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Hungry, drunk, and not real mad: The effects of alcohol injections on aggressive responding

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Two studies were conducted to assess the effects of acute, low-dose ethanol injections on single-subject shock-elicited aggression. Ethanol mixtures of 30% and 60% were used in the first study, and 0% and 30% ethanol mixtures were used in conjunction with 48-h periods of fasting or nonfasting in the second study. The results of the first study, while nonsignificant, indicated that a possible depression of aggressive responding was produced by the low-dose injections. A significant decrease in aggressive responses following low-dose injections was shown in the second study.

Several investigators have reported the effects of alcohol (ethanol) on aggression in animals. However, the findings have been quite inconsistent. Elimination of aggression was reported in a study of domestic cocks (Kovach, 1967). Similarly, Bertilson, Mead, Morgret, and Dengerink (1977) reported that the principal effect of alcohol on white mice seemed to be sedation and that alcohol tended to inhibit aggressive responding. However, Raynes and Ryback (1970) reported that an alcohol solution in the tank of Siamese fighting fish (*Beta splendens*) resulted in a significant increase in fighting response. Also, Weitz (1974) has reported that lower dose injections of ethanol increased fighting in rats in the paired-animal shock-elicited aggression situation.

It should be noted that the procedure used by Weitz (1974), while sound, failed to take into account several factors that have been found to affect the rate of responding in the shock-elicited aggression situation. Weitz (1974) employed a time-series, counterbalanced experimental design. In such a design, each subject is administered all treatment levels. Thus, in the Weitz (1974) study, each subject was tested in the shock chamber a minimum of eight times. Several studies (e.g., Creer & Powell, 1971; Powell & Creer, 1969; Powell, Silverman, & Schneiderman, 1970) have reported that prior fighting in response to shock increases the later fighting probability of pairs of rats. Additionally, Weitz (1974) indicated that shock intensity was adjusted until fighting responses were elicited from each pair of animals. The reported range of shock intensity utilized ranged from 2 mA to 6 mA. On the other hand, the shock-elicited aggression literature indicates that shock intensity level can significantly affect the rate or amount of aggressive fighting that is expressed (e.g., Creer & Powell, 1971; Dreyer & Church, 1968; Follick & Knutson, 1974; Ulrich & Azrin, 1962). In light of these uncontrolled variables, one is left to speculate on the actual differential effects of alcohol on aggressive responding. The present investigations were designed to further investigate the effects of ethanol on aggressive responding in the single-animal shock-elicited aggression situation.

EXPERIMENT 1

Method

Subjects. Fifteen male albino rats obtained from the Holtzman Company, Madison, Wisconsin, served as subjects. The animals were approximately 180 days old at the time of testing. All subjects were individually housed, with food and water freely available until 12 h prior to testing.

Apparatus. A rat-restraining device similar to that described by Azrin, Rubin, and Hutchinson (1968) served as the apparatus used in shock-elicited aggression testing. This apparatus consisted of an opaque plastic tube, measuring 21.5 cm in length and 7.5 cm in diameter, mounted on a Plexiglas sheet. The Plexiglas sheet was, in turn, stabilized on a wooden platform to facilitate placement of the subject into the tube and to permit easy removal of fecal material and urine that accumulated in the tube during testing. A 1.4-cm hole at the enclosed end of the table allowed the subject's tail to be extended from the apparatus and secured to a wood restraining rod by means of adhesive tape. The other end of the tube was open. Two pieces of Number 14 copper wire, permanently attached to the rod 7 cm apart, served as tail electrodes. Thus, when the rod was in place, it served as both a restraining device to prohibit unauthorized escape from the apparatus and an electrode carrier. A BRS/LVE shock generator (SG-905) provided a 1.5-mA shock source. Two BRS/LVE timers (TI-904/252-50) were employed to regulate the frequency and duration of shock delivery.

The aggression target was a LaFayette Instrument omnidirectional lever (Model 80111). This lever was mounted on the wood platform, perpendicular to the open end of the restraining tube and parallel to the wood platform on which the tube was mounted. When the tube was in place on the platform, the lever extended across the open midportion of the end of the tube. The lever was 1.5 cm from the tube and required a movement of 1 cm to activate the microswitch. Closure of the microswitch,

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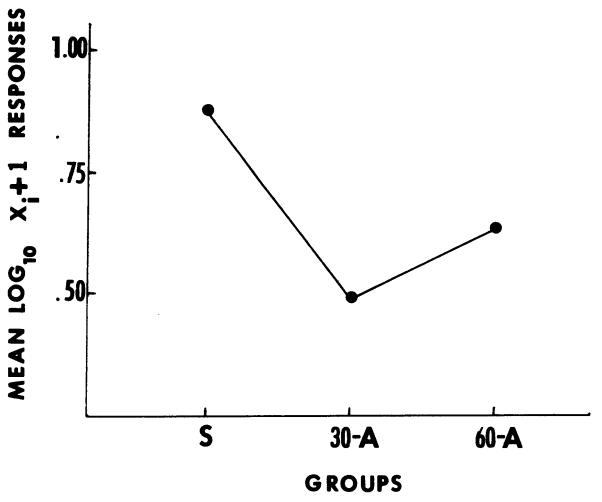

Figure 1. Group mean aggressive responses, Experiment 1.

in turn, activated a LaFayette (Model 5707PS) impulse counter.

Procedure. At the beginning of the experiment, the subjects were randomly assigned to one of three equal groups: Group S (control) received an isotonic saline solution injection; Group 30-A received an injection of an alcohol-saline mixture consisting of 30% ethanol (v/v); Group 60-A received an injection of an alcohol-saline mixture consisting of 60% ethanol (v/v). Prior to testing, all animals were weighed, and injection levels were determined by the ratio of .25 cc/100 g of body weight. In order to establish uniformly stabilized metabolic levels, all food was removed 12 h prior to testing. Appropriate intraperitoneal injections were administered 15 min prior to testing in order to provide consistency with the Weitz (1974) report. The injection schedule was staggered so that each subject had an individual testing session. The order for injecting, and hence the order for testing, was determined by a table of random numbers.

An individual testing session consisted of securing the designated subject in the restraining tube 15 min after injection. Following a 5-min habituation period, the subject received 10 min of shock administration. During this time, 200 1.5-mA shocks of 300 msec duration were administered at 3-sec intervals. The number of responses made by each subject during the 10-min test session was recorded.

Results and Discussion

The response data were transformed into $\log_{10}(X_i + 1)$ scores prior to analysis of variance. Group means are shown in Figure 1. The analysis of variance yielded nonsignificant differences between groups [$F(2,12) = 1.358$, $p > .25$]. While nonsignificant, Group 30-A expressed rate of aggressive responding to shock presentation. The results of the present study fail to lend support to Weitz's (1974) conclusions regarding increased aggression as a function of alcohol injections. As previously suggested, it may well be that Weitz's results were a function of the particular procedural methodology employed. In any event, it would appear that the specific parameters relating ethanol injections and shock-elicited aggression are far from completely understood.

EXPERIMENT 2

Experiment 2 was prompted by the unresolved questions raised by Experiment 1. In a recent investigation (Tramill, Turner, Harwell, & Davis, Note 1), it was found that small, acute doses of alcohol were followed by moderate reductions of blood sugar in the fasted rat. Also, while only tentative at present, pilot research in our laboratory has also indicated that the complete development of behavioral and physiological effects of alcohol may take longer than the 15-min interval between injection and testing that was employed in the Weitz (1974) study and Experiment 1. Additionally, several investigators have reported the existence of a negative relationship between blood-sugar levels and aggressive responding (Davis, Cronin, Meriwether, Neideffer, & Travis-Neideffer, 1978; Davis, Gussetto, Tramill, Neideffer, & Travis-Neideffer, 1978; Neideffer, Travis, Davis, Voorhees, & Prytula, 1977). However, moderate reductions of blood-sugar levels (via insulin injections) have not resulted in an increase of aggressive responding (Neideffer et al., 1977). Thus, one might well predict that an increase in aggressive responding will not follow an acute challenge of ethanol in the fasted rat. One might further predict, based on the results of Experiment 1, that acute challenges of small doses of alcohol will result in depressed rates of responding in the nonfasted animal when increased intervals between injection and testing are employed. The present study was designed to investigate these predictions.

Method

Subjects. Twenty-eight male Sprague-Dawley rats served as subjects. The subjects were approximately 180 days old and were housed as in Experiment 1.

Apparatus. The shock-elicited aggression apparatus employed in Experiment 1 was used in Experiment 2.

Procedure. At the beginning of the experiment, the subjects were randomly assigned to one of four equal groups ($n = 7$); Group NF-S (nonfast, saline), Group NF-A (nonfast, alcohol), Group 48-S (48-h fast, saline), and Group 48-A (48-h fast, alcohol). Groups NF-S and NF-A had food freely available until time of injection; Groups 48-S and Groups 48-A had all food removed from their cages 48 h prior to injection. Prior to intraperitoneal injections, all animals were weighed, and subsequent injection amounts were determined by the ratio of .25 cc solution/100 g of body weight. Groups NF-S and 48-S received injections of an isotonic saline solution 2 h prior to testing. Groups NF-A and 48-A received injections of a saline-alcohol mixture containing 30% ethanol (v/v) 2 h prior to testing. The order of subject injection, and hence the order for testing, was random. Testing procedures were identical to those used in Experiment 1, with the exception of time interval between injection and testing.

Results and Discussion

Prior to factorial analysis of variance, the response data were converted to $\log_{10}(X_i + 1)$ scores. Figure 2 shows group mean responses for Experiment 2. Subsequent comparisons of significant effects were performed using t tests for mean comparisons.

The effect of alcohol on aggressive responding was found to be significant [$F(1,24) = 7.87$, $p < .01$], with alcohol-injected subjects showing fewer aggressive responses. Group mean comparisons provided support

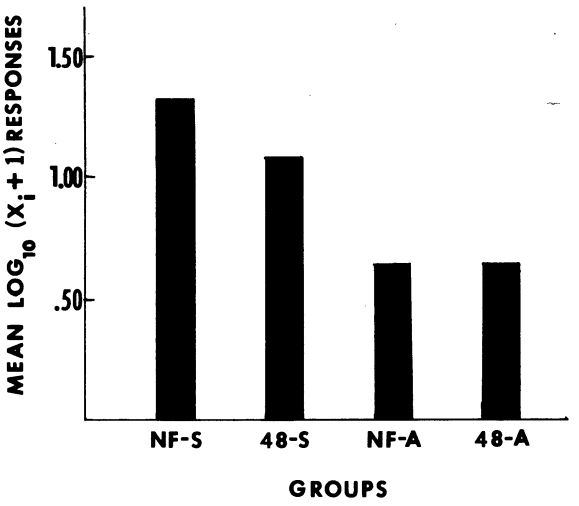

Figure 2. Group mean aggressive responses, Experiment 2.

for the predicted inhibition of aggressive responding as a function of alcoholic challenges in the nonfasted animal when an increased interval of time was used between injection and testing [$t(24) = 2.44$, $p < .05$]. While not significant, this group also expressed a depression in aggressive responding. Nonsignificant results were found for the effects of food deprivation [$F(1,24) = .43$, $p > .50$]. The interaction effect also failed to yield significance [$F(1,24) = .40$, $p > .50$]. The results of Experiment 2 failed to provide support for the proposed relationship between alcohol and increased aggressive responding in the shock-elicited aggression situation as expressed by Weitz (1974). The present results appear to be more in line with the proposed inhibition of aggressive responding as a function of low-dose alcohol challenges that have been reported (Bertilson et al., 1977; Kovach, 1967) and the reported relationship between moderate reductions of blood-sugar levels via insulin injection and aggression (Neideffer et al., 1977).

GENERAL DISCUSSION

Considering the results of both studies, one is tempted to propose an inverse relationship between low-dose alcohol injections and aggressive responding. However, several unanswered questions remain. As various investigators have employed a wide range of animal subjects, and the resultant literature is inconsistent, this proposed relationship may be species specific. Hence, one might fruitfully engage in a series of investigations employing various species in the pain-elicited aggression situation.

A second unanswered question also remains. Namely, is the phenomenon situation specific? The present results may be a function of the procedure employed, that is, the single restrained-animal shock-elicited aggression situation. It is therefore suggested that a replication of the Weitz (1974) paired-animal study be conducted using uniform shock intensities for all pairs and single exposure of each pair of animals to the shock chamber.

A third area for possible fruitful investigation lies in the coincident manipulation of various deprivation states. For example, additional food-deprivation periods, various periods of REM sleep deprivation, and water-deprivation states may well be found to have an interactive effect with alcohol injections.

In summation, the results of the present studies indicate a possible inhibition of aggressive responding as a function of low-dose injections of alcohol solutions. However, only future investigations can resolve certain unanswered questions regarding this proposed relationship.

REFERENCE NOTE

1. Tramill, J. L., Turner, P. E., Harwell, G., & Davis, S. F. *Alcoholic hypoglycemia as a result of acute challenges of ethanol.* Unpublished manuscript, 1979.

REFERENCES

Azrin, N. H., Rubin, H. B., & Hutchinson, R. R. Biting attack by rats in response to aversive shock. *Journal of the Experimental Analysis of Behavior*, 1968, **11**, 633-639.

Bertilson, M. S., Mead, J. D., Morgret, M. K., & Dengerink, H. A. Measurement of mouse squeals for 23 hours as evidence of long term effects of alcohol on aggression in pairs of mice. *Psychological Reports*, 1977, **41**, 247-250.

Creer, T. L., & Powell, D. A. Effects of repeated shock presentations and different stimulus intensities on shock induced aggression. *Psychonomic Science*, 1971, **24**, 133-134.

Davis, S. F., Cronin, E. L., Meriwether, J. A., Neideffer, J., & Travis-Neideffer, M. N. Shock-elicited attack and biting as a function of chronic vs. acute insulin injection. *Bulletin of the Psychonomic Society*, 1978, **12**, 149-151.

Davis, S. F., Gussetto, J. K., Tramill, J. L., Neideffer, J., & Travis-Neideffer, M. N. The effects of extended insulin dosage on target-directed attack and biting elicited by tailshock. *Bulletin of the Psychonomic Society*, 1978, **12**, 80-82.

Dreyer, D. K., & Church, R. M. Shock-induced fighting as a function of the intensity and duration of the aversive stimulus. *Psychonomic Science*, 1968, **10**, 271-272.

Follick, M. H., & Knutson, J. F. Shock source and intensity: Variables in shock-induced fighting. *Behavior Research Methods & Instrumentation*, 1974, **6**, 477-480.

Kovach, J. K. Maternal behavior in the domestic cock under the influence of alcohol. *Science*, 1967, **156**, 835, 837.

Neideffer, J., Travis, M. N., Davis, S. F., Voorhees, J. W., & Prytula, R. E. Sweet and sour rats: The effect of insulin dosage on shock-elicited aggression. *Bulletin of the Psychonomic Society*, 1977, **10**, 311-312.

Powell, D. A., & Creer, T. L. Interaction of developmental and environmental variables in shock-elicited aggression. *Journal of Comparative and Physiological Psychology*, 1969, **69**, 219-225.

Powell, D. A., Silverman, J. F., & Schneiderman, N. E. The effects of sex and prior experience with fighting on shock-induced aggression. *Communications in Behavioral Biology*, 1970, **5**, 51-56.

Raynes, A. E., & Ryback, R. S. Effects of alcohol and congeners on aggressive response in *Beta splendens*. *Quarterly Journal of Studies on Alcohol*, 1970, **5**, 130-133.

Ulrich, A. F., & Azrin, N. H. Reflective fighting in response to aversive stimulation. *Journal of the Experimental Analysis of Behavior*, 1962, **5**, 511-520.

Weitz, M. K. Effects of ethanol on shock-elicited fighting behavior in rats. *Quarterly Journal of Studies on Alcohol*, 1974, **35**, 953-958.

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