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A withdrawal-related reinforcing effect of alcohol

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Rats were induced to consume large quantities of alcohol. Periodically they were withdrawn from alcohol, then tested for alcohol consumption. Across 12-h withdrawal tests, drinking latency progressively declined and consumption remained high. After 120-h, latencies were longer and consumption was low. This suggests that reinforcement via withdrawal discomfort reduction may be involved in maintaining alcohol consumption.

Theories of alcoholism often place considerable emphasis upon addiction as one process supposedly involved in the maintenance of at least some instances of abusive alcohol consumption (e.g., Kissin, 1974; Seevers, 1968). According to this type of theory, when an organism has experienced alcohol in its system in sufficient quantities for a sufficient time, it enters a state typically referred to as addiction, hereafter referred to as the "A state." A further common assumption is that, if alcohol is withheld from an organism that is in the A state, the organism enters a second state, generally called withdrawal (the "W state").

Four assumptions commonly made about the W state are of primary importance to the research described here. (1) The W state is assumed to be transitory. Under conditions of continued unavailability of alcohol, the addicted organism enters the W state, remains in that state for a period of time, and then leaves it. (2) The W state is assumed to be aversive. Its attenuation or elimination is reinforcing. (3) The W state is assumed to be attenuated by reintroduction of alcohol into the organism's system. (4) The W state is assumed to provide the organism with distinctive feedback stimulation that can assume stimulus control over overt behavior.

According to this set of assumptions, then, one reason alcoholics sometimes continue to drink is because not drinking puts them into the W state, where they have previously been differentially reinforced for alcohol consumption by alcohol-induced attenuation of the W state. In other words, the reinforcing value of alcohol is enhanced during the W state, and this enhancement provides a basis for differential reinforcement of alcohol-getting behavior in the stimulus context of withdrawal.

Despite the frequency with which this addiction theory of alcoholism is encountered in the clinical literature, we have been unable to locate any experimental evidence that the reinforcing function of alcohol is, indeed, enhanced in withdrawal or that through this enhancement, the likelihood of alcohol behavior is increased during withdrawal. In fact, even though there is a large literature on how the consumption of or preference for alcohol changes as a function of various histories of exposure to alcohol, few of the published studies are even relevant to this issue for the following reasons.

According to the assumptions of addiction theory just summarized, in conjunction with what is known about the role of reinforcers in the control of behavior, at least four conditions must be met before one would expect to see alcohol behavior based upon this W-state enhancement of the reinforcing function of alcohol. (1) The organism must have a history of alcohol exposure sufficient to produce the A state. (2) The organism must then be put into the W state. (3) While in the W state, the organism must experience the alcohol-produced attenuation of the W state, and (4) the organism must have repeated opportunities to have alcohol behavior reinforced by such W-state attenuation.

We have found no published experiments that unequivocally meet all four of these conditions. Very often it is questionable whether the organism's history of exposure to alcohol was sufficient to produce the A state. Or it is questionable whether subjects were in the W state when reexposed to alcohol. Or subjects are given only one (or very few) exposure to alcohol in the W state, a situation not conducive to much learning on the basis of alcohol-induced W-state reduction, especially given that there is no doubt a substantial temporal delay between alcohol ingestion and W-state attenuation.

The experiment reported here is our first attempt to meet more closely the theoretically necessary conditions for showing a W-enhanced reinforcing effect of alcohol upon alcohol consumption.

METHOD

Subjects and Apparatus

Subjects were three male Sprague-Dawley albino rats approximately 180 days old at the outset. The apparatus consisted

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of three conventional operant conditioning chambers, each housed in its own sound-attenuating enclosure, controlled by electromechanical equipment located in an adjacent room. On one wall of each chamber was a food hopper into which .045-g Noyes food pellets were delivered automatically and a 1-cm-sq Lucite rod that protruded 6.3 cm into the chamber. Downward pressure of the rod activated a microswitch and delivered a small shot of liquid to a conical depression milled into the top surface of the rod, midway between the wall and the end of the protruding rod.

Procedure

Subjects were reduced to 80% of ad-lib body weight. They were then given a series of 1-h preliminary sessions in the chambers, during which they were trained to eat food pellets that were delivered noncontingently to the food hopper and to press the rod to deliver shots of saccharine-flavored water.

Following preliminary training, each subject was kept in its assigned chamber continuously, except for a 5-min period each day to service the chamber and for withdrawal periods (see below). While in the chamber, subjects were continuously exposed to a polydipsia induction schedule (Falk, Samson, & Winger, 1972; Gilbert, 1978) in which, during every 4th h, a food pellet was delivered every 2 min. These pellets constituted the entire daily food ration. During the intervening 3 out of 4 h no food was delivered. At all times, liquid was available by leverpressing at the rate of approximately .10 ml/press. The liquid was initially saccharine- (.25%) flavored water; over days, progressively larger amounts of alcohol were added, up to a final concentration of 10% (W/V).

The amount of liquid subjects received by leverpressing was recorded hourly. Extensive observation of subjects revealed that they immediately drank all the liquid delivered. Thus, alcohol consumption figures cited hereafter were estimated directly from quanitites of liquid delivered.

After the daily intake of alcohol solution had stabilized, we conducted a series of alcohol consumption learning sessions while subjects were in withdrawal. Each learning session was preceded by at least 5 consecutive days of polydipsia-induced alcohol consumption and then a withdrawal period. During the withdrawal periods, subjects were removed to home cages, with water continuously available. During the first two withdrawal periods, subjects were observed on an hourly basis for physical withdrawal symptoms. Since these observations yielded nothing of interest, observation during remaining withdrawal periods was less systematic.

At the end of each withdrawal period, subjects were returned to their chambers where, for 1 h, they were permitted to leverpress for 10% alcohol. No food was delivered during the hour. Amount of alcohol consumed and latency to initiate pressing for alcohol were recorded.

The first six learning sessions followed 12 h of alcohol withdrawal. Then three tests following 120-h withdrawal were conducted. During these longer withdrawal periods, subjects were also kept in home cages, with water continuously available; an amount of food was provided daily, at the beginning of each withdrawal day, equal to that normally received during 24 h on the polydipsia schedule. Then three further 12-h tests, three more 120-h tests, three more 12-h tests, and a final series of three 12-h tests with water substituted for the 10% alcohol solution were given.

RESULTS AND DISCUSSION

Figure 1 shows mean daily alcohol intake, expressed in grams of alcohol per kilogram of body weight, produced by the polydipsia induction procedure throughout the entire experiment. Alcohol intake rose to a maximum of between 18 and 19 g/kg after 30 days, then gradually declined to vary between about 13 and 16 g/kg/day.

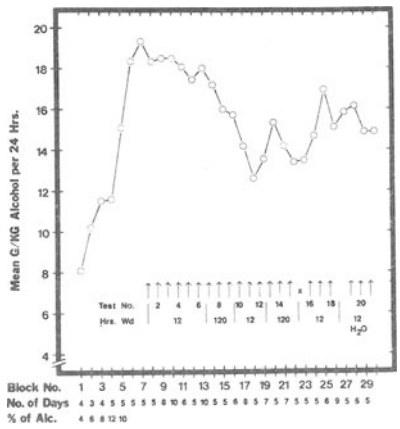

Figure 1. Mean daily alcohol consumption in grams of alcohol per kilogram of body weight produced by the polydipsia induction schedule throughout the experiment. The upward-pointing arrows indicate the points at which withdrawal and subsequent testing were inserted into the alcohol maintenance schedule. The x notes the point after which saccharine was permanently deleted from the alcohol solution. The "No. of Days" scale indicates the number of days over which each plotted point was averaged. During testing this equaled the number of days of alcoholization between successive withdrawals. From the fifth block onward, 10% alcohol solution was used consistently.

The upward pointed arrows indicate the placement in the alcohol-intake maintenance schedule of the several series of withdrawal tests described above. The x indicates the point at which saccharine was deleted from all further alcohol solutions. Saccharine was eliminated at that point to enable us to conduct the final series of tests (with a no-alcohol solution) without the confounding of saccharine water being a very preferred solution.

Two aspects of these daily intake data are noteworthy. First, the amounts of alcohol consumed daily by the subjects was at least as great as any other 24-h alcohol-intake data we have ever seen. Thus, the polydipsia induction schedule was effective in inducing subjects to consume a large quantity of alcohol. Second, removal of the saccharine produced no noticeable decrease in daily alcohol intakes, suggesting that at that relatively late point in the experiment, at least, saccharine played no important role in maintaining liquid consumption.

Figure 2 presents the data from the several series of withdrawal tests. Overall, the results are much more striking in terms of latency to initiate alcohol responding than in terms of amount consumed during the tests.

Over the first series of 12-h withdrawal tests, latency showed a gradual decrease from over 100 sec on the first test to around 20 sec by the sixth test. Consumption volume, however, changed little over the six tests. Switching subjects to 120 h of withdrawal resulted in a

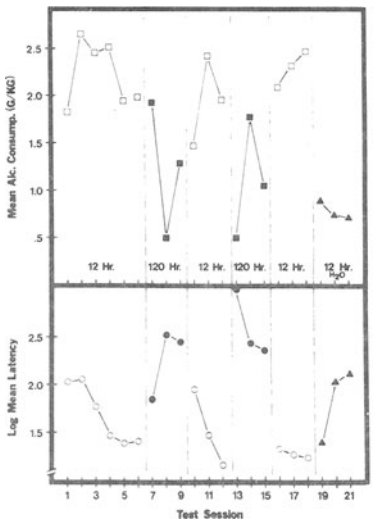

Figure 2. Mean latency (plotted on log coordinate) to initiate drinking and mean amount of liquid consumed in a 1-h test following withdrawal periods of 12 and 120 h, as indicated. The available fluid on all test series except the last was 10% (W/V) alcohol. The available fluid on the last test series was tap water.

very substantial increase in latency and a slight decrease in volume. This pattern was repeated and became more striking over two more series of three 12-h withdrawal tests and one more series of 120-h withdrawal tests. Finally, on the final series of tests, in which subjects were 12-h withdrawn but received water for responding on the test, latency increased and volume consumed decreased substantially.

Overall, then, these data demonstrate that when subjects were 12-h withdrawn from alcohol, alcohol was an effective enough reinforcer to promote acquisition and maintenance of the barpressing response. However, when subjects were 120-h withdrawn, a time presumably long enough to allow subjects to get completely through the W state, the same alcohol solution was not a sufficient reinforcer to maintain the barpressing response at the same high level. Further, the results of the final test with water at 12-h withdrawal indicates that the alcohol solution, and not liquid per se, maintained responding after 12-h withdrawal.

In other words, the reinforcing value of alcohol appears to be potentiated during active withdrawal relative to other times. Further, as suggested in the introduction, the enhancement of alcohol's reinforcing potential in withdrawal requires repeated experience before its impact is observed in the form of strengthened alcohol behavior in withdrawal.

We interpret these data as being generally supportive of the addiction theory assumptions outlined in the introduction.

One further aspect of this experiment deserves mention. Numerous other investigators have reported very obvious physical withdrawal symptoms following alcoholization regimes similar to those employed here, and alcohol intakes actually lower than those we obtained. However, we observed no reactions during withdrawal resembling those that are typically listed as part of the rat abstinence syndrome (e.g., Falk et al., 1972; Majchrowics, 1975; Mello, 1973). Why no such reactions were obtained is unknown. However, it is interesting that we did find the reinforcing value of alcohol to vary appropriately with the point of testing in withdrawal, in spite of the subjects showing no observable withdrawal symptoms.

Addiction theory is usually couched such that withdrawal symptoms are the defining criterion of addiction. The present data suggest, however, that withdrawal-based modulation of alcohol's reinforcing value may very well be the more sensitive indicator of addiction, that is, of a change in an organism's reaction to alcohol that is conducive to further alcohol consumption.

REFERENCES

FALK, J. L., SAMSON, H. H., & WINGER, G. Behavioral maintenance of high concentrations of blood ethanol and physical dependence in the rat. *Science*, 1977, **1972**, 811-813.

GILBERT, R. M. Schedule-induced self-administration of drugs. In D. E. Blackman & D. J. Sanger (Eds.), *Contemporary research in behavioral pharmacology*. New York: Plenum Press, 1978.

KISSIN, B. The pharmacodynamics and natural history of alcoholism. In B. Kissin & H. Bezleiter (Eds.), *The biology of alcoholism. Vol. 3: Clinical pathology*. New York: Plenum Press, 1974.

MAJCHROWICZ, E. Induction of physical dependence upon ethanol and associated behavioral changes in rats. *Psychopharmacologia* (Berlin), 1975, **43**, 245-254.

MELLO, N. K. A review of methods to induce alcohol addiction in animals. *Pharmacology, Biochemistry, and Behavior*, 1973, **1**, 89-101.

SEEVERS, M. H. Pharmacologic elements of drug dependence. *Journal of the American Medical Association*, 1968, **206**, 1263-1266.

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