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Administration of Corticosterone After the First Downshift Trial Enhances Consummatory Successive Negative Contrast

Mariana Bentosela
Instituto de Investigaciones Médicas Lanari and Universidad de Buenos Aires

Eliana Ruetti and Rubén N. Muzio
Instituto de Biología y Medicina Experimental and Universidad de Buenos Aires

Alba E. Mustaca
Instituto de Investigaciones Médicas Lanari and Universidad de Buenos Aires

Mauricio R. Papini
Texas Christian University

Rats given access to a 32% sucrose solution and then downshifted to a 4% solution exhibit less contact with the sipper tube than unshifted controls always given access to 4% solution. This phenomenon, called *consummatory successive negative contrast*, was facilitated in Experiment 1 by a posttrial injection of corticosterone (3 mg/kg) administered immediately after the first downshift trial. Experiment 2 demonstrated that this facilitatory effect of posttrial corticosterone does not occur when administered 3 hr after the first downshift trial. These results support the hypothesis that corticosterone strengthens an aversive emotional component elicited by the surprising downshift in reward magnitude during the initial downshift trial.

Keywords: incentive contrast, corticosterone, aversive memory, frustration, rats

After a downshift from a 32% sucrose solution to a 4% sucrose solution (32 → 4), rats exhibit a temporary suppression of consummatory behavior relative to rats that always drank 4% sucrose (4 → 4). This phenomenon, called *consummatory successive negative contrast* (cSNC), serves as a model situation to study the intersection between learning, motivation, and emotion (see Flaherty, 1996; Papini, 2003). An unresolved issue about cSNC concerns the nature of the initial reaction to reward downshift. According to Flaherty's (1996) multistage model of cSNC, during the first trial of exposure to the downshifted 4% solution, rats go through a series of stages involving the detection of the shift, the rejection of the 4% solution, and the search for the missing 32% solution. Flaherty (1996) suggested that this "early reaction to reward reduction might be considered to be cognitive—a search for the 'missing' substance" (p. 95). Consistent with this model, during the first postshift trial rats display behaviors that may be interpreted as involving appetitive search (Pecoraro, Timberlake, & Tinsley, 1999; Pellegrini & Mustaca, 2000). Furthermore, rats show no reduction in the intensity of cSNC when treated with benzodiazepine anxiolytics and no evidence of corticosterone release after the first postshift trial (Flaherty, Becker, & Pohorecky, 1985; Flaherty, Grigson, & Rowan, 1986; Mitchell & Flaherty, 1998). The multistage model assumes that starting on the second postshift trial, an emotional response is added to the situation in the form of an approach–avoidance conflict. According to Flaherty (1996), the approach component is triggered by the need to consume the 4% solution, whereas the avoidance component is triggered either by an emotional opponent process (e.g., Solomon & Corbit, 1974) or by the failure to locate the 32% solution during the search phase. The presence of a conflict accounts for the effectiveness of benzodiazepine anxiolytics to reduce or eliminate cSNC when administered in the second postshift trial (Flaherty et al., 1986). Furthermore, corticosterone is elevated both before (possibly in anticipation of conflict) and after (possibly as an aftereffect of conflict) the second postshift trial (Flaherty et al., 1985; Mitchell & Flaherty, 1998).

Papini (2003) suggested a second possible interpretation of cSNC based on Amsel's (1992) frustration theory. Frustration theory was originally developed to deal with instrumental conditioning situations involving surprising omissions or reductions in reward magnitude or quality, so its application to cSNC is consistent with the original data set this theory was designed to explain. This view is similar to the multistage model in assuming the development of an approach–avoidance conflict, but it differs from it in assuming that the suppression observed during the initial trial after the downshift also involves an emotional aversive reaction. The frustration hypothesis of cSNC is consistent with the cognitive stage of detection assumed by the multistage hypothesis. However, the frustration hypothesis suggests that the initial rejection of the

Mariana Bentosela and Alba E. Mustaca, Laboratorio de Psicología Experimental y Aplicada (PSEA), Instituto de Investigaciones Médicas Lanari and Facultad de Psicología, Universidad de Buenos Aires, Buenos Aires, Argentina; Eliana Ruetti and Rubén N. Muzio, Laboratorio de Biología del Comportamiento, Instituto de Biología y Medicina Experimental (CONICET) and Facultad de Psicología, Universidad de Buenos Aires, Buenos Aires, Argentina; Mauricio R. Papini, Department of Psychology, Texas Christian University.

Eliana Ruetti is now at PSEA, Instituto de Investigaciones Médicas Lanari.

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Correspondence concerning this article should be addressed to Mauricio R. Papini, Department of Psychology, Texas Christian University, Box 298920, Fort Worth, TX 76129. E-mail: m.papini@tcu.edu

4% solution is triggered by a state of primary frustration induced by the discrepancy between the expectation of a 32% reward acquired during the preshift trials and the actual 4% reward encountered on the first postshift trial. This initial emotional reaction (primary frustration) is hedonically aversive, and it induces rejection with minimal or no conflict, serving as an unconditioned stimulus for the acquisition of secondary frustration through Pavlovian conditioning (i.e., pairings of stimuli present at the time of reward downshift with the internal state of primary frustration; Amsel, 1992). Secondary frustration is theorized to be an anticipatory version of primary frustration that the rat may experience when not in actual contact with the sipper tube but when anticipating the downshifted solution. Once acquired, secondary frustration plays its role as the avoidance component of the conflict induced at full strength during the second postshift trial.

There is some independent evidence supporting the distinction between primary and secondary frustration at the neural level from experiments involving instrumental behavior (Gray, 1977; Henke, 1973, 1977), as well as of the hedonically aversive nature of reward downshift (Daly, 1974). There is also support for the hypothesis that the first postshift trial may involve emotional processes. For example, rats downshifted from 32% sucrose to water exhibit suppression of aggressive behavior relative to rats given access only to water (Mustaca, Martínez, & Papini, 2002). Moreover, opioid antagonists, including morphine and DPDPE, attenuate cSNC during the first postshift trial (Rowan & Flaherty, 1987; Wood, Daniel, & Papini, 2005), whereas opioid antagonists, including naloxone and naltrindole, enhance cSNC during the first postshift trial (Pellegrini, Wood, Daniel, & Papini, 2005). Furthermore, partial reinforcement training during preshift trials (a random mixture of access to 32% sucrose and water that may be thought of as a stress inoculation procedure) attenuates cSNC after a 32 → 4 downshift (Pellegrini, Muzio, Mustaca, & Papini, 2004). Of interest, the ameliorating effect of partial reinforcement on the first postshift trial is eliminated by the administration of the benzodiazepine anxiolytic chlordiazepoxide before nonreinforced preshift trials (Pellegrini et al., 2004). Convergent evidence from a variety of sources, including aggressive behavior, opiate treatment, and partial reinforcement training, suggests the presence of an emotional response during the first postshift trial. This evidence is more consistent with the account of cSNC based on frustration theory than with the multistage model of cSNC.

The evidence reported in this article comes from yet another approach: posttrial administration of corticosterone. The glucocorticoid hormone corticosterone is a well-known marker of stress (see Korte, 2001), also active in situations involving reward loss, including cSNC and extinction (see Papini & Dudley, 1997). Moreover, the increase in behavior that occurs in the early trials of appetitive extinction, which has been attributed to frustration, is also eliminated by adrenalectomy (Thomas & Papini, 2001). Of importance here are the effects of corticosterone administration after fear conditioning induced by pain, given the mechanistic similarities between frustration, fear, and pain (Eisenberger & Lieberman, 2004; Gray, 1987; Papini, 2003; Wood et al., 2005). In one experiment (Hui et al., 2004), the administration of corticosterone (3 mg/kg sc) after the last of three tone–shock pairings enhanced suppression of activity in a test administered a day later. Similarly, posttrial corticosterone (5 mg/kg ip) enhanced contextual fear conditioning induced by the administration of three unsignaled shocks and tested 1 and 7 days after acquisition (Cordero & Sandi, 1998). Posttraining administration of corticosterone also improves the retention of both T-maze active avoidance learning and punished drinking (Flood et al., 1978; Kovács, Telegdy, & Lissák, 1977). This evidence is usually interpreted as demonstrating the role of adrenal stress hormones in the consolidation of memories induced by emotional arousal (McGaugh, 2000; Roozendaal, 2000). Therefore, if the first postshift trial in the cSNC situation involves primary frustration, an aversive source of emotion (see Amsel, 1992; Papini & Dudley, 1997), then posttrial administration of corticosterone should enhance consummatory suppression and lengthen cSNC relative to vehicle controls. If, however, the rejection stage is part of a cognitive appraisal of the downshift, as suggested by the multistage model (Flaherty, 1996), then corticosterone should have little or no effect on cSNC.

Experiment 1

On the basis of the evidence from fear conditioning, we predicted that the posttrial administration of a moderate dose of corticosterone (3 mg/kg ip) would enhance cSNC. Such enhancement should be reflected in terms of a slower recovery of normal consummatory levels, compared with that of a vehicle control. The posttrial procedure of administering corticosterone has the advantage of eliminating the potential direct influence of this drug on consummatory behavior. Given that the half-life of corticosterone (5 mg/kg), as measured in plasma, is about 25 min (Sainio, Lehtola, & Roininen, 1988) and that the trial after corticosterone administration (Trial 12) occurred approximately 24 hr later, it is safe to assume that any effects of this procedure on consummatory behavior were not the result of high levels of circulating corticosterone produced by the posttrial injection.

Method

Subjects. The subjects were 48 naive male Wistar rats, about 4 months old at the start of the experiment. Ten days before the experiment, the subjects were transferred to individual cages with water freely available. The daily amount of food was gradually reduced until the rats' weights were lowered to 85% of individual ad libitum weights. The mean weight for the entire sample was 310 g. During training, the rats were fed daily at least 20 min after the training trial. The colony was under a 12-hr light–dark cycle (lights on at 0700). All training trials were administered between 1000 and 1400, during the light portion of the cycle. Temperature and humidity levels in the testing rooms and animal colony were kept relatively constant throughout the experiment.

Apparatus. Rats received training in four similar conditioning boxes enclosed in a sound-attenuating cubicle (MED Associates, East Fairfield, VT). Each box measured 24.1 cm in length, 29.2 cm in width, and 21 cm in height. The floor was made of aluminum bars (0.4 cm in diameter, 1.1 cm apart). In the center of one of the lateral walls there was a 5-cm hole, 3.5 cm deep, 1 cm above the floor level, through which a sipper tube could be introduced from the outside. When fully inserted into the hole, the sipper tube protruded 2 cm. A diffuse house light was located above the sipper tube, 18 cm above the floor. The goal-tracking time (in 0.01-s units) was the main dependent variable, and it was measured by detecting the insertion of the rat's head into the hole by means of a photocell. Goal-tracking time has been shown to yield results similar to more conventional dependent variables, such as licking rate measure (Riley & Dunlap, 1979) or amount of fluid consumed (Papini, Mustaca, & Bitterman, 1988). Furthermore, goal-tracking time correlates positively and significantly with the amount of fluid intake during 5-min trials (Mustaca, Freidín, & Papini,

2002). Under the conditions used in the present experiments, goal-tracking time yields data with less individual variability than the more typical licking frequency measure.

Procedure. Rats were randomly assigned to one of four groups (n = 12). Groups differed in terms of the reinforcer magnitude during the preshift trials (32% vs. 4% sucrose solution) and the drug treatment received immediately after the first postshift trial (vehicle vs. corticosterone). A total of 15 trials, one per day, were administered; each trial lasted 5 min from the first recording of goal tracking. During preshift trials (Trials 1–10), rats received 5 min of free access to either a 32% or a 4% sucrose solution, whereas during postshift trials (Trials 11–15), all rats received access to the 4% solution. Solutions were prepared weight to volume, by mixing 320 g (or 40 g) of commercial sugar in 1 L of tap water. Pairs of rats matched in terms of the goal-tracking performance obtained in Trials 9 and 10 were established, and individuals within each pair were randomly assigned to either the corticosterone or the vehicle condition. This gave rise to four groups: 32/veh, 32/cort, 4/veh, and 4/cort. Immediately after Trial 11, the first postshift trial, half of the rats received a subcutaneous injection of corticosterone (3 mg/kg). To prepare corticosterone (Upjohn Laboratories, Kalamazoo, MI), ethanol 100% was diluted in 0.9% isotonic saline to a 5% ethanol concentration; corticosterone was then diluted in this vehicle to a volume of 2 μl/kg. The rest of the rats received a vehicle injection (5% ethanol diluted in 0.9% isotonic saline).

Animals were tested in squads of four; the order of the squads was randomized across days. Each box was swept with a damp towel after each training trial. Goal-tracking times were subject to analysis of variance (ANOVA). Post hoc least significant difference (LSD) pairwise comparisons of selected trials were included when necessary to understand the source of interaction effects. In all of the statistical results reported in this article, the value of alpha was set at the .05 level.

Results and Discussion

Figure 1 shows the average goal-tracking times for all four groups and across all the trials of this experiment. During preshift trials, scores increased across trials and there was a tendency for rats in the groups receiving access to 32% solution to display higher goal-tracking times. A Contrast (32%, 4%) $\times$ Drug (corticosterone, vehicle) $\times$ Trial (1–10) ANOVA indicated the following significant effects. The groups receiving 32% solution had a higher performance than the groups receiving 4% solution, $F(1, 44) = 9.98$, $p < .004$, and consummatory performance increased across trials, $F(9, 396) = 45.72$, $p < .001$. None of the other effects reached a significant level (Fs < 3.23).

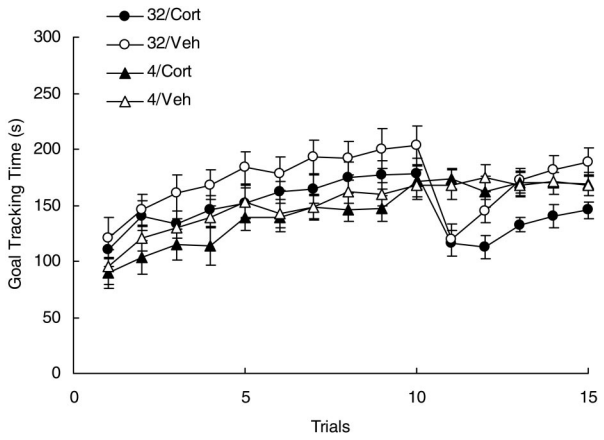

Figure 1. Consummatory performance of groups across trials. During Trials 1–10, groups labeled 32 (32/Cort, 32/Veh) received exposure to 32% sucrose solution, whereas groups labeled 4 (4/Cort, 4/Veh) were exposed to 4% sucrose solution. On Trials 11–15, all groups were exposed to the 4% solution. Rats received an injection of corticosterone (Cort) or vehicle (Veh) after Trial 11. Means and standard errors are plotted.

Figure 1 also shows the results of the five postshift trials. A typical cSNC effect was observed in the two groups that received a vehicle injection after Trial 11, with complete recovery of consummatory behavior by Trial 13. The two groups that received corticosterone treatment after Trial 11 also showed cSNC, but recovery was slower than in the vehicle controls. Corticosterone administration did not affect performance in the unshifted condition (compare groups 4/cort and 4/veh), but it had a more noticeable suppressive effect on performance in the downshifted conditions (compare groups 32/cort and 32/veh).

A Contrast $\times$ Drug $\times$ Trial (11–15) ANOVA indicated significant effects for all double interactions and main effects. Thus, there were Contrast $\times$ Trial, $F(4, 176) = 12.77$, $p < .001$; Drug $\times$ Trial, $F(4, 176) = 2.71$, $p < .04$; and Contrast $\times$ Drug interactions, $F(1, 44) = 4.72$, $p < .04$. The main effects of contrast, $F(1, 44) = 11.45$, $p < .001$; drug, $F(1, 44) = 5.53$, $p < .03$; and trial, $F(4, 176) = 11.88$, $p < .001$, were all significant. Only the triple interaction was nonsignificant, $F(4, 176) = 1.67$, $p > .15$. To determine the source of these interactions, separate Drug $\times$ Trial analyses were computed for the two shifted and for the two unshifted groups. Group 32/cort was significantly more suppressed than group 32/veh, $F(1, 22) = 7.56$, $p < .02$, and the recovery across trials for both groups was significant, $F(4, 88) = 19.88$, $p < .001$. A significant Group $\times$ Trial interaction indicates that the recovery was faster for group 32/veh than for group 32/cort, $F(4, 88) = 3.04$, $p < .03$. For the two unshifted groups, however, none of the factors or their interaction reached significant levels (Fs $<$ 1). Pairwise LSD tests computed on the scores of Trials 11 and 12, the postshift trials administered the day before and after the corticosterone treatment, indicated that cSNC was still observable in both the vehicle controls and corticosterone groups, in both trials (ps $< .04$). However, whereas groups 32/cort and 32/veh were not significantly different on Trial 11 ($p > .80$), group 32/cort exhibited significantly more suppression than group 32/veh on Trial 12 ($p < .025$).

A single post-Trial 11 injection of corticosterone after the first encounter with the downshifted solution lengthened the cSNC relative to the vehicle controls. Corticosterone, however, had no detectable effect on the consummatory behavior of the unshifted 4% group, confirming that its effect is contingent on the experience of a reward downshift and not just on drinking behavior, on sucrose consumption, or on some other contextual factor. Because corticosterone was administered after Trial 11, and almost 1 day before Trial 12, this also eliminates potential alternative accounts based on a direct action of corticosterone on some contextual factor.

Experiment 2

If the effect of corticosterone described in Experiment 1 depends on the experience of reward downshift, then the effect should occur only when corticosterone is administered immediately after Trial 11. Experiment 2 compared immediate corticosterone treatment with a group receiving corticosterone 3 hr after the

end of Trial 11. It was expected that the enhancing effect of corticosterone on the suppression of consummatory behavior after a reward downshift would be present when corticosterone was injected immediately but would be attenuated or absent when injected 3 hr after the trial. An interval of 3 hr was selected because Hui et al. (2004) demonstrated the time-dependent properties of corticosterone on fear conditioning using a similar interval. This prediction is based on the assumption that the state of primary frustration induced by reward downshift and recruited during Trial 11 would tend to decay in time. Experiments on the immediate consequences of reward omission in instrumental situations confirm that the increase in responding decays over time (Stout, Boughner, & Papini, 2003). Thus, if corticosterone is enhancing the internal state of primary frustration induced in the course of Trial 11, it should be ineffective if administered after this state has completely decayed, as is expected to be the case after 3 hr.

This experiment also allows for a test of the nonspecific effects of corticosterone on consummatory behavior because the group injected 3 hr after Trial 11 receives the same amount of the drug and is exposed to the same reward downshift as the group injected immediately. Unshifted 4 → 4 controls were not included in Experiment 2 for two reasons. First, the goal of this experiment was to compare different degrees of consummatory suppression in groups exposed to different degrees of immediacy between Trial 11 and corticosterone administration. Others have followed similar practice under analogous conditions (e.g., Flaherty, Greenwood, Martin, & Leszczuk, 1998). Second, Experiment 1 provided no evidence that corticosterone had any appreciable effect on the consummatory behavior in the unshifted control conditions.

Method

Subjects and apparatus. The subjects were 38 male naive Wistar rats, about 4 months old at the start of the experiment. The average ad-lib weight of all the rats was 388 g. Other maintenance conditions, daily training times, and the conditioning boxes were as described in Experiment 1.

Procedure. The general procedure was the same as that described for Experiment 1, except for the following. Rats were randomly assigned to one of four groups: 32/veh/imm ($n = 8$), 32/cort/imm ($n = 10$), 32/cort/3 ($n = 9$), and 32/veh/3 ($n = 9$). Thus, all groups received training under the same procedure used for the groups exposed to a 32 → 4 downshift in Experiment 1. Owing to an error, one rat that should have been assigned to group 32/veh/imm was instead assigned to group 32/cort/imm, thus causing a deviation from equal sample sizes. Drug preparation, dose, administration procedure, behavioral training, preparation of sucrose solutions, and the recording of the dependent measure were all as described in Experiment 1. Corticosterone was purchased from Sigma-Aldrich (St. Louis, MO).

Results and Discussion

Figure 2 shows the performance of the four groups across all trials of this experiment. No differences were observed among the groups during the preshift trials; notice that all of the groups received exposure to the 32% sucrose solution during Trials 1–10. A Drug (corticosterone, vehicle) × Interval (immediate, 3 hr) × Trial (1–10) analysis indicated only a significant acquisition effect, $F(9, 288) = 34.28$, $p < .001$. All other effects were nonsignificant (Fs < 1.56, ps $> .12$). As also shown in Figure 2, the administration of corticosterone immediately after Trial 11 retarded recovery from reward downshift. However, this retardation was not

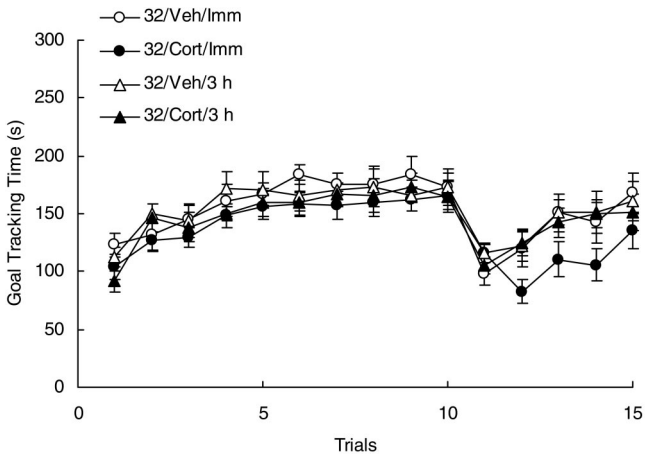

Figure 2. Consummatory performance of groups across trials. All the groups received exposure to 32% sucrose solution on Trials 1–10 and were shifted to 4% solution on Trials 11–15. Groups differed in the treatment received after Trial 11, either corticosterone (Cort) or vehicle (Veh) injections, and either immediately (Imm) at the end of Trial 11 or after 3 hr. Means and standard errors are plotted.

observed when corticosterone was administered 3 hr after the end of Trial 11. A Drug × Interval × Trial (11–15) analysis indicated a significant triple interaction, $F(4, 128) = 2.71$, $p < .04$. Also significant was the recovery effect across trials, $F(4, 128) = 23.18$, $p < .001$. All other factors and interactions were nonsignificant (Fs < 1.73, ps $> .14$).

The triple interaction was further analyzed by computing Group × Trial ANOVAs for each pair of groups injected immediately or after 3 hr. For groups 32/veh/imm and 32/cort/imm, there was a significant Group × Trial effect, $F(4, 64) = 4.01$, $p < .007$, and a significant recovery effect, $F(4, 64) = 11.97$, $p < .001$, but a nonsignificant group effect, $F(1, 16) = 1.58$, $p > .20$. The significant interaction reflects the increase in consummatory suppression observed after post-Trial 11 corticosterone administration. This effect disappeared entirely when the corticosterone treatment was administered 3 hr after the end of Trial 11. A comparison of groups 32/veh/3 and 32/cort/3 yielded a significant recovery across trials, $F(4, 64) = 12.52$, $p < .001$, but nonsignificant group or Group × Trial effects (Fs < 1). Further pairwise LSD tests were computed on the scores of Trials 11 and 12, the postshift trials administered the day before and after the corticosterone treatment. These tests indicated nonsignificant differences among the groups on Trial 11 but significantly more suppression in group 32/cort/imm than in group 32/cort/3 on Trial 12 (ps $< .05$).

General Discussion

Corticosterone administration immediately after the first postshift trial enhanced cSNC relative to vehicle controls. Corticosterone had no detectable effect when administered 3 hr after the first postshift trial or when administered to rats exposed only to the 4% solution. These results suggest that the action of corticosterone on cSNC cannot be attributed to effects of this stress hormone on some contextual aspect controlling consummatory behavior, such as changes in taste perception, motivational effects, and motor

effects. The time-dependent nature of this effect also suggests that corticosterone acts on some internal state induced by the events of Trial 11, namely, the downshift from the expected 32% sucrose solution to the actual 4% solution encountered by the rats on this trial. This feature fits well with the decaying time course postulated for primary frustration (Stout et al., 2003).

It was argued in the introduction that the effect of corticosterone observed in these experiments would provide support for the frustration hypothesis of cSNC (Papini, 2003). According to this hypothesis, the detection of a change in the concentration of the solution at the start of Trial 11 induces an internal state of primary frustration whose properties involve an aversive hedonic state that leads to the rejection of the downshifted solution and to a switch to alternative (search) behaviors (see Wood et al., 2005, Figure 2). Unlike the explanation offered by the multistage model (Flaherty, 1996), the presence of primary frustration introduces an emotional component that has behavioral effects in its own right, and also supports the acquisition of an anticipatory response via Pavlovian pairings with stimuli prevailing in the conditioning situation. It was reasoned, therefore, that the posttrial administration of corticosterone would either increase the intensity of this emotional component, enhance the consolidation of the Pavlovian association leading to secondary frustration, or both, thus leading to extended suppression in subsequent trials.

As mentioned in the introduction, corticosterone administration is usually interpreted as strengthening the consolidation of emotional memories, such as those that are formed during fear conditioning (McGaugh, 2000; Roozendaal, 2000). The overlap between the mechanisms underlying fear, pain, and frustration has been recognized in terms of analogous behavioral, pharmacological, brain lesion, and brain activity patterns (Eisenberger & Lieberman, 2004; Gray, 1987; Papini, 2003; Wood et al., 2005). In the cSNC situation, for example, rats exposed to reward downshift and tested immediately after the second postshift trial in the hot plate test for pain perception exhibit hypoalgesia (Mustaca & Papini, 2005). The present results extend the overlap to the enhancing effects of posttrial corticosterone, which enhances the memory for a frustrative experience just as it does for painful experiences with electric shock in fear conditioning situations (Hui et al., 2004).

These results seem inconsistent with Flaherty's (1996) multistage model, which posits that the rejection of the downshifted sucrose solution during the first postshift trial is part of a cognitive process aiming at searching for the missing substance. Independent evidence suggests that corticosterone has no consistent effects on activity levels, which may be interpreted as inconsistent with the view that the main initial effect of reward downshift is to induce search behavior guided by cognitive processes. For example, repeated corticosterone injections reduced wheel running activity (Isobe, Torii, Kawaguchi, & Nishino, 2004) but had no effects on open field activity (Gregus, Wintink, Davis, & Kalynchuk, 2005), both of which may be considered as involving mild or low levels of emotional arousal. However, glucocorticoids modulate behavioral levels when animals are emotionally aroused. For example, the removal of glucocorticoids by adrenalectomy retards the emergence of freezing behavior in infant rats after they are removed from the nest (Takahashi, 1994), it eliminates the behavioral burst typical of the early stages of appetitive extinction (Thomas & Papini, 2001), and it decreases the frequency of freezing responses during contextual fear conditioning (Pugh, Tremblay, Fleshner, & Rudy, 1997).

The effects of posttrial corticosterone administration on fear conditioning have been generally interpreted as enhancing the consolidation of the memory underlying fear (McGaugh, 2000; Roozendaal, 2000). In the cSNC situation, however, there is a confound pointed out by Wood et al. (2005). When a factor (i.e., a drug) modulates consummatory performance in the first postshift trial, it is difficult to distinguish between two potential effects: first, an action on the intensity of the perceived aversiveness induced by reward loss (i.e., primary frustration) and, second, a facilitatory effect on the establishment of the Pavlovian association leading to an anticipatory form of that aversive response (i.e., secondary frustration; see Wood et al., 2005, Figure 2). In the more typical fear conditioning situation, this confounding would amount to a difference between an enhancing effect on the degree of activation of the unconditioned stimulus representation, versus an enhancing effect on the establishment of the Pavlovian association between the conditioned and unconditioned stimuli (see Delamater, 2004). A resolution of this issue may require testing the effects of corticosterone on situations that reflect more clearly than cSNC the effects of primary frustration (e.g., Stout et al., 2003).

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