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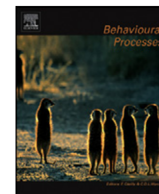
Behavioural Processes 82 (2009) 352–354

Contents lists available at ScienceDirect

Behavioural Processes

journal homepage: www.elsevier.com/locate/behavproc



Short report

Ethanol facilitates consummatory extinction☆

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ARTICLE INFO

Article history:
Received 16 April 2009
Received in revised form 2 July 2009
Accepted 3 August 2009

Keywords:
Ethanol
Consummatory extinction
Consummatory successive negative contrast
Rats

ABSTRACT

Rats given access to an empty sipper tube after having obtained 32% sucrose in the same situation undergo extinction of consummatory behavior (cE). Ethanol (0.75 and 1 g/kg, i.p.) accelerated cE when administered before the second extinction session. The effect was not attributable to increased activity or state-dependent reduction in consummatory behavior. These data are discussed in the context of research on the effects of ethanol on behavioral assays involving incentive downshifts.

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Rats display consummatory suppression after a downshift from 32% to 4% sucrose relative to unshifted controls only given access to 4% sucrose (Vogel et al., 1968). This effect, known as consummatory successive negative contrast (cSNC), is modulated by a variety of drugs, including anxiolytics (Flaherty, 1996) and opioids (Papini et al., 2006). These drug effects suggest that cSNC involves an emotional component derived from the unexpected reduction in incentive magnitude (Crespi, 1942; Elliott, 1928). For example, chlordiazepoxide (Flaherty et al., 1986), diazepam (Mustaca et al., 2000), and ethanol (Becker and Flaherty, 1982) attenuate the cSNC effect in rodents.

SNC has traditionally been linked to appetitive extinction, in which the incentive is downshifted to a zero value, rather than to a small value (Mackintosh, 1974). In the consummatory extinction (cE) assay used here, animals are downshifted from sucrose availability to an empty sipper tube. While little is known about the neurochemical mechanisms underlying cE, Flaherty (1990, p. 317) concluded that "the procedural similarity between extinction and negative contrast is paralleled in the effectiveness of the anxiolytics in moderating both, and the failure of antidepressants to substantially influence either." Consistent with this conclusion, consummatory behavior increased during extinction in animals treated with anxiolytics (Bialik et al., 1982; Soubrie et al., 1978). Conversely, consummatory behavior was suppressed during both cSNC and cE by the nonselective opioid receptor antagonist naloxone (Norris et al., 2008; Pellegrini et al., 2005).

Despite these agreements, available behavioral evidence suggests that cSNC and cE may not be based on the same mechanisms. For example, spontaneous recovery is readily observed in cE (Mustaca et al., 2002), but not in cSNC (Norris et al., 2008). Moreover, extinction after training with a large sucrose concentration is slower than after training with a small concentration (Mustaca et al., 2002), just the opposite of cSNC. This experiment provides evidence that the effects of ethanol on cE are different from those described for cSNC (Flaherty, 1996).

1. Method

1.1. Subjects

24 male, naïve Wistar rats, approximately 3 months old at the start of the experiment served as subjects. Ad libitum weights varied from 362 g to 513 g. Rats were transferred to individual cages with free water 10 days before the start of the experiment and deprived to an 85% of their free-food weight. The housing room was set to a light–dark cycle of 12 h (lights on at 07:00 h) and to constant temperature (23 °C).

1.2. Apparatus

Three conditioning boxes (MED Associates, St. Albans, VT), 30 cm long, 24 cm wide, and 24 cm high, were used. The floor of each box was made of aluminum bars 0.4 cm in diameter and separated by 1.1 cm. In the front wall, made of aluminum, was a 3.5-cm deep

☆ This research was partially supported by grants from Agencia de Promoción y Desarrollo, CONICET, and UBACYT, Argentina.

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doi:10.1016/j.beproc.2009.08.001

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cavity, measuring 5 cm × 5 cm × 3.5 cm (length × width × depth), and located 1 cm above the grid floor. A sipper tube attached to a drinking bottle was inserted through a hole into the back of this cavity. When fully inserted, the sipper tube protruded 2 cm inside the cavity. Rats drank by inserting their snout into the cavity. Head insertion was detected by a photocell located in front of the sipper tube. The dependent variable was the cumulative time of photocell activation (in 0.01-s units, called goal-tracking time). Goal-tracking time is significantly and positively correlated with amount of fluid intake (Mustaca et al., 2002). Each conditioning box had a house light (GE 1820) and was located inside a sound-attenuating cubicle. White noise and a fan provided masking noise and ventilation. The 32% sucrose solution was prepared (w/v) by adding 500 ml of tap water for every 160 g of commercial sugar. Animals were videotaped on Session 12 (Sony camera).

1.3. Procedure

Rats were preexposed to 32% sucrose in the home cage during a single 40-min session. The experiment lasted 14 sessions, one per day. On Sessions 1–10 (acquisition), rats received access to 32% sucrose. The first photocell activation initiated the 5-min long session. At the end of each session, the animal was placed back in its cage and the floor and walls of the box were wiped with a wet cloth. On Sessions 11–13 (extinction), animals were treated as before, except that the sipper tube was empty. At the end of the first extinction session, triplets matched in goal-tracking time for Sessions 8–11 were established. Individuals from each triplet were randomly assigned to one of three groups ($n = 8$) differing in the drug administered 10 min before the second extinction session: 0 (equal-volume saline injection), 0.75, or 1 g/kg ethanol (doses from Becker and Flaherty, 1982). Ethanol was diluted to a 15% solution (w/v) and injected i.p. Session 13 was similar to Session 11 (i.e., no drugs were administered). On Session 14 (reacquisition), rats received the same drug treatments as before Session 12, but they were reexposed to the 32% sucrose to test for state dependency.

Session 12 was videotaped and 5 behaviors were scored: *sipper* (snout inserted in the sipper-tube cavity), *activity* (movement of at least one leg while walking), *rearing* (standing on the rear legs), *grooming* (licking the forepaws or the flanks), and *motionless* (absence of locomotion). An observer scored each behavior using a one-zero sampling every 10 s (Pellegrini and Mustaca, 2000). A second observer scored 70% of the sessions. Interobserver agreement for each behavior was greater than 90%.

2. Results

Goal-tracking times increased gradually during acquisition, from a mean (±SEM) of 106.7 s (±5.7) on Session 1 to 191.9 s (±5.5) on Session 10. A Drug × Session (1–10) analysis indicated a significant acquisition effect, $F(9, 189) = 33.36$, $p < 0.001$; other effects were nonsignificant, $Fs < 1$. Groups were similar during the last acquisition session (Fig. 1A) and first extinction session, $Fs < 1$ (Fig. 1B).

Both ethanol doses reduced consummatory responding on Session 12, but the effect dissipated on Session 13 (Fig. 1B). This was reflected in a significant drug by Session (11–13) interaction, $F(4, 42) = 3.19$, $p < 0.03$. Extinction was also significant, $F(2, 42) = 16.80$, $p < 0.001$, but the main effect of ethanol was not, $F < 1$. Separate analyses indicated nonsignificant group effects on Sessions 11 and 13, $Fs < 1$, but a significant group difference on Session 12, $F(2, 21) = 3.70$, $p < 0.05$. Post hoc LSD comparisons indicated that Group Sal scored significantly higher than Group 0.75 ($p < 0.04$) and Group 1 ($p < 0.01$). When rats were given again access to 32% sucrose on Session 14, goal-tracking scores were similar to those in saline controls (Fig. 1A). There was a marginal group difference, $F(2, 21) = 3.43$, $p < 0.051$, but post hoc LSD comparisons revealed that Group Sal scored significantly below Group 0.75 ($p < 0.01$).

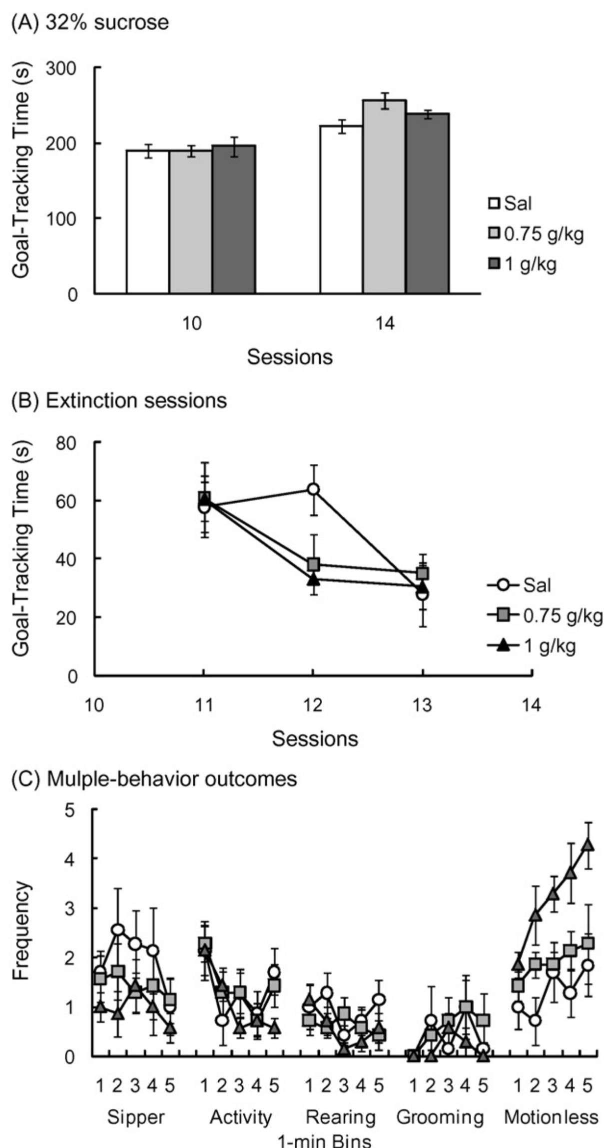

Fig. 1. (A) Goal-tracking times for each of the groups in the last acquisition session (Session 10) and the postextinction upshift session (Session 14). (B) Goal-tracking times for each group during the three extinction sessions. (C) Behaviors recorded on Session 12 after drug treatments. Sal: saline control. Means (±SEMs).

Problems with video recordings affected one subject in each group, so the multiple-behavior sampling analyses are based on an $n = 7$ (Fig. 1C). A Drug × 1-min Bin analysis was calculated for each behavior. The only significant group difference was observed in motionless behavior, $F(2, 18) = 12.01$, $p < 0.001$. The increase across bins was also significant, $F(4, 72) = 4.23$, $p < 0.005$, but the interaction was not, $F < 1$. Post hoc LSD tests indicated that Group 1 scored significantly above Groups Sal and 0.75 ($p < 0.005$), which did not differ from each other ($p > 0.14$). Other group effects were nonsignificant, $Fs < 1.84$, $ps > 0.13$, except for a significant decrease in activity across 1-min bins, $F(4, 72) = 5.84$, $p < 0.001$.

3. Discussion

Ethanol suppressed consummatory behavior during extinction in rats. Some explanations can be ruled out by the data. First, there was no evidence of biased group assignment. Second,

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state-dependency could explain the reduction in consummatory behavior during Session 12, when rats were treated with ethanol. Ethanol supports state-dependent learning (Nakagawa and Iwasaki, 1995). However, ethanol treatment should have also reduced consummatory performance when animals were upshifted to 32% sucrose on Session 14. Instead, treated and saline rats were not different. Third, activity was not a likely factor; in fact, ethanol-treated rats showed a dose-dependent increased in motionless behavior during Session 12. Although this may suggest ethanol-induced motor and/or motivational interference, the upshift data contradict these alternatives. Fourth, the extinction performance of the saline group is also strikingly similar to that found in previous studies on cE (Mustaca et al., 2002), suggesting that the effects of ethanol observed in Session 12 were not an artifact of an unusually high goal-tracking time in the saline controls.

These results add to a growing body of evidence suggesting that complete (cE) and incomplete (cSNC) incentive downshifts engage different mechanisms (Mustaca et al., 2002; Norris et al., 2008). Ethanol's anxiolytic effects on cSNC (Flaherty, 1990) led to the expectation that cE would be retarded, but the opposite effect was found here. Other anxiolytics (e.g., chlordiazepoxide) facilitate instrumental extinction of lever pressing in rats (Williams et al., 1990), thus suggesting that cE resembles instrumental extinction more than cSNC.

Three theoretical possibilities must be considered. First, cE may involve an emotional response not attenuated by ethanol. Second, the effects of ethanol on cE may be unrelated to the aversive emotional responses induced by the downshift. Finally, a more complex, but still non-emotional explanation of these results would suggest that ethanol facilitated extinction on Session 12 because of state dependency, motor interference, or a reduction in motivation, but these effects were counteracted by the positive emotional response induced by the incentive upshift on Session 14. These possibilities await further experimental examination.

Acknowledgments

The authors thank the members of the Laboratory for Experimental and Applied Psychology, J.N. Norris, and L.A. Ortega for helpful comments.

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