Bulletin of the Psychonomic Society
1982, 20(5), 263-265

Mutually antagonistic effects on behavioral variability of ethanol and an aversive CS+

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Serial dependency of components of the rat's water-reinforced operant responding was observed after acute intraperitoneal ethanol injections (1.0 g/kg) and after an aversive CS+ tone. It was found that either the aversive CS+ or ethanol injections decreased behavioral variability, but there was no additional decrease in uncertainty of responding in a combined treatment group. The results were interpreted as support for Devenport and Merriman's theory of the behavioral effects of alcohol and discussed in terms of possible control mechanisms involved.

There is accumulating evidence that the behavioral effects of ethanol include a diminution of behavioral variability (BV). Alcohol has been shown to reduce spontaneous alternation behavior (Cox, 1970), to reduce errors in simple alternation (Crow, 1982), to stabilize operant responding (Crow, McWilliams, & Ley, 1979), to decrease the frequency of deviations from goal-directed activity in a straight alley (Devenport, Devenport, & Holloway, 1981a), and to diminish the number of arms chosen, the sequences of visitation, and the deviations from goal-directed activity in the radial arm maze (Devenport & Merriman, in press). This alcohol-associated BV reduction is believed to occur independently of direct depression or stimulation effects (Devenport & Merriman, in press), and the isolation of possible mechanisms involved arises as a paramount issue.

One strategy of uncovering these underlying processes is a comparison of the experimental manipulations that appear to result in diminished BV and to see which, if any, of these variables interact. Among experimental procedures that appear to alter BV are frontal cortical lesions (Crow & McWilliams, 1979; Pribram, 1960) and hippocampal lesions (Devenport, Devenport, & Holloway, 1981b; Kimble & Kimble, 1970). The latter reduction in BV as a result of hippocampal lesions was not found to be affected by administration of alcohol (Devenport et al., 1981a).

It is possible that alcohol exerts its effects on BV via mechanisms apart from inhibition and/or excitation of specific central regions that are common to many other experimental effects and represent some general stress, "BV shutdown," or "systems monopolization" response. In order to explore this possibility using the interaction strategy, the present study was designed to assess the effects on BV of a general aversive CS+ (Rescorla & Solomon, 1967) on operant responding and to note the influences of alcohol upon these effects.

METHOD

Subjects

Twenty-four male albino rats from the Western colony were used. The animals ranged in weight from 277 to 441 g.

Apparatus

The operant chambers were Gerbrands water-delivery boxes programmed on a continuous reinforcement schedule. A Grason-Stadler module was used for the aversive conditioning with a 1-mA footshock and a tone from a Sonalert Audible Signal (P. R. Mallory Company).

Alcohol Dosages

Alcohol was administered intraperitoneally via a 10% (w/v) aqueous solution of 95% ethanol in an amount that was 1.0 g/kg. Control injections were isotonic saline.

Procedure

Animals were adapted to a 23.5-h water-deprivation schedule; they were then trained and maintained on a 30-min daily operant session of water reinforcement. On pretest day, animals were given the aversive CS+ conditioning training, which consisted of a 1.5-sec tone followed by a 1-sec shock. Pairings of tone and shock were given every 15 sec for a total of 10 min. Control animals received the tone alone.

On test day, animals were injected with the appropriate solution 10 min prior to the time of the daily run and subjected to the tone at 15-sec intervals (without shock) for the 30-min operant session. Measures were made of barpressing and approach-to-dipper response sequences and total number of responses per 30 min.

RESULTS AND DISCUSSION

The basic design was a 2 by 2 factorial of alcohol or saline treatment vs. tone paired with shock or no shock. ANOVAs were carried out both for uncertainty scores (Frick & Miller, 1951; Crow et al., 1979) and for total number of responses on test day.

For the number-of-responses data, there was no significant between-groups effect [$F(3,20) = .222$]. For

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the uncertainty data, there was no significant alcohol main effect [$F(1,20) = 1.62$], no significant aversive CS+ effect [$F(1,20) = 3.58$], but a significant Alcohol by Aversive CS+ interaction [$F(1,20) = 17.85$]. Follow-up comparisons between the groups yielded significant differences between the no-alcohol, no-shock (control) group and the alcohol, no-shock group [$t(10) = 4.72$], between the control group and the shock, no-alcohol group [$t(10) = 5.47$], and between the control group and the combined alcohol, shock group [$t(10) = 2.29$]. No other single-group comparisons were significant at the 5% level.

The mean uncertainty scores are presented in Figure 1. It can be seen that both the aversive CS+ condition and the alcohol treatment reduced the uncertainty of the response sequences (barpressing and approach to the dipper) and that the combined treatment conditions did not result in a compensatory reduction in uncertainty but, rather, showed a tendency (albeit nonsignificant) toward an increase (reduced stereotypy).

Figure 2 depicts the stereotypy scores (1 = uncertainty) for each group with respect to $p_A(A)$ and $p_B(B)$, where $p_A(A) = p(AA)/p(A)$ from Frick and Miller (1951). Although the alcohol-treated group differed from that noted earlier, especially in terms of $p_A(A)$, the present study used an acute 1.0-g/kg dosage whereas the earlier study (Crow et al., 1979) involved chronic 1.5-g/kg doses. Thus alcohol tolerance may increase BV, possibly by enhancing $p_A(A)$.

Most remarkable about Figure 2 is the close clustering of all three experimental groups beyond the isostereotypic contour of .6. In terms of the stereotypy measure, there appears to be no difference between the alcohol-treated group and the group receiving the aversive conditioned stimulus, and, further, the combination of these treatments appears to produce no additional effect either qualitatively (the isostereotypic space) or quantitatively (the value of uncertainty). It appears possible that alcohol produces, aside from its more direct effects, a "stress" not totally unlike that of the aversive CS and that this common response may be characterized in part by a kind of "systems monopoly" in which superfluous acts are minimized.

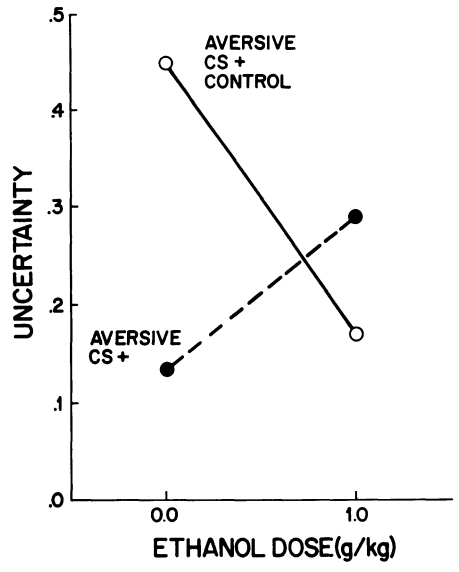

Figure 1. Mean uncertainty scores for each of the four conditions. Each point represents six animals.

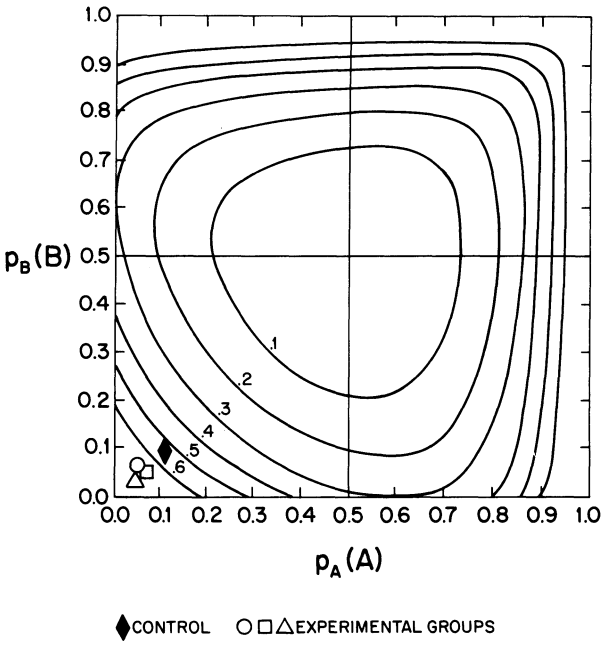

Figure 2. Mean stereotypic resting point (Frick & Miller, 1951) for each of the four groups.

The evidence on alcohol's capacity to reduce BV now appears compelling, but a continuing delineation of those behavioral changes to be considered as reduced variability is needed. For example, response location variability (Antonitis, 1951) shows a puzzling inertness to drug effects (Crow, 1982; Moerschbaecher, Thompson, & Thomas, 1979) that may be more a reflection of our misunderstanding of different classes of BV than of the indirect drug effects upon behavior.

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(Received for publication October 8, 1982.)