggests that associative learning rests on a simple strengthening–weakening mechanism (see introduction for references). The results of Experiment 1 provide an interesting challenge to this picture: in pigeons, partial reinforcement increases persistence relative to continuous reinforcement (i.e., PREE), but it is large rewards, rather than small ones, that increase persistence in extinction (i.e., reversed MREE).

One difference between partial reinforcement training and magnitude of reinforcement training lies in the types of reinforcing outcomes that are presented in each case. The former involves the random presentation of rewarded and nonrewarded trials, whereas the latter involves access to either a large or a small reward magnitude in a consistent manner. A spaced-trial key-pecking experiment shows that the reversed MREE occurs even when the large and small rewards are paired with different stimuli in a within-subject design (Papini, 1997, Experiment 3). Thus it seemed possible that pigeons use different mechanisms for assessing nonreward than for assessing different reward magnitudes. Experiment 2 tested the hypothesis that response-outcome inconsistency increases persistence in extinction, rather than mere exposure to nonreinforcement. This was accomplished by training pigeons in a situation involving a random mixture of large and small reinforcers. Unlike in Papini's (1997) experiment in which each magnitude was signaled by its own discriminative stimulus, a single stimulus situation was paired with both magnitudes randomly in Experiment 2, thus creating reward uncertainty. The results confirmed that uncertainty in reward size also increases persistence, having an effect on extinction similar to that of partial reinforcement.

The dissociation between the effects of reward uncertainty and reward magnitude in pigeons may be understood in terms of two different hypotheses: either pigeons

and rats differ quantitatively in some crucial mechanism underlying paradoxical extinction, or there is simply a species difference in the mechanisms engaged by extinction as a function of prior training. The idea of a quantitative difference is suggested by developmental research with rats. Paradoxical effects emerge gradually and at different rates during the initial 25 days of life in infant rats, thus producing a similar dissociation to that observed in adult pigeons. Using a runway situation, access to milk as the reinforcer, and relatively massed training conditions, the PREE emerges at days 12–14, the VMREE at days 16–18, and the MREE at days 20–21 (Chen & Amsel, 1980; Chen, Gross, & Amsel, 1981; Stanton & Amsel, 1980). Thus an 18-day-old rat demonstrates both the PREE and VMREE, but not the MREE, exactly the pattern found in analogous experiments with adult pigeons. During these initial 25 days of age, the septo-hippocampal formation of infant rats is undergoing maturation (see Gahwiler, 1984). About 72% of the granule cells in the dentate gyrus form after birth (Altman & Bayer, 1975), whereas the perforant pathway and mossy fibers form during the first week of life and reach maturation between days 9 and 25 (Zimmer & Haug, 1978). This correlation between the emergence of the paradoxical effects and the maturation of the septo-hippocampal system in infant rats is relevant in light of the fact that lesions of this system in adult rats also affect paradoxical extinction and related phenomena (Feldon & Gray, 1979; Feldon, Rawlins, & Gray, 1985; Flaherty, Coppotelli, Hsu, & Otto, 1998; Franchina & Brown, 1971). Daly (1991) reasoned that the gradual maturation of septo-hippocampal circuits could be modeled in terms of a gradual increase in the impact of surprising nonreward on extinction performance. With this simple assumption, Daly was able to simulate the order of emergence of the paradoxical effects as determined experimentally. For the purpose of the present discussion, what is important is the possibility—suggested by Daly's simulations—that the PREE and VMREE may be more sensitive to the effects of surprising nonreward than the MREE. If this were the case, then the dissociation found in adult pigeons would reflect simply the differential sensitivity of learning phenomena to the consequences of surprising nonreward, rather than a species difference in underlying mechanisms.

The second hypothesis, which suggests that the dissociation of paradoxical extinction effects in pigeons reflects a qualitative difference in underlying mechanisms relative to rats, is more consistent with the results of Experiment 3. Behavioral research alone can rarely produce unambiguous evidence of homology of mechanism because such data are open to the possibility that similar learning phenomena may be the result of different, homoplastic mechanisms (Papini, 2002a). One way to distinguish between homology and homoplasia of mechanisms is to use drug manipulations, as was done in Experiment 3. The goal of this approach is to use drugs as tools to disentangle the two possible evolutionary hypotheses of homology and homoplasia. Such an approach would shed light on the evolutionary pattern characterizing learning mechanisms and also provide support (or not) for the use of the pigeon as an animal model of anxiety-related processes (see, e.g., Barrett & Gleeson, 1991). Although generally implicit, it is perhaps relevant to emphasize that the development of a valid animal model for any behavioral or physiological disorder implies a degree of homology in underlying mechanisms between the model species and the human species.

None of the drugs examined produced effects in pigeons that were similar to the results reported in analogous experiments with rats (see introduction to Experiment 3 for references and Table 1 for a summary of predicted and obtained results). In rats, CDP eliminates the PREE, but in pigeons it only delays its emergence without disrupting it. Nicotine was shown to enhance the PREE in rats, but it increased persistence after continuous reinforcement in pigeons, thus eliminating the PREE. Haloperidol does not affect the size of the PREE in rats but, in pigeons, it increased persistence after continuous reinforcement training leading to the elimination of the PREE. The drugs studied targeted different neurotransmitter systems (i.e., GABAergic, cholinergic, and dopaminergic), a choice that seemed appropriately wide to initiate a systematic study of drug effects on the PREE in an avian species. The results of Experiment 3 suggest two alternative possibilities. The first possibility is that the pharmacological action of the drug is the same in pigeons and rats, but the behavioral consequences of the drug are different. The second possibility is that reward inconsistency engages different learning mechanisms in pigeons and rats.

Do these drugs produce similar behavioral effects in pigeons and rats? Consider CDP. Witkin and Barrett (1986) and Barrett and Witkin (1991) reported dose–response curves for CDP in pigeons trained in a conflict paradigm in which key-pecking is paired with a food and, occasionally, with electric shock. Saline controls exhibit a greater degree of suppression of key-pecking than pigeons given CDP. Importantly, punished responding is not a product of analgesia, muscle relaxation, or the anticonvulsant qualities of anxiolytic drugs. With a dose of CDP similar to that used in Experiment 3, pigeons made about 10 times as many responses during the punishment period as the controls. This suggests that the dose used in Experiment 3 should have been sufficient to produce an anxiolytic effect that would have been expected to increase persistence after continuous reinforcement training and to decrease persistence after partial reinforcement training, as it does in rats. Of course, this assumes that both fear (i.e., triggered by an expectation of impending pain) and anticipatory frustration (i.e., triggered by an expectation of reward loss) can be described as forms of anxiety, as indeed research with mammals suggests (Gray & McNaughton, 2000). The failure of CDP to eliminate the PREE in pigeons is consistent with the conclusion that frustration resulting from reward inconsistency is not a determinant of persistence in pigeons. However, the similar effects of CDP on conflict behavior supported by fear conditioning resulting from the administration of painful stimuli in rats and pigeons suggest a degree of homology. One implication of these results is that the brain mechanisms subserving fear and anticipatory frustration are dissociable, with the fear mechanisms being common to both species, whereas the frustration mechanism is either present only in rats, or is present in both species but organized on the basis of different physiological processes. This last possibility would be consistent with the effects of nicotine and haloperidol in Experiment 3. The fact that both of these drugs eliminated the PREE suggests that the mechanism underlying adjustment to reward uncertainty is based on cholinergic and dopaminergic pathways in the pigeon, whereas that mechanism may be based on GABAergic pathways in the rat.

Whereas these results are encouraging in terms of a comparative analysis of learning in birds and mammals, there are a number of caveats that mitigate the strength of the conclusions that may be derived from them. First, dose–response functions would have to be determined for each drug before firm conclusions can be drawn concerning the involvement of a given neurochemical system on the PREE in pigeons. This is particularly true for CDP; it is plausible that a larger dose would attenuate or even eliminate the PREE, which would implicate a GABAergic involvement in the pigeon PREE, just as is the case for rats. Second, a more precise determination of the neurochemical system involved in the production of the PREE will require drugs that are more specific than those used here. For example, haloperidol binds to several receptor types and therefore cannot provide unambiguous evidence for one specific mechanism of action. The encouraging results of the present experiments provide an impetus for studying the effects of more selective drugs on the pigeon PREE in future experiments. Third, the use of ethanol as a control condition was appropriate to evaluate the effects of haloperidol but was not the ideal control condition for evaluating CDP and nicotine. Finally, the experiment reported by Grigoryan and Gray (1996), used as a source for the effects of nicotine on the PREE in rats, is really quite different from the way in which nicotine was administered to pigeons in Experiment 3. Grigoryan and Gray administered a single injection at the outset of training, whereas pigeons received 48 daily injections of nicotine. Furthermore, they had no evidence of a PREE in the saline controls, but only in the nicotine groups. The inference they made was that nicotine caused the emergence of a PREE under conditions that would not normally produce it, but this is clearly different from observing an increase in the size of the PREE from saline to nicotine controls.

The present studies illustrate the importance of a multilevel analysis of learning phenomena for a comparative study of learning mechanisms. In comparing the ways in which pigeons and rats adjust to changes in reward parameters, the behavioral similarities in terms of the PREE and VMREE suggested common underlying mechanisms. However, a modulation of the PREE by drugs selected on the basis of their action in analogous experiments with rats revealed a different pattern of results in the pigeon. The comparative study of a particular set of learning phenomena (e.g., the paradoxical effects) must be extended to several levels of analysis (e.g., behavioral, neurobiological, neurochemical, cell-molecular) before hypotheses about the evolutionary pattern of underlying mechanisms can be successfully tested (Papini, 2002a). With multi-level information at hand, it will be possible to develop a more complete theory of persistence in pigeons and to understand the evolution of learning mechanisms underlying the behavioral adjustment to surprising reward downshifts in vertebrates.

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