FEAR REDUCTION AND AVOIDANCE LEARNING FOLLOWING ADMINISTRATION OF ALCOHOL DURING PRIOR CS-SHOCK EXPOSURE

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Abstract

To determine if the classical conditioning of fear was responsible for the facilitative effects of prior exposure to CS-shock pairings, 2 groups of 48 rats were given either 25 light-tone and shock pairings or 25 light-tone presentations only. One-half of the Ss in each of these groups were injected with 1.5 cc/kg of alcohol 5 minutes prior to the pretraining procedure, while the remaining Ss were injected with 1.5cc/kg of physiological saline. 24 hours after initial treatment one-half of the Ss received 90 avoidance conditioning trials under the same drug state as during prior training, while the remaining Ss were tested under the other drug condition.

Results indicated that Ss which received prior CS-shock pairings responded faster and learned the avoidance task more readily than Ss given only prior CS presentations. In addition, Ss which received saline during prior CS-shock exposure responded more rapidly during the first 5 and last 5 trials than did Ss who either received alcohol during prior CS-shock exposure, or Ss who received alcohol or saline and only prior CS presentations. Moreover, Ss whose initial treatment consisted of prior CS-shock exposure with saline made more avoidance responses during the first block of 30 trials, and made the first avoidance response and 3 consecutive avoidance responses earlier than Ss in each of the other groups. These results were taken to indicate that prior CS-shock exposure results in the conditioning of fear to the CS, thereby motivating escape from the CS. Escape from the CS reduces the fear, thus reinforcing the avoidance response.

The present study also indicated that administration of alcohol during avoidance training increased the inter-trial response rate, but decreased the avoidance response rate. These results were interpreted as further support for the hypothesis that fear motivates, and that fear reduction reinforces the avoidance response.

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Chapter 1

Statement of Problem

In the two factor theory of avoidance learning advanced by Mowrer (1960, 1967) and Rescorla and Solomon (1967) it is maintained that components of classical conditioning and instrumental learning are involved in the acquisition of an avoidance response. According to this notion, fear is classically conditioned to the CS during the early trials of avoidance training. During the later trials fear is reduced by the instrumental response of escaping from the CS and hence the avoidance response is reinforced.

Rescorla and Solomon (1967) have derived several predictions from two factor theory including one which states that there should be a facilitation in the acquisition of the avoidance response following exposure to separately conducted Pavlovian procedures employing shock. Several investigators (e.g., Baum, 1969a; DeToledo & Black, 1967; Overmier & Leaf, 1965; Slotnick, 1968) have in fact found that Ss given prior CS-shock pairings (classical conditioning) learned an avoidance response more readily than Ss given no such prior training. The superior performance in Ss given prior CS-shock pairings has been interpreted to be a function of fear being classically conditioned to the CS during prior training. As such, the subsequent avoidance task merely involves the acquisition of the correct instrumental response. If this is in fact what does occur, then it follows that if the fear is reduced or eliminated during prior training, the avoidance task should not be acquired as readily as when no such interference is introduced. The present study is an attempt to retard the acquisition of the avoidance response by using ethyl alcohol to suppress the conditioned fear during prior classical conditioning. Since the present study dealt with both two factor theory and the use of alcohol as a fear depressant, the research on these topics relevant to the present problem are reviewed in the next two sections. The introduction is concluded with a final section in which specific hypotheses tested are stated.

Two Factor Theory

Drive reduction theorists (e.g., Hull, 1952, p.111) have maintained that the motive or drive operating in avoidance learning is produced by the aversive UCS. Accordingly, termination of the UCS serves to reinforce the avoidance response. However, a successful avoidance response precludes the occurence of the UCS, thus eliminating the drive as well as the basis for reinforcement of the avoidance response. As such it is difficult to explain how the avoidance response is maintained (Cofer & Appley, 1965; Herrnstein, 1969) unless an internal drive state, such as that suggested by Mowrer (1960) is postulated.

Mowrer (1960, 1967) has stated that an organic need, such as pain, or the anticipation of an organic need, such as fear, is capable of motivating the organism. Consistent with the Hullian notion of drive reduction, Mowrer, (1960) has postulated that a habit can be acquired when either of these specific factors was reduced. With reference to the avoidance conditioning paradigm, he has postulated that in the early stages of avoidance training the UCS elicits pain and fear. With continued pairings of the CS and UCS, fear becomes a classically conditioned response to the CS and hence serves to motivate escape from that stimulus. Escape from the CS (i.e., an avoidance response) reduces the fear and thereby the avoidance response is reinforced. According to this position then, S learns to escape from the CS by reducing fear, and in doing so he avoids the UCS (Feather, 1963; Rescorla & Solomon, 1967; Solomon & Wynne, 1964). Based on this analysis, successful avoidance learning involves two processes; (a) the acquisition of fear through classical conditioning, and (b) the reduction of pain and fear via an instrumental response.

Consistent with Mowrer's position, other two factor theorists have assumed that any established principle of Pavlovian conditioning should apply to already established or to-be-established instrumental response. For example, it has been argued that if Ss are given prior training in which a CS (e.g., light) is consistently paired with a UCS (e.g., shock), such that fear is conditioned to the CS, then a subsequent avoidance task should involve only the learning of the correct instrumental response. There are however a

number of studies in which it has been found that prior exposure to a CS and inescapable shock interfered with subsequent escape training (e.g., Carlson & Black, 1960; Dinsmoor & Campbell, 1956a, 1956b; Seligman & Maier, 1967) or avoidance training (e.g., Baron, 1959; Overmier, 1968; Overmier & Seligman, 1967; Weiss, Krieckhaus & Conte, 1968).

Several hypotheses have been suggested to account for the results of such studies. For example, one such hypothesis suggests that responses such as rearing or freezing are learned during prior training, and that these responses are incompatible with those necessary for avoidance learning (Brenner & Goesling, 1970; Brown & Jacobs, 1949; Dinsmoor & Campbell, 1956a, 1956b; Kent, Wagner & Gannon, 1960; Mullin & Morgenson, 1963; Weiss et al., 1968). A second hypothesis, proposed by Baron and Antonitis (1961) and supported by Baum (1969b) and Weiss et al., (1968) suggests that prior exposure to CS-shock pairings results in an increased emotional state which is so great as to interfere with avoidance learning. Thirdly, Walters (1963) has suggested that S adapts to the shock during prior training and reacts with decreased responsivity on further encounters with that level of shock. Finally, it has been proposed that the interference may result from the acquisition of "helplessness" during prior exposure to the CS-shock pairings. That is to say, S learns that he cannot escape or avoid the UCS (Overmier, 1968; Overmier & Seligman, 1967; Seligman & Maier, 1967; Smith, Cohen & Turner, 1968).

In contrast to the above mentioned studies, a number of experiments have indicated that preliminary exposure to Pavlovian procedures can facilitate; (a) to-be-established instrumental behavior (Baum, 1969a; Brown & Jacobs, 1949; Brookshire, Littman & Stewart, 1961; Brown, Kalish & Farber, 1951; Carlson & Black, 1960; DeToledo & Black, 1967; Kurtz & Pearl, 1960; Overmier & Leaf, 1965; Slotnick, 1968; Walters, 1963; Zielenski & Soltysik, 1964), (b) already established instrumental behavior (Baum, 1965, 1967, 1969a; Brogden, 1970; Bull & Overmier, 1968a, 1968b; Gilbert, 1970; Grossen & Bolles, 1968; Kamano, 1968; Martin & Reiss, 1969; Overmier & Leaf 1965; Rescorla, 1966, 1967, 1968; Rescorla & LoLordo, 1965; Solomon & Turner, 1962; Weisman & Litner, 1969a, 1969b), and (c) resistance

to extinction of the classically conditioned fear response (Desiderato, 1964; Desiderato, Butler & Meyer, 1966; Kalish, 1954; McAllister & McAllister, 1962a, 1962b, 1963, 1965, 1968).

There appear to be three basic differences between those studies supporting two factor theory and those which do not. Firstly, in the case of Pavlovian procedures influencing already established instrumental behavior, it seems that if Ss were given prior instrumental training, then any interference established through subsequent classical conditioning was negated (Weiss et al., 1968). These authors have suggested that this may be a result of competing responses, such as freezing, not occuring as readily if Ss are given prior instrumental training.

A second factor differentiating those studies which support the two factor position and those which do not, is that in the studies indicating that Pavlovian procedures can interfere with to-be-established instrumental behavior, a substantial delay was introduced between prior CS-shock exposure and subsequent avoidance training. Several hypotheses have been suggested in order to explain why such a delay should facilitate avoidance learning. Firstly, it has been proposed that fear, which is acquired during prior training has an opportunity to incubate during the delay, and subsequently it can energize avoidance responding (Bindra & Cameron, 1953; Kamin, 1957a). Secondly, it has been suggested that the stimulus generalization gradient becames flatter as a result of the delay, and consequently S responds non-differentially to a number of other stimuli, thus increasing the probability that S will make an avoidance response (Desiderato, 1964; Desiderato, Butler & Meyer, 1966; McAllister & McAllister, 1962a, 1962b, 1963, 1965, 1968; Perkins & Weyant, 1958; Thomas & Lopez, 1962). Finally, Baron & Antonitis (1961) have noted that during the delay interval there is an opportunity for the dissipation of an emotional state which ordinarily interferes with performance. The important point however is that prior CS-shock pairings facilitate subsequent avoidance learning if a delay between the two conditioning procedures is employed. Moreover, research by Desiderato et al., (1966) and McAllister and McAllister (1963, 1965) has indicated that the optimum delay interval is 24 hours.

A third factor differentiating those studies supporting the two factor position and those which do not, is that in the latter,

the procedures employed invariably led to the establishment of competing responses. For example, Overmier (1968), Overmier and Seligman (1967), Seligman and Maier (1968) and Smith et al. (1968) all used Pavlovian harnesses during prior exposure to CS-shock pairings. Since S is incapable of moving while in the harness, the probability of the freezing response being conditioned to the CS increases. In fact, studies by Brown and Jacobs (1949) and Weiss et al. (1968) have demonstrated that by decreasing the probability of the freezing response occuring during prior training, the probability of S rapidly acquiring the avoidance response increased.

In summary then, two factor theorists have postulated that avoidance conditioning involves both classical conditioning and instrumental learning. Support for this hypothesis has come mainly from studies which involve two distinct phases; i.e., Pavlovian fear conditioning followed by avoidance training. Presumably, the facilitative effects of such a procedure indicate support for the two factor position. One shortcoming of this approach, however, is that it does not lead unequivocally to the conclusion that the CR's acquired during prior CS-shock exposure motivates the instrumental response. A large body of experimental literature exists which indicates that both humans and lower animals are capable of using general, nonspecific information acquired in one experimental situation in other learning situations (e.g., Harlow, 1949). The net effect of learning to learn in the present situation would be increased proficiency in the acquisition of the avoidance response following the Pavlovian training, a result not different from that predicted by two factor theory, but occuring for an entirely different reason. Specifically, the facilitative effects of prior CS-shock exposure could result from either the association of fear with the CS, or, more generally, as a result of Ss learning to learn. If the responses are acquired specifically and solely as a function of the classical conditioning of fear to the CS, then (a) it should be possible to reduce or eliminate these CR's, and (b) in such a case the facilitative effects of the prior training will be lost. One purpose of the present experiment was an attempt to evaluate these possibilities. The next

section includes the rationale under which the study was conducted.

Alcohol as a fear depressant

One approach to the problem just specified would be to use a drug which will eliminate or reduce the effectiveness of the conditioned fear response, and at the same time will not interfere with the experiences in which learning-to-learn are involved. Alcohol (ethanol), which is classified as a CNS depressant, is one such drug. It has been found to decrease number of avoidances in an avoidance task (Berger, 1969; Kaplan, 1956), while increasing distance run and decreasing latency in an approach-avoidance task (Conger, 1951; Freed, 1967, 1968a, 1968b; Masserman & Yum, 1946). It is believed that this is due to the fact that alcohol acts in reducing fear associated with the experimental situation (Cofer & Appley, 1965; Miller, 1956).

Three strategies have generally been employed in order to ascertain whether or not alcohol reduces fear. The first of these has concerned itself with the effects of alcohol on instrumental avoidance responding. It has been found that Ss treated with alcohol made fewer avoidance responses compared with Ss treated with saline (Berger, 1969; Kaplan, 1956; Pawlowski, Dennenberg & Zarrow, 1961). Moreover, Ss treated with alcohol took longer to react to the CS in a bar press avoidance task (Scarborough, 1957), pole climbing avoidance tasks (Berger, 1969), and in extinction of a shuttle box avoidance task (Baum, 1969b). In addition, Scarborough (1957) noted that Ss treated with alcohol kept the bar depressed for shorter periods of time, and also extinguished a bar press avoidance task faster than Ss given saline. In order to determine whether these results were a function of some deterioration of the retention of the learned response, Scarborough (1957) required Ss previously treated with alcohol or saline to relearn the avoidance response. The results of this experiment indicated that alcohol acted by reducing fear, and in no way interfered with the retention of the learned response.

Contrary evidence has been presented by McMurray and Jacques (1959), who found that 1mg/kg of alcohol did not affect avoidance responding. In addition, Wallgren and Savolainen (1962) found that

alcohol affected latency of escaping from the CS, but not the number of avoidances made. However, in both of these studies the avoidance response had previously been firmly established, and the dosage of alcohol used was relatively small. Consequently, the possibility exists, as both Freed (1968a) and Miller and Barry (1960) have noted, that alcohol is not effective in reducing fear if the fear reaction is too great, or if the avoidance response is too well established. To add some credence to this suggestion, Wallgren and Savolainen (1962) have reported that Battig and Grandjean found that 1mg/kg of alcohol did reduce avoidance responding in rats trained to a criterion of 50% correct responding. Therefore, even in light of this contrary evidence, it seems safe to conclude that alcohol is effective in reducing avoidance responding if the dosage is of a sufficient level, and if the fear reaction is not well established.

A second line of investigation has started from a hypothesis suggested by Miller (1948, 1951), that a reduction in fear can serve as the reinforcement for the learning of new habits. Thus, if consumption of alcohol produces a reduction in fear, then the consummatory response will be reinforced and hence increase in frequency during a subsequent free choice situation. Results in accordance with this hypothesis have been found by a number of investigators (e.g., Adamson & Black, 1959; Brown, 1968; Casey, 1960; Clay, 1964; Freed, 1967; Masserman & Yum, 1946; Powell, Kamano & Martin, 1966; Smart, 1965).

Finally, the third approach to the problem is concerned with the effect of alcohol on behavior in an approach-avoidance situation. It has been shown that Ss placed in a conflict situation after injection of alcohol ran greater distances and with shorter latencies than Ss treated with saline (Barry & Miller, 1962, 1965; Conger, 1951; Freed, 1967, 1968a, 1968b; Grossman & Miller, 1961; Masserman, Jacques & Nicholson, 1945; Masserman & Yum, 1946; Miller, 1956, 1960, 1961; Miller & Barry, 1960). Conger (1951) demonstrated that these effects of alcohol in a conflict situation were a function of alcohol reducing the avoidance tendency while leaving the approach tendency intact. Moreover, studies by Grossman and Miller (1961), Miller and Barry (1960) and Smart (1965) have indicated that alcohol decreased the avoidance tendency solely by diminishing fear, and in no way

affected the strength of responding to the painful stimulus.

In summary then, it is safe to conclude that alcohol reduces the fear associated with the experimental situation without affecting either the effectiveness of the UCS (Grossman & Miller, 1961; Miller & Barry, 1960; Smart, 1965), or the retention of the learned response (Scarborough, 1957).

Specific hypotheses tested

As stated earlier, the present study was an attempt to determine whether the classically conditioned fear response motivates instrumental avoidance responding. In order to do so, a factorial design was used. (See Figure 1.)

It was hypothesized that if preliminary exposure to CS-shock pairings results in fear being conditioned to the CS, then Ss given prior CS-shock exposure (group P) would learn the avoidance response more readily than Ss given only prior CS presentations (group N). Conversely, a reduction or elimination of the fear ordinarily conditioned to the CS would result in a diminution of the facilitative effects of the prior CS-shock experience. Hence Ss injected with alcohol during prior CS-shock exposure (group PA) would not learn the avoidance response as readily as Ss injected with saline during that same phase of the study (group PS), but just as readily as Ss who received only preliminary CS exposure and injected with alcohol or saline (groups NA and NS, respectively).

It follows from the two factor position, that if the conditioning of fear to the CS serves as the basis for the reinforcement of the avoidance response, then any interference in the establishment of fear should likewise interfere with the rate of acquiring the avoidance response. It was thus predicted that Ss injected with alcohol during avoidance training (groups PA' and NA') should not learn the avoidance response as readily as Ss injected with saline during avoidance training (groups PS' and NS').

It was predicted that Ss in group P who receive saline during both prior training and avoidance conditioning should learn the avoidance task more readily than any of the remaining groups in P (i.e. groups PAS', PSA' and PAA').

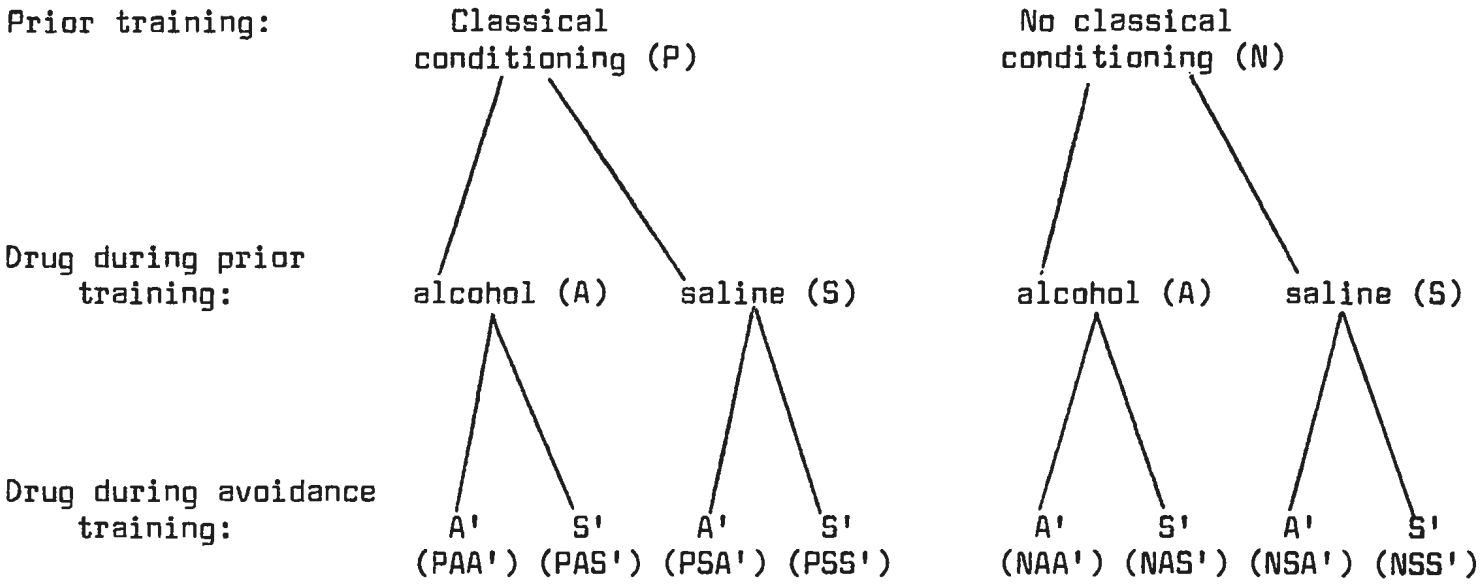

Fig. 1. Experimental design of the present study

In addition, it was expected that a reduction of fear during both phases of the study would be more effective in disrupting the rate of acquiring the avoidance response than a reduction of fear during only one phase of the study. Specifically, groups PAS' and PSA' should learn the avoidance response more rapidly than group PAA'. Moreover, it was expected that if alcohol did mitigate the facilitative effects of the fear conditioning procedure, then there should be no difference between groups PAS', NAS' and NSS', and none between groups PAA', NAA' and NSA'.

Chapter 2
Method

Subjects

Ninety-six experimentally naive, male, hooded rats, whose ad lib. weights were between 190-230 gms. were procured from the Canadian Breeding Laboratories. Ss were housed communally and allowed ad lib. food and water for the duration of the experiment. Ss were randomly assigned to eight groups with the stipulation that each group contained an equal number of Ss.

Apparatus

The apparatus used for avoidance conditioning was a modified Miller-Mowrer shuttle box modeled after that used by Kamin (1957b). The shuttle box, whose interior dimensions were 76.20 cm. x 12.70 cm. x 20.30 cm. was painted flat black. During pretraining, a barrier extending from the roof of the apparatus through the grid floor divided the shuttle box into two halves. Each half of the shuttle box contained a 1 watt lamp situated half-way between the center and ends of the apparatus, and 18.80 cm. above the grid floor.

The UCS, electric shock of 1 ma, was administered through a grid floor made up of 0.5 cm. brass rods spaced 1.10 cm. apart. The CS consisted of the illumination of the 1 watt light, and the simultaneous sounding of a tone 15 db louder than the prevailing sound produced by a white noise generator. Simultaneous with the CS onset, a timer, used to measure response latency, was activated. A photoelectric relay system, situated in middle of the shuttle box, and 3.8 cm. above the grid floor served as a switch which, when interrupted, turned off the CS, UCS and timer.

Alcohol solutions were prepared according to the method described by Thor, Weisman and Boshka (1967) i.e., cc. stock solution required = % volume/volume desired solution x cc. solution to be prepared ÷ % volume/volume stock solution. Injections consisted of administering 15cc/kg (12gm/kg) of alcohol in a 10% solution as suggested by Barry and Wallgren (1968). This dosage of alcohol was selected for use since studies by Buckalew and Cartwright (1968) and Cartwright and Buckalew (1969) have indicated that this dosage does not affect respiration, vestibular functioning,

muscle tone, auditory sensitivity, exploratory activity, muscular coordination, shock escape latency and balance. Moreover, pilot studies conducted by the present investigator indicated that this level of alcohol did not affect shock escape latency. In addition, Fazekas (1966), and Majchrowicz, Lipton, Meek and Hall (1968) have found that blood alcohol level was normal six hours after injection of dosages of alcohol as large as 2.4gm/kg. This would thus suggest that this amount of alcohol injected during prior training would not have an effect during subsequent avoidance testing, yet, at the same time would be of sufficient strength to reduce fear during pre-training or avoidance testing.

Procedure

Design. The basic design of the present experiment involved a 2^3 (prior CS-shock exposure (P) vs. prior CS exposure only (N) x alcohol (A) vs. saline (S) during prior training x alcohol (A') vs. saline (S') during avoidance learning) factorial design (Winer, 1962). Repeated measures were included on two dependent variables.

Pretraining. For two days prior to pretraining, all Ss were handled and allowed to explore the apparatus for one-half hour per day. On day three one-half of the Ss were placed one at a time in one side of the shuttle box and given 25 light-tone and shock pairings. A delay conditioning paradigm was used, such that every 30 sec. the CS, light and tone, were turned on for 5 sec. followed by the presentation of the UCS for 0.5 sec. Both the CS and UCS were terminated simultaneously. The remaining Ss were given identical treatment except that the shock source was disconnected from the shuttle box and hence the UCS was not presented. Five minutes prior to placement in the shuttle box for pretraining, one-half of the Ss in each group were given an interperitoneal injection of 15cc/kg of alcohol in a 10% solution. The remaining Ss were given an equivalent amount (volume/weight) of physiological saline.

Avoidance conditioning. Twenty-four hours after the end of pretraining, Ss were given 90 avoidance trials. The intertrial and

interstimulus intervals were the same as that used during pretraining, i.e., 30 sec. and 5 sec., respectively. The main difference between pretraining and avoidance conditioning was that the barrier dividing the two compartments during pretraining was removed for avoidance conditioning, thereby allowing Ss to run from one compartment to the other. If S crossed the center of the shuttle box after UCS onset, both the CS and UCS were terminated. If that response was made after CS onset, but before UCS onset, the CS was terminated and the UCS was withheld. Intertrial responding was suppressed by shocking S whenever he crossed to the other compartment without the CS being presented.

Five minutes prior to avoidance training, one-half of the Ss in each of the four groups were given an IP injection of alcohol consisting of 15cc/kg in a 10% solution. The remaining Ss were given an equivalent amount (volume/weight) of physiological saline. Following suggestions made by Grossman and Miller (1961) and Miller (1961), the factorial combination of drug vs. no drug during prior training and avoidance conditioning was employed to control for state dependent learning effects. That is, several investigators (e.g. Barry, Koepfer & Lutch, 1965; Crow, 1966; Overton, 1966); have demonstrated that if an animal learns a task after injection of alcohol, it may be unable to perform that task while in a non-drug state. Conversely, if an animal learns a task while in the non-drug state it may not be able to perform that task after injection of alcohol. This phenomenon, referred to as state dependent or drug dissociated learning (Belleville, 1963; Overton, 1969; Stewart, 1962) is believed to be a function of Ss learning to respond on the basis of some physiological state produced by the drug (Barry, 1968; Kubena & Barry, 1969; Stewart, 1962).

Chapter 3
Results

The dependent variables used in the present study were designed to detect (a) motivation to escape from the CS, as reflected by the latency of responding on the first five and last five trials, (b) level of avoidance learning during the early trials of avoidance conditioning, reflected by the number of trials to the first avoidance and by the number of trials to three consecutive avoidances, (c) level of avoidance learning on the later trials of avoidance conditioning, reflected by number of trials to a criterion of nine out of ten consecutive avoidances, and (d) performance throughout avoidance training, reflected by frequency of avoidances over blocks of thirty trials. In addition, the number of responses made during the intertrial-interval (ITR) by each group was analyzed to determine whether prior training and/or administration of alcohol affected the discrimination between the danger and safe periods (i.e., when the CS was on, or off, respectively). A summary of means and standard deviations on each of these dependent measures for each of the eight groups tested is presented in Table 1.

Trial to first avoidance

A 2^3 (prior training x drug during prior training x drug during avoidance training) factorial design analysis of variance (Winer, 1962) was computed on the number of trials to first avoidance. As the source table indicates (see Table 2), the analysis revealed significant main effects for prior training ($F=14.43$; $df=1,88$; $p<.001$) and drug during avoidance training ($F=9.39$; $df=1,88$; $p<.01$). A comparison of the means involved revealed that Ss who received prior CS-shock pairings made the first avoidance response earlier than Ss who did not receive this prior exposure. Administration of alcohol during avoidance training increased the number of trials needed for S to make the first avoidance response relative to those Ss who received saline during this phase. In addition to these main effects a significant interaction between prior training and drug during prior training was found ($F=4.54$; $df=1,88$; $p<.05$), and is illustrated in Figure 2. Newman Keuls multiple comparisons (Winer, 1962) on this

Table 1

Means and standard deviations for trials to first avoidance (TFA), trials to three consecutive avoidances (TCA), trials to nine out of ten consecutive avoidance (NTA), reciprocal of latency over the first five (RL_1) and last five trials (RL_2), avoidances over blocks of thirty trials (BTT) and number of intertrial responses (ITR) for all groups

	Dependent variables								
	TFA	TCA	NTA	RL		BTT			ITR
				1	2	1	2	3	
PSS $\bar{x}$	8.50	21.25	42.58	.205	.379	9.33	20.91	21.75	6.25
PSS s.d.	5.90	8.71	26.53	.029	.127	3.38	7.10	6.49	9.07
PAS $\bar{x}$	24.70	44.44	59.58	.137	.256	3.33	16.50	16.16	3.50
PAS s.d.	22.55	23.94	29.35	.033	.060	3.08	9.06	8.28	2.64
PSA $\bar{x}$	16.83	42.41	69.33	.163	.286	4.00	11.83	16.83	8.58
PSA s.d.	12.02	19.66	19.31	.031	.088	2.59	6.79	7.92	6.37
PAA $\bar{x}$	27.50	56.41	69.58	.149	.247	2.16	12.58	16.16	5.66
PAA s.d.	11.13	22.24	20.16	.028	.101	1.93	6.04	8.14	3.84
NSS $\bar{x}$	24.16	51.16	68.12	.155	.266	4.33	12.16	14.83	2.66
NSS s.d.	15.61	23.77	30.45	.025	.089	5.58	7.70	8.92	2.64
NAS $\bar{x}$	25.16	50.41	68.33	.154	.248	2.83	13.00	14.33	3.50
NAS s.d.	14.64	24.81	23.46	.024	.085	3.35	7.71	8.18	3.31
NSA $\bar{x}$	40.91	70.25	76.58	.156	.249	3.58	7.41	15.00	6.66
NSA s.d.	17.82	19.19	18.27	.016	.220	1.44	6.89	8.48	4.39
NAA $\bar{x}$	38.33	60.16	75.60	.155	.278	2.25	10.00	11.91	5.33
NAA s.d.	23.71	30.65	28.95	.021	.159	3.49	9.84	10.58	4.00

interaction indicated that Ss in group PS made the first avoidance response earlier than Ss in groups PA, NA and NS. As predicted, the difference between the means of groups PA, NA and NS were minimal and not statistically reliable.

Trials to three consecutive avoidances

A 2^3 (prior training x drug during prior training x drug during avoidance training) factorial design analysis of variance (Winer, 1962) was computed on the number of trials to three consecutive avoidances. The analysis, summarized in Table 3, yielded results similar to the analysis on trials to first avoidance. Specifically, the present analysis revealed significant main effects for the prior training ($F=12.81$; $df=1,88$; $p<.001$) and for drug during avoidance training ($F=13.67$; $df=1,88$; $p<.001$). A comparison of the means involved indicated that Ss given prior CS-shock exposure made three consecutive avoidances earlier than Ss given no prior CS-shock exposure. As in the case of trials to first avoidance, administration of alcohol retarded the rate at which Ss reached the criterion of three consecutive avoidance responses.

In addition to these main effects, a prior training x drug during prior training interaction, illustrated in Figure 3, was found to be statistically significant ($F=5.26$; $df=1,88$; $p<.05$). Newman Keuls multiple comparisons (Winer, 1962) on the interaction revealed that Ss in group PS reached the criterion of three consecutive avoidance responses earlier than Ss in groups PA, NA and NS. As before, there were no statistically significant differences between the means of groups PA, NA and NS.

Trials to nine out of ten consecutive avoidances

A 2^3 (prior training x drug during prior training x drug during avoidance training) factorial design analysis of variance (Winer, 1962) was computed on the number of trials to a criterion of nine out of ten consecutive avoidances. The significant main effects for this analysis, shown in Table 4, indicated that Ss given prior exposure to CS-shock pairings reached the criterion earlier than Ss who received prior CS exposure only ($F=4.83$; $df=1,88$; $p<.05$). Moreover, administration of alcohol during avoidance training significantly retarded the rate of reaching criterion ($F=7.39$; $df=1,88$; $p<.01$).

Table 2

Analysis of variance on trials to first avoidance for a 2 x 2 x 2 factorial combination of prior classical conditioning vs. no prior classical conditioning x alcohol vs. saline during prior training x alcohol vs. saline during avoidance training

Source	df	MS	F
Prior training (A)	1	3888.76	14.43**
Drug during prior training (B)	1	956.34	3.54
Drug during avoidance training (C)	1	2531.76	9.39**
A x B	1	1225.51	4.54*
A x C	1	536.76	1.99
B x C	1	123.76	<1
A x B x C	1	6.51	<1
Within cell (experimental error)	88	269.43	

* $p < .05$

** $p < .01$

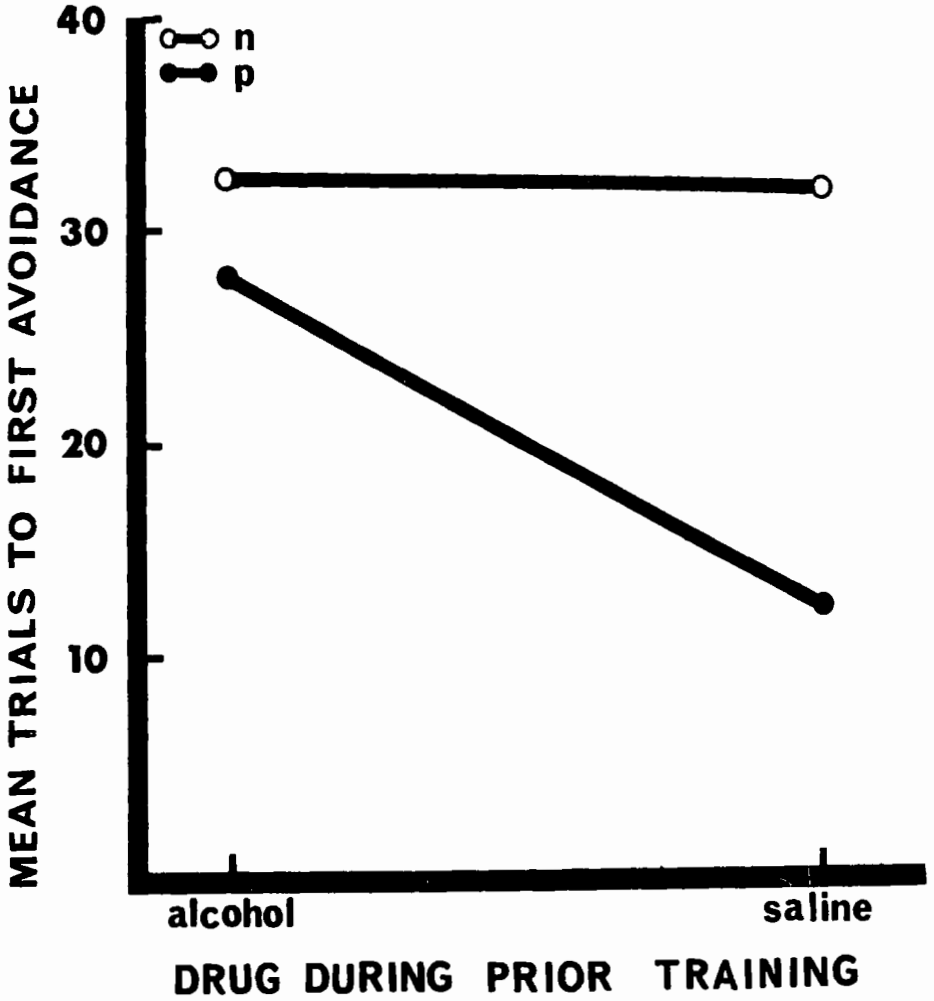

Fig. 2. Interaction between prior training (P vs. N) and drug during prior training (A vs. S) for a 2 x 2 factorial design on trials to first avoidance.

Table 3

Analysis of variance on trials to three consecutive avoidances for a 2 x 2 x 2 factorial combination of prior classical conditioning vs. no prior classical conditioning x alcohol vs. saline during prior training x alcohol vs. saline during avoidance training

Source	df	MS	F
Prior training (A)	1	6419.01	12.81**
Drug during prior training (B)	1	858.01	1.71
Drug during avoidance training (C)	1	6851.26	13.67**
A x B	1	2635.51	5.26*
A x C	1	58.59	<1
B x C	1	605.01	1.20
A x B x C	1	7.60	<1
Within cell (experimental error)	88	500.99	

* $p < .05$

** $p < .01$

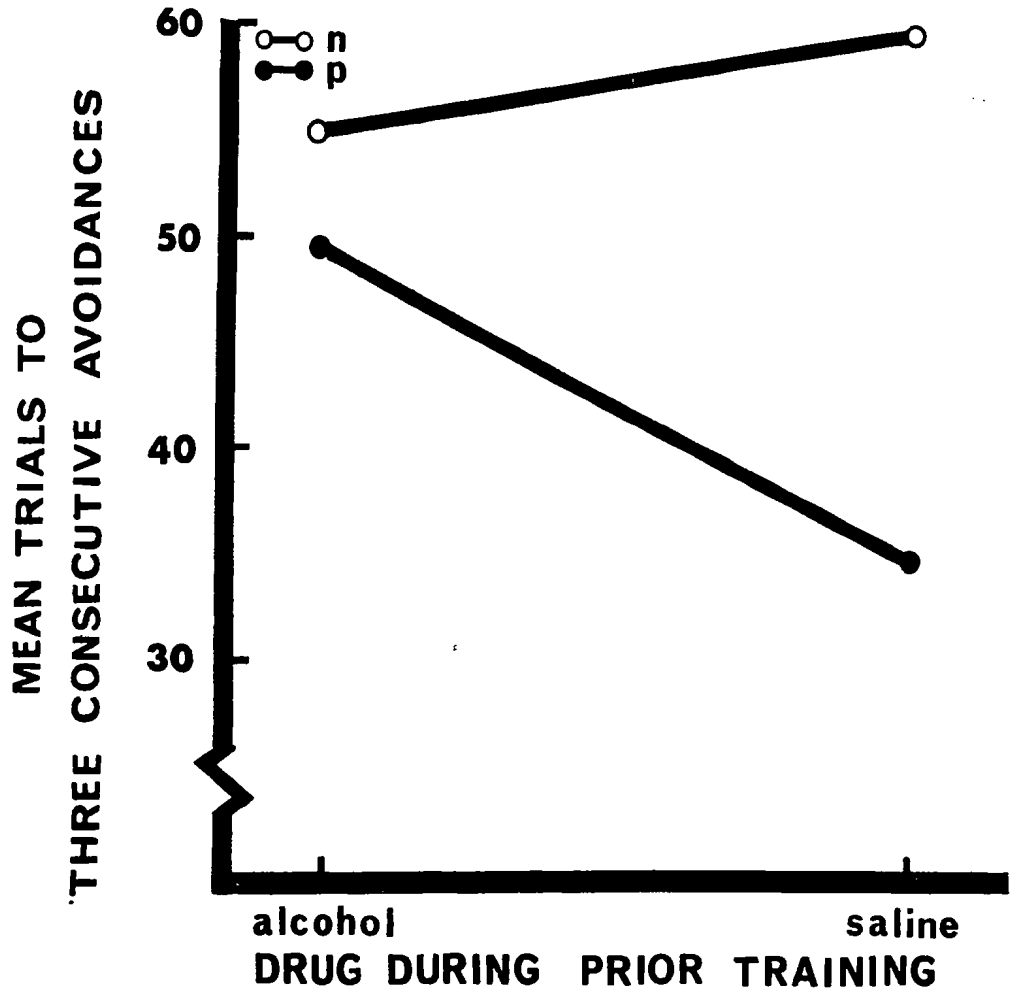

Fig. 3. Interaction between prior training (P vs. N) and drug during prior training (A vs. S) for a 2 x 2 factorial design on trials to three consecutive avoidance responses.

No interactions were found to be significant for this dependent variable.

Latency of response on the first five and last five trials

An F-max test on the variances of the latencies for groups PAS and NSA was computed. The value of the F-max statistic (F-max=15.83; df=8,11; $p < .01$) was found to be significant, indicating non-homogeneous variances and the necessity for a reciprocal transformation. The F-max test on the reciprocals (F-max=4.21; df=8,11) indicated that the transformation successfully reduced the heterogeneity.

A $2^3 \times 2$ (prior training x drug during prior training x drug during avoidance training x the first five vs. the last five trials) repeated measures factorial design analysis of variance (Winer, 1962) was computed on the reciprocal of the response latency. As can be seen in the summary table (see Table 5), the only significant main effects were found for drug during prior training ($F=4.83$; df=1,88; $p < .05$), and for the first five vs. the last five trials ($F=108.33$; df=1,88; $p < .001$). These results indicated that Ss given alcohol during prior training responded more slowly during subsequent avoidance training than did Ss given saline during prior training. Moreover, the analysis revealed that the speed of responding increased significantly from the first five trials to the last five trials.

In addition to these main effects, the analysis revealed a significant prior training x drug during prior training interaction ($F=5.66$; df=1,88; $p < .05$), illustrated in Figure 4. Newman Keuls multiple comparisons (Winer, 1962) on the interaction revealed that the speed of responding in group PS was significantly faster than that of groups PA, NA and NS. There were no reliable differences between the means of groups PA, NA and NS. No other main effects or interactions were found to be significant.

Number of avoidances in blocks of thirty trials

A $2^3 \times 3$ (prior training x drug during prior training x drug during avoidance training x blocks of thirty trials) repeated measures factorial design analysis of variance (Winer, 1962) was computed on the number of avoidances in blocks of thirty trials. The analysis, shown in Table 6, yielded main effects for prior

Table 4

Analysis of variance on trials to nine out of ten consecutive avoidances for a 2 x 2 x 2 factorial combination of prior classical conditioning vs. no prior classical conditioning x alcohol vs. saline during prior training x alcohol vs. saline during avoidance training

Source	df	MS	F
Prior training (A)	1	2948.17	4.83*
Drug during prior training (B)	1	532.04	<1
Drug during avoidance training (C)	1	4510.04	7.39**
A x B	1	368.17	<1
A x C	1	522.67	<1
B x C	1	672.05	1.10
A x B x C	1	228.15	<1
Within cell (experimental error)	88	610.00	

* $p < .05$

** $p < .01$

Table 5

Repeated measures analysis of variance on the mean reciprocal of latency for a 2 x 2 x 2 x 2 factorial combination of prior classical conditioning vs. no prior classical conditioning x alcohol vs. saline during prior training x alcohol vs. saline during avoidance training x the first five and last five trials

Source	df	MS	F
Between subjects	95		
Prior training (A)	1	.001	1.83
Drug during prior training (B)	1	.029	4.83**
Drug during avoidance training (C)	1	.005	<1
A x B	1	.034	5.66**
A x C	1	.008	1.25
B x C	1	.015	2.49
A x B x C	1	.003	<1
Subjects within groups error between	88	.006	
Within subjects	96		
Blocks of trials (D)	1	.605	108.33**
A x D	1	.002	<1
B x D	1	.000	<1
C x D	1	.000	<1
A x B x D	1	.002	<1
A x C x D	1	.001	<1
B x C x D	1	.002	<1
A x B x C x D	1	.003	<1
D x subjects within groups error within	88	.006	

* $p < .05$

** $p < .01$

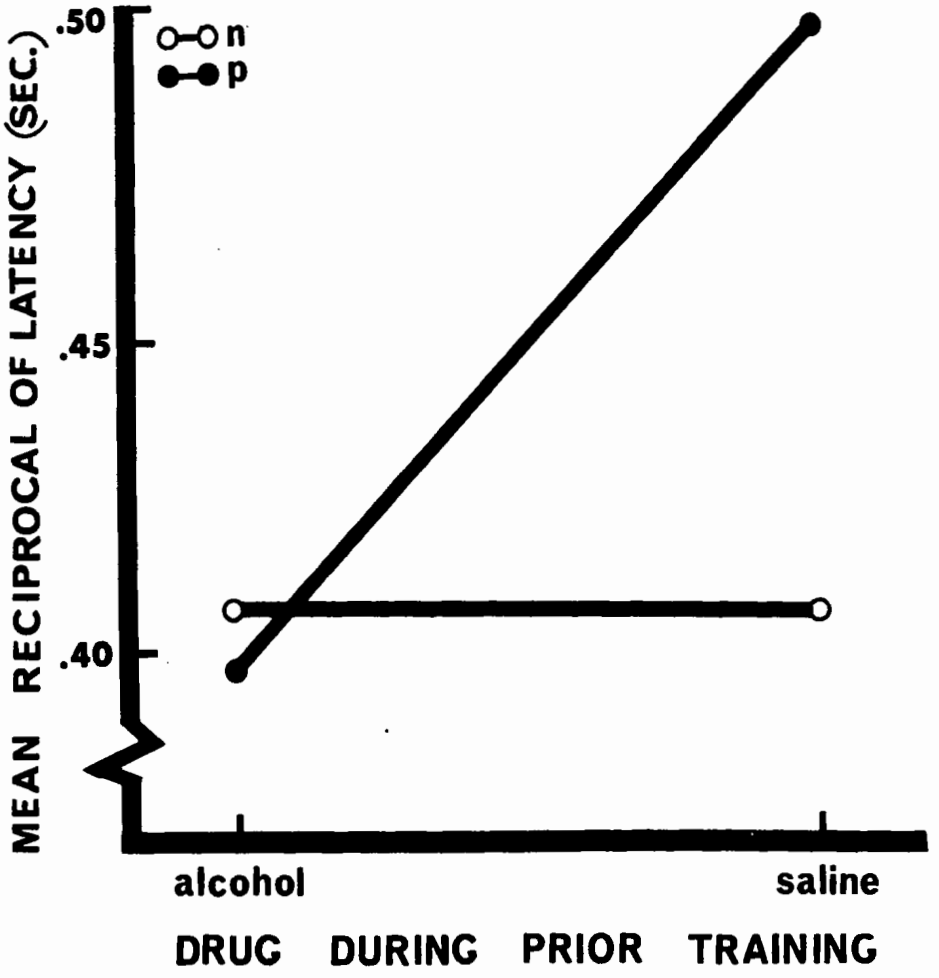

Fig. 4. Interaction between prior training (P vs. N) and drug during prior training (A vs. S) for a 2 x 2 factorial design on reciprocal of latency over the first five and last five trials

training (F=8.46; df=1,88; $p<.05$), drug during avoidance training (F=7.23; df=1,88; $p<.05$), and blocks of trials (F=211.85; df=2,176; $p<.001$). These results indicate that prior exposure to the light-tone and shock pairings facilitated avoidance learning, while administration of alcohol, as opposed to saline, during avoidance training resulted in Ss making fewer avoidance responses. Moreover, the number of avoidances made by each group increased over blocks of trials.

In addition to these main effects, the analysis also revealed a significant drug during avoidance x blocks of trials interaction (F=3.99; df=2,176; $p<.05$), illustrated in Figure 5. Newman Keuls multiple comparisons (Winer, 1962) on the interaction revealed that Ss who received alcohol during avoidance conditioning made fewer avoidance responses during the first and second blocks of trials when compared with Ss who received saline during avoidance training. There were, however, no differences between the means of the two groups on the third block of trials. In addition to this, the multiple comparisons also indicated that the number of avoidances increased significantly over all three blocks of trials for those Ss who received alcohol. In contrast, avoidances for those Ss injected with saline during avoidance training increased significantly between the first and second block of trials, but not between the second and third block of trials.

Although a prior training x drug during prior training x blocks of trials interaction was hypothesized to be significant, the analysis of variance did not bear out this prediction. Winer (1962, p.208) has suggested that if an *a priori* hypothesis has been postulated, the predicted comparisons should be made regardless of whether the over-all F test is significant. Since specific predictions were made concerning the differential effects of alcohol and saline on pre-exposure to CS-shock pairings, multiple comparisons were performed on the individual means comprising the prior training x drug during prior training x blocks of trials interaction.

As predicted, these comparisons, illustrated in Figure 6, revealed that group PS performed significantly better than groups PA, NA and NS on the first block of trials. Performance over the

Table 6

Repeated measures analysis of variance on number of avoidances for a 2 x 2 x 2 x 3 factorial combination of prior classical conditioning vs. no prior classical conditioning x alcohol vs. saline during prior training x alcohol vs. saline during avoidance training x blocks of thirty trials

Source	df	MS	F
Between subjects	95		
Prior training (A)	1	885.50	8.46**
Drug during prior training (B)	1	172.67	1.65
Drug during avoidance training (C)	1	757.25	7.23**
A x B	1	143.09	1.36
A x C	1	49.18	<1
B x C	1	140.28	1.34
A x B x C	1	69.03	<1
Subjects within groups error between	88	104.62	
Within subjects	192		
Blocks of trials (D)	2	3892.83	211.85**
A x D	2	48.19	2.61
B x D	2	40.42	2.19
C x D	2	73.57	3.99*
A x B x D	2	9.94	<1
A x C x D	2	4.41	<1
B x C x D	2	12.01	<1
A x B x C x D	2	16.95	<1
D x subjects within groups error within	176	18.41	

* $p < .05$

** $p < .01$

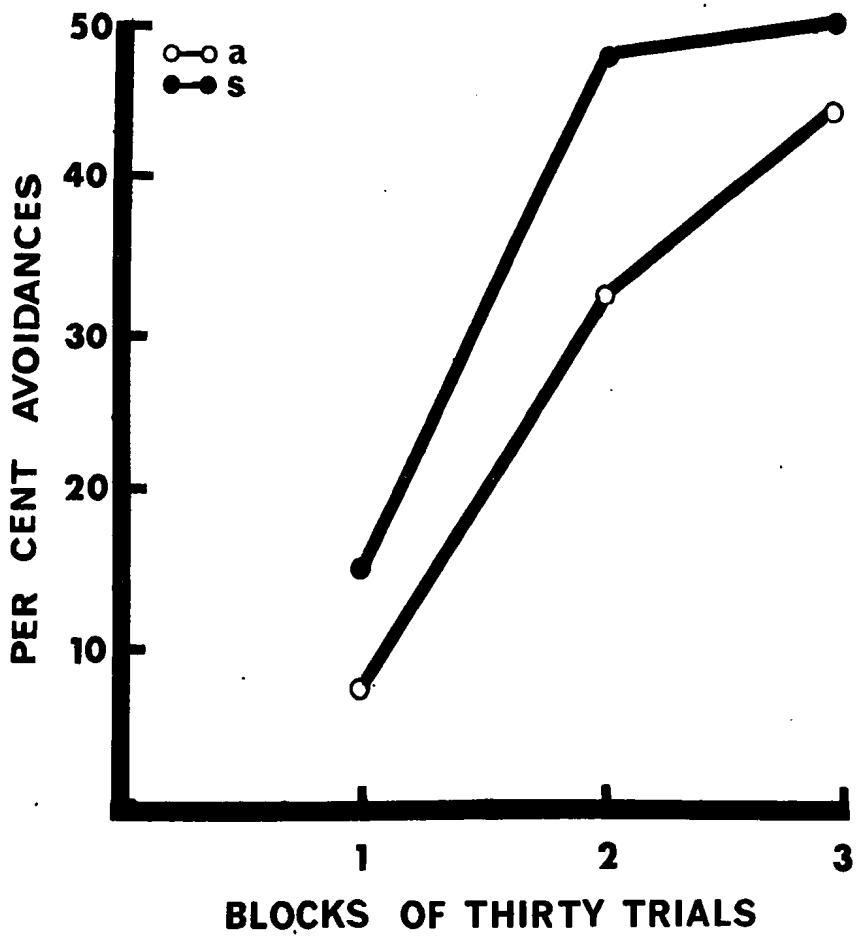

Fig. 5. Interaction between drug during avoidance training (A' vs. S') and blocks of trials for a 2 x 3 factorial design on number of avoidances over blocks of thirty trials.

second block of trials, however, indicated no differences between groups PS and PA, but both of these groups performed better than groups NS and NA. On the third block of trials, only PS and NA differed significantly. No other specific predictions or tests were made for this particular factorial combination.

Intertrial Resposes

A 2^3 (prior training x drug during prior training x drug during avoidance training) factorial design analysis of variance (Winer, 1962) was computed on the number of intertrial responses. The analysis, shown in Table 7, indicated that drug during avoidance was the only significant main effect ($F=6.59$; $df=1,88$; $p<.05$). Comparisons between the means involved indicated that administration of alcohol resulted in the occurence of a greater number of ITR's. No other main effects or interactions were found to be significant on this variable.

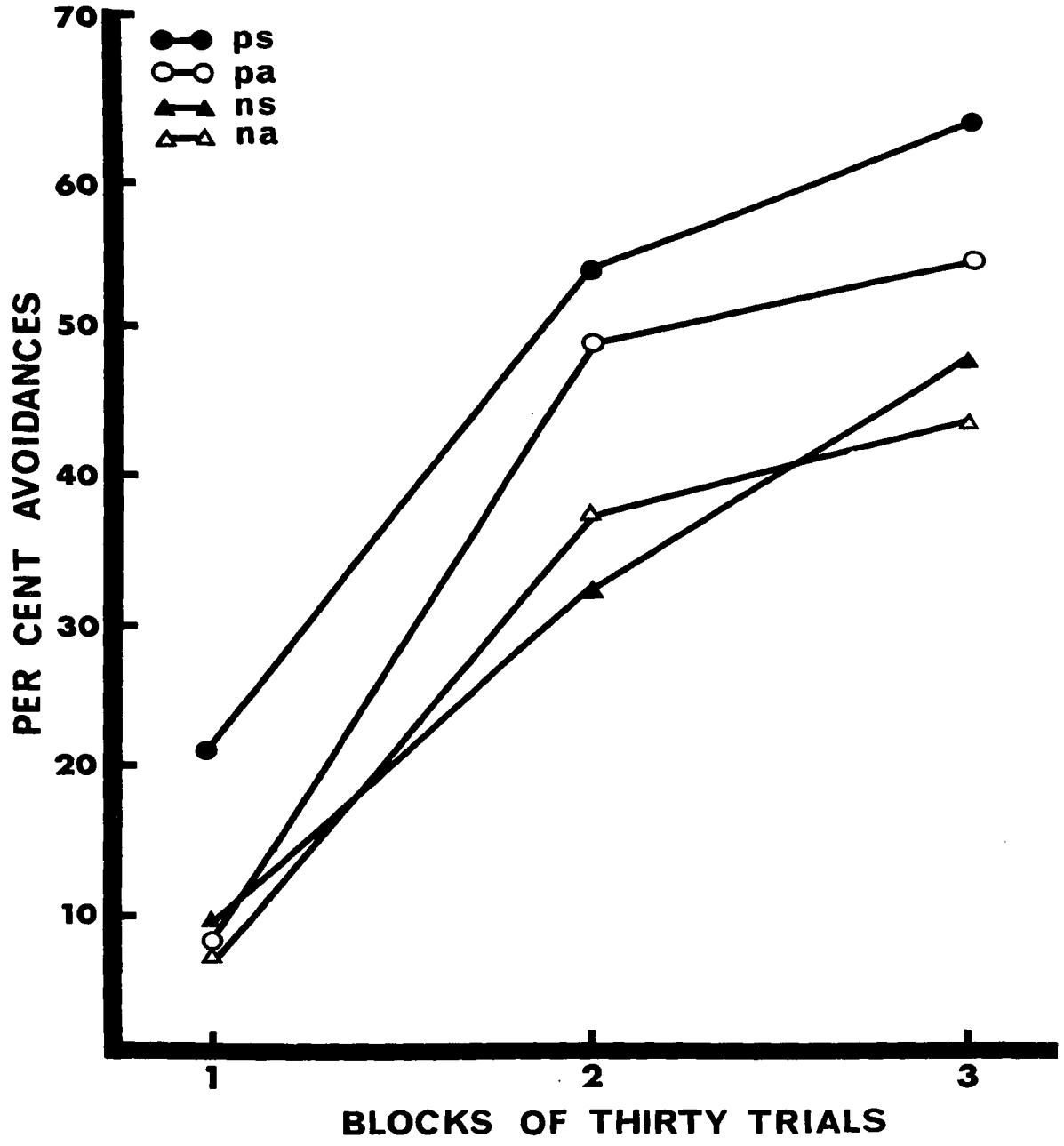

Fig. 6. Interaction between prior training (P vs. N), drug during prior training (A vs. S) and blocks of trials for a 2 x 2 x 3 factorial design on number of avoidances over blocks of thirty trials.

Table 7

Analysis of variance on the number of intertrial responses for a 2 x 2 x 2 factorial combination of prior classical conditioning vs. no prior classical conditioning x alcohol vs. saline during prior training x alcohol vs. saline during avoidance training

Source	df	MS	F
Prior training (A)	1	51.04	2.10
Drug during prior training (B)	1	57.04	2.35
Drug during avoidance training (C)	1	160.16	6.59*
A x B	1	40.04	1.64
A x C	1	2.67	<1
B x C	1	8.17	<1
A x B x C	1	6.00	<1
Within cell (experimental error)	88	24.27	

* $p < .05$

Chapter 4
Discussion

Two factor theorists assume that avoidance learning involves two distinct phases; (a) the classical conditioning of fear to the CS, and (b) escape from the CS via an instrumental response. These theorists (e.g., Rescorla & Solomon, 1967) have suggested that prior CS-UCS (shock) pairings (i.e., classical conditioning) should facilitate subsequent avoidance learning. Specifically, the mechanism underlying this process is thought to be that fear is conditioned to the CS during prior CS-shock exposure, hence the first phase of avoidance learning is completed prior to S receiving any avoidance trials. It has been argued that, under these conditions, the subsequent avoidance task merely involves the acquisition of the correct instrumental response.

In accordance with other tests of the two factor position (e.g., Baum, 1969a; DeToledo & Black, 1967; Slotnick, 1968) the analysis on all dependent measures in the present study indicated that Ss given prior CS-shock pairings learned the subsequent avoidance task more readily than Ss who received prior CS exposure only. Although it is clear that prior Pavlovian procedures facilitated subsequent avoidance learning, the question still remains whether this facilitation was, in fact, a function of fear being conditioned to the CS during pre-exposure to the CS-shock pairings. It was hypothesized, that if the conditioning of fear to the CS was responsible for the facilitation, then a reduction or elimination of the fear should mitigate the facilitative effects of prior exposure to CS-shock pairings. That is to say, Ss injected with a fear reducing drug like alcohol (Barry & Miller, 1962; Conger, 1951; Freed, 1967, 1968a, 1968b; Grossman & Miller, 1961) during Pavlovian training should not have been as motivated to escape from the CS, nor have learned the avoidance task as readily as Ss injected with saline during that same phase.

Effects of alcohol administered during prior training

In accordance with the prediction made above, the analysis on latency of responding over the first five and last five trials

indicated that Ss who had been injected with saline during prior CS-shock pairings (PS), responded more rapidly than Ss injected with either saline or alcohol and given prior CS exposure (NS and NA). When Ss were injected with alcohol during prior exposure to the CS and shock (PA), the latency of responding was increased to such an extent that there were no differences between these Ss and those in groups NS or NA. An interpretation of these results from the viewpoint of two factor theory is that prior CS-shock pairings served to establish Pavlovian CR's involving fear, which in turn motivated escape from the CS during avoidance learning. In contrast, by injecting Ss with alcohol during prior CS-shock exposure, the fear ordinarily conditioned to the CS was reduced and consequently the motivation to escape from the CS was reduced.

It follows, from a drive reduction point of view, that if the motivation to escape from the CS was greater in group PS than in groups PA, NA and NS, then escape from the CS would be more reinforcing for group PS than for any of the remaining groups (PA, NA and NS). Accordingly, the avoidance response should be acquired more readily by group PS than by groups PA, NA and NS, resulting in a prior training x drug during prior training interaction on those dependent variables reflecting the learning of the avoidance response. This in fact was found to be the case, in that significant prior training x drug during prior training interactions were found on the dependent variables of trials to first avoidance and trials to three consecutive avoidances. In addition, multiple comparisons for these interactions indicated that saline treated Ss given prior CS-shock pairings made both the first avoidance and three consecutive avoidances earlier than Ss given prior CS-shock pairings and treated with alcohol, or Ss received only prior CS exposure and injected with alcohol or saline. As predicted, the differences between PA, NA and NS were minimal and not statistically significant. These results can be taken to indicate that the relatively high motivational state in PS led to superior avoidance learning compared to groups PA, NA and NS in which the motivational state was relatively low.

The above conclusion is, however, equivocal in that the prior training x drug during prior training interaction was

significant only for latency over the first five and last five trials, trials to first avoidance and three consecutive avoidances, and not significant when trials to nine out of ten consecutive avoidances and avoidances over blocks of trials were used as dependent variables. It will be recalled that these measures were taken to indicate level of learning on the later avoidance trials. Also, as noted earlier, it follows from a drive reduction position that if the motivation was relatively higher for Ss in group PS than for Ss in groups PA, NA and NS, then the avoidance response should have been acquired more readily by Ss in group PS than by the other Ss. The apparent discrepency between this expectation, and the fact that a significant prior training x drug during prior training interaction was not found for those measures reflecting later avoidance performance, can be accounted for by making the assumption that alcohol merely reduced rather than eliminated the fear conditioned to the CS during prior exposure to the CS and shock. To be more explicit, if it is assumed that alcohol only reduced fear during the preshock phase of the study, then performance should have been initially poorer for Ss treated with alcohol than those treated with saline. However, only a few trials may have been necessary to increase the fear to a level sufficient to motivate instrumental escape from the CS during the later avoidance trials. If this were the case, then performance in group PS should have been superior to that of group PA during the early trials of avoidance training, but the two groups should not have differed on the later trials of avoidance conditioning. In fact, multiple comparisons for the prior training x drug during prior training x blocks of trials factorial combination yielded results consistent with this prediction. That is, those Ss injected with saline during prior CS-shock exposure made significantly more avoidance responses on the first block of trials than did Ss who received alcohol during prior exposure to CS-shock pairings. In addition, there were no differences between Ss injected with alcohol during prior CS-shock exposure and those Ss who received alcohol or saline and who were given only prior CS presentations. On the second and third blocks of trials, however, there were no differences between Ss who received alcohol and those

who received saline during prior exposure to the CS and shock. It thus appears that although alcohol reduced the fear conditioned to the CS during prior CS-shock exposure, this reduction was not sufficiently great to completely interfere with the facilitative effects of prior CS-shock exposure throughout avoidance training. It is essential to note that the results discussed earlier for latency over the first five and last five trials, trials to first avoidance, trials to three consecutive avoidances and trials to a criterion of nine out of ten consecutive avoidances, are all consistent with this assumption.

When one considers the dependent variables discussed thus far, it seems apparent that the facilitative effects of prior CS-shock exposure were a result of fear being conditioned to the CS during that phase. The fear, so conditioned, served to motivate escape from the CS during avoidance training. When alcohol was administered during prior exposure to the CS and shock, there was a reduction in the fear ordinarily conditioned to the CS, and consequently the motivation to escape from the CS was reduced.

Alternative explanations for the present results, i.e., learning to learn, state dependent learning, and long term fear reducing properties of alcohol do not account for the data as well as the conditioned fear hypothesis. Had prior CS-shock pairings functioned in a non-specific sense and provided a basis for learning to learn, administration of alcohol would not have disrupted the effects of this experience as it did in the present study unless there was a state dependent learning effect. For example, it was found that Ss who received saline during prior exposure to the CS and shock responded more rapidly during the first five and last five trials, made more avoidances over the first block of thirty trials, and made the first avoidance and three consecutive avoidances earlier than Ss injected with alcohol during this same phase of the study. Evidently, the facilitative effects of prior exposure to the CS-shock pairings was not a function of Ss learning to learn.

It might then be argued that administration of alcohol during prior exposure to the CS and shock led to drug dissociated

learning thereby disrupting the facilitative effects of prior fear conditioning. Had the poorer avoidance learning in group PA, as compared to group PS, been due to a lack of transfer from the drug state to the non-drug state, both non-shifted groups i.e., PSS' and PAA', would have learned the avoidance task more readily than both shifted groups, i.e., PAS' and PSA'. The analysis on those measures reflecting motivation and learning did not indicate any decrement in learning the avoidance task as a result of drug dissociated learning. That is, since no drug during prior training x drug during avoidance training interaction was found to be significant, it seems that group PS performed significantly better than did group PA regardless of the drug treatment received during avoidance training. Moreover, on all those measures reflecting learning of the avoidance task, group S' performed better than group A' regardless of the drug treatment received during prior training. It thus appears that the poorer performance in group PA as compared to group PS was not a result of a lack of transfer from the drug to the non-drug state.

Finally, it could be argued that administration of alcohol had had long term fear reducing properties, which affected performance 24 hours after initial injection. If this was the case, then these effects should also have been evident in Ss who received alcohol and prior exposure to the CS only. The results, on all of the dependent measures, indicated no differences between groups NS and NA, suggesting that administration of alcohol did not have fear reducing properties lasting for 24 hours.

The most reasonable explanation of these results is that prior exposure to the CS and shock pairings alone facilitated subsequent avoidance learning. This facilitative effect appears to be a function of fear being conditioned to the CS during prior training, thereby providing a motivational basis for escape from the CS. Under these conditions then, subsequent avoidance learning involves the acquisition of the correct instrumental response and, therefore the rate of acquiring the avoidance response progresses more rapidly in Ss who received Pavlovian fear conditioning than in Ss who received no such prior training. Moreover, the results of this

experiment also indicated that administration of alcohol during Pavlovian fear conditioning acted in such a way as to reduce the fear ordinarily conditioned to the CS, thereby mitigating the facilitative effects of the fear conditioning procedure. In addition, the present study yielded results indicating that administration of alcohol during avoidance conditioning reduced the rate of avoidance responding and increased the intertrial response (ITR) rate also as a result of alcohol reducing fear. A more detailed discussion of the effects of alcohol administered during avoidance conditioning is presented in the following section.

Effects of alcohol administered during avoidance training

The present study indicated that Ss who received alcohol during avoidance training made significantly more intertrial responses (ITR) than did Ss who received saline during avoidance conditioning. One likely possibility for this is that the effects of alcohol diminished the distinction between the danger periods (when the CS was on) and the safe periods (during the intertrial interval when the CS was off). Specifically, alcohol reduced the fear conditioned to the CS, as demonstrated by the fact that alcohol mitigated the facilitative effects of the fear conditioning procedure, and consequently Ss respond non-differentially to the entire stimulus situation. The net result of this was that Ss responded more often during the intertrial interval. The important point, however, is that the present study suggests that it is not merely a breakdown in the discrimination between the CS and non-CS periods which was responsible for the increased ITR rate, but a failure on the part of S to distinguish between the relatively small level of fear aroused by the CS and that of the rest of the experimental situation. It is of interest to note that Thompson, Sachson and Higgins (1969) investigating intertrial responding arrived at conclusions similar to those of the present study.

Based on the analysis presented above, one would expect that administration of alcohol during avoidance training should also have decreased the rate of avoidance responding. In line with previous investigations (e.g., Berger, 1969; Pawlowski et al., 1961; Scarborough, 1957), the analysis on all of the dependent measures

reflecting learning of the avoidance response indicated that administration of alcohol retarded the avoidance response rate. The analysis on the number of avoidances over blocks of trials indicated that the effectiveness of alcohol in reducing avoidance responding did not, however, continue throughout avoidance training. Specifically, Ss who received alcohol during avoidance training, made fewer avoidance responses during the first and second blocks of trials. The difference between these two groups on the third block of trials was not statistically reliable. This resulted from the fact that the number of avoidances in the alcohol treated Ss increased significantly over each of the three blocks of trials, while avoidance responses in saline treated Ss increased only between the first and second block of trials. In view of these results, it does not seem unreasonable to conclude that this effect was a function of the effectiveness of alcohol diminishing over blocks of trials.

The fact that alcohol administered during avoidance training reduced the number of avoidances can be interpreted in terms of the two factor position of avoidance learning. Such an interpretation would hold that fear is classically conditioned to the CS during the early trials of avoidance conditioning, and that subsequent escape from the CS would reduce the fear, thereby reinforcing the response of escaping from that stimulus. It follows, that if the fear was at a relatively low level, the reinforcement resulting from to an avoidance response would be less than the reinforcement that would occur if fear was at a relatively high level. Thus in the present study it appears that alcohol reduced the fear and, concomitantly, the basis for reinforcement. As a result of the reduction in fear, alcohol treated Ss did not learn the avoidance response as readily as Ss treated with saline during avoidance training.

It could be argued that the poorer performance in the alcohol treated Ss was a function of motor impairments which interfered with instrumental responding. However, experiments carried out by Buckalew and Cartwright (1968) and Cartwright and Buckalew (1969), as noted earlier, indicated that this particular dosage of alcohol (1.2gm/kg) did not affect respiration, muscle tone, muscular coordination, auditory sensitivity, balance, shock escape latency and

vestibular functioning. Moreover, the analysis on latency of responding over the first five and last five trials in the present study did not yield significant differences between Ss treated with alcohol and those treated with saline during avoidance training, indicating that alcohol did not affect shock escape latency. Thus it was very unlikely that the alcohol caused motor impairments which in turn interfered with instrumental avoidance responding.

Since alcohol administered during either prior fear conditioning or avoidance training reduced the fear ordinarily conditioned to the CS during that phase of the study, Ss injected with alcohol during both phases (PAA) should have performed worse than Ss who received alcohol during only one of these phases (PAS and PSA). Under these circumstances, a significant interaction between drug during prior training and drug during avoidance training should have been found. The analysis on all dependent measures did not yield any such significant interactions, thus groups PAS and PSA did not differ from group PAA. This appears to be a result of both the task difficulty involved in the present study and the fact that alcohol only reduced rather than eliminated the fear conditioned to the CS. To be more explicit, if one assumes that alcohol only reduced the fear conditioned to the CS during Pavlovian fear conditioning, then it is not unreasonable to assume that avoidance learning will still be facilitated to some extent. For example, Figure 6 illustrates that although group PA did not differ from groups NA and NS during the first block of trials, Ss in group PA did perform significantly better on the second block of trials than did Ss in groups NA and NS. It thus appears that, as a result of alcohol reducing the fear conditioned to the CS during prior light-tone and shock pairings, there was a level below which avoidance responding did not fall. On the other hand, the avoidance task used in the present study was a very difficult one (Stewart & Anisman, 1970), and as a result avoidance learning progressed slowly. Moreover, the rate of responding in groups PAS, PSA and PAA did not surpass the 55% level in any single block of thirty trials. Taken together, it seems that avoidance responding was limited to a narrow range and consequently, the effects of alcohol administered during

both phases of the study could not be acequately detected.

In summary then, the present study clearly indicated that the facilitative effects of prior CS-shock exposure resulted solely as a function of fear being conditioned to the CS during that phase of the study. The fear so conditioned motivated escape from the CS which in turn reinforced the avoidance response. Accordingly, when the fear ordinarily conditioned to the CS during prior CS-shock exposure was reduced, both the increased motivation to escape the CS and the resulting high rate of avoidance responding were also reduced.

In accordance with the two factor position, the present study strongly supports the hypothesis that fear acts as a motivator and fear reduction as a reinforcer of the avoidance response.

References

Adamson, R. & Black, R. Volitional drinking and avoidance learning in the white rat. Journal of Comparative and Physiological Psychology, 1959, 52, 734-736.

Baron, A. Functions of the CS and UCS in fear conditioning. Journal of Comparative and Physiological Psychology, 1959, 52, 591-593.

Baron, A. & Antonitis, J.J. Punishment and preshock as determinants of bar pressing behavior. Journal of Comparative and Physiological Psychology, 1961, 54, 716-720.

Barry, H. Prolonged measurements of discrimination between alcohol and non-drug states. Journal of Comparative and Physiological Psychology, 1968, 65, 349-352.

Barry, H., Koepfer, E. & Lutch, J. Learning to discriminate between alcohol and non-drug condition. Psychological Reports, 1965, 16, 1072.

Barry, H. & Miller, N.E. Effects of drugs on approach-avoidance conflict tested repeatedly by means of a telescope alley. Journal of Comparative and Physiological Psychology, 1962, 55, 201-210.

Barry, H. & Miller, N.E. Comparison of drug effects on approach, avoidance and escape motivation. Journal of Comparative and Physiological Psychology, 1965, 58, 18-24.

Barry, H. & Wallgren, H. A further note on preparing alcohol solutions. Quarterly Journal of Studies in Alcohol, 1968, 29, 176-178.

Baum, M. Reversal learning of an avoidance response as a function of prior fear conditioning and fear extinction. Canadian Journal of Psychology, 1965, 19, 85-93.

Baum, M. Perseveration of fear measured by changes in rate of responding in dogs. Canadian Journal of Psychology, 1967, 24, 535-548.

Baum, M. Dissociation of respondent and operant processes in avoidance learning. Journal of Comparative and Physiological Psychology, 1969, 67, 83-88. (a)

Baum, M. Paradoxical effects of alcohol on the resistance to extinction of an avoidance response in rats. Journal of Comparative and Physiological Psychology, 1969, 69, 238-240. (b)

Belleville, R.E. Control of Behavior by drug produced internal stimuli. Psychopharmacologia, 1963, 5, 95-105.

Berger, H.J. Chlorpromazine and ethanol combination: Effects on respiration, random motor activity and conditioned avoidance-escape in mice. Quarterly Journal of Studies in Alcohol, 1969, 30, 862-869.

Bindra, D. & Cameron, L. Changes in experimentally produced anxiety with the passage of time: Incubation effect. Journal of Experimental Psychology, 1953,45, 197-203.

Brenner, J. & Goesling, W.J. Avoidance conditioning of activity and immobility in rats. Journal of Comparative and Physiological Psychology, 1970, 70, 276-280.

Brogden, W.J. Locus effects of single classical trials during extinction of avoidance CR's in rabbits (Oryctolagus Cuniculus) Journal of Comparative and Physiological Psychology, 1970, 70, 270-275.

Brookshire, K.H., Littman, R.A. & Stewart, C.N. Residua of shock trauma in the white rat; a three factor theory. Psychological Monographs, 1961, 75, (10, Whole No. 514).

Brown, J.S. & Jacobs, A. The role of fear in the motivation and acquisition of responses. Journal of Experimental Psychology, 1949, 39, 747-759.

Brown, J.S., Kalish, H.I. & Farber, I.E. Conditioned fear as revealed by magnitude of startle response to an auditory stimulus. Journal of Experimental Psychology, 1951, 41, 317-328.

Brown, R.V. Effects of stress on voluntary alcohol consumption in mice. Quarterly Journal of Studies in Alcohol, 1968, 29, 49-53.

Buckalew, L.W. & Cartwright, G.M. General and differential behavioral effects of five ethanol dosages on the Albino rat. Psychological Reports, 1968, 23, 1151-1154.

Bull, J.A. & Overmier, J.B. Transfer of control of avoidance is not dependent upon the maintenance of the original discriminative response. Proceedings of the 76th annual Convention of the American Psychological Association, 1968, 3, 143-144 (a).

Bull, J.A. & Overmier, J.B. Additive and subtractive properties of excitation and inhibition. Journal of Comparative and Physiological Psychology, 1968, 66, 511-514 (b).

Carlson, N.J. & Black, A.H. Traumatic avoidance learning; the effect of preventing escape response. Canadian Journal of Psychology, 1960, 14 21-28.

Cartwright, G.M. & Buckalew, L.W. Quantitative indices of alcohol effects on the rat. Psychonomic Science, 1969, 14, 140-141.

Casey, A. The effect of stress on the consumption of alcohol and reserpine. Quarterly Journal of Studies in Alcohol. 1960, 21, 208-216.

Clay, M.L. Conditions affecting voluntary alcohol consumption in rats. Quarterly Journal of Studies in Alcohol, 1964, 25, 36-55.

Cofer, C.N. & Appley, M.H. Motivation: Theory and Research. New York, Wiley, 1965.

Conger, J.J. The effect of alcohol on conflict behavior in the albino rat. Quarterly Journal of Studies in Alcohol, 1951, 12, 1-29.

Crow, L.T. The effects of alcohol on conditioned avoidance responding. Physiology and Behavior, 1966, 1, 89-91.

Desiderato, O. Generalization of acquired fear as a funtion of CS intensity and number of acquisition trials. Journal of Experimental Psychology, 1964, 67, 41-47.

Desiderato, O., Butler, B. & Meyer, C. Changes in fear generalization gradients as a function of delayed testing. Journal of Experimental Psychology, 1966, 72, 678-682.

DeToledo, L. & Black, A.H. Effects of preshock on subsequent avoidance learning. Journal of Comparative and Physiological Psychology, 1967, 63, 493-499.

Dinsmoor, J.A. & Campbell, S.L. Escape from shock training following exposure to inescapable shock. Psychological Reports, 1956, 2, 43-50. (a)

Dinsmoor, J.A. & Campbell, S.L. Levels of current and time between sessions as factors in adaptation to shock. Psychological Reports, 1956, 2, 441-444. (b)

Fazekas, I.G. Hydrocortisone content of human blood, and alcohol content of blood and urine, and wine consumption. Quarterly Journal of Studies in Alcohol, 1966, 27, 439-446.

Feather, N.T. Mowrers revised two factor theory and the motive expectance-value theory. Psychological Review, 1963, 70, 500-515.

Freed, E.X. The effects of alcohol on approach-avoidance conflict in the white rat. Quarterly Journal of Studies in Alcohol, 1967, 28, 236-253.

Freed, E.X. Effects of alcohol on conflict behavior. Psychological Reports, 1968, 23, 151-159. (a)

Freed, E.X. Effects of self intoxication upon approach-avoidance behavior in the rat. Quarterly Journal of Studies in Alcohol, 1968, 29, 323-329. (b)

Gilbert, R.M. Maintenance of a conditioned avoidance response in rabbits (Oryctolagus Cuniculus) through random presentations of classical trials. Journal of Comparative and Physiological Psychology, 1970, 70, 264-269.

Grossen, N.E. & Bolles, R.C. Effects of classical conditioned fear signal and safety signal on non-discriminated avoidance behavior. Psychonomic Science, 1968, 11, 321-322.

Grossman, S.P. & Miller, N.E. Control for stimulus change in the evaluation of alcohol and chlorpromazine as fear reducing drugs. Psychopharmacologia, 1961, 2, 342-351.

Harlow, H.F. The formation of learning sets. Psychological Review, 1949, 56, 51-65.

Herrnstein, R.J. Method and theory in the study of avoidance. Psychological Review, 1969, 76, 46-69.

Hull, C.L. A Behavior System, New York, Wiley, 1952.

Kalish, H.I. Strength of fear as a function of acquisition and extinction trials. Journal of Experimental Psychology, 1954, 47, 1-9.

Kamano, D.K. Effects of an extinguished fear stimulus on avoidance behavior. Psychonomic Science, 1968, 13, 271-272.

Kamin, L.J. The retention of an incompletely learned avoidance response. Journal of Comparative and Physiological Psychology, 1957, 50, 457-460. (a)

Kamin, L.J. The gradient of delay of secondary reward in avoidance learning tested on avoidance trials only. Journal of Comparative and Physiological Psychology, 1957, 50, 450-456. (b)

Kaplan, H.S. Effects of alcohol on fear extinction. Dissertation Abstracts, 1956, 55, 571-572.

Kent, N.D., Wagner, M.G. & Gannon, D.R. Effects of unconditioned response restrictions on subsequent acquisition of a habit motivated by fear. Psychological Reports, 1960, 60, 335-338.

Kubena, R.K. & Barry, H.III. Two procedures for training differential responses in alcohol and non-drug conditions. Journal of Pharmaceutical Science, 1969, 59, 99-101.

Kurtz, K.H. & Pearl, J. The effect of prior experience on acquired drive learning. Journal of Comparative and Physiological Psychology, 1960, 53, 214-219.

Majchrowicz, E., Lipton, M.A., Meek, J.L. & Hall, L. Effects of chronic ethanol consumption on the clearance of acutely administered ethanol and acetaldehyde from blood in rats. Quarterly Journal of Studies in Alcohol, 1968, 29, 353-357.

Martin, L.K. & Reiss, D. Effects of US intensity during previous discrete delay conditioning on conditioned acceleration during avoidance extinction. Journal of Comparative and Physiological Psychology, 1969, 67, 196-200.

Masserman, J.H., Jacques, M.G. & Nicholson, M.R. Alcohol as a preventative of experimental neurosis. Quarterly Journal of Studies in Alcohol, 1945, 6, 281-299.

Masserman, J.H. & Yum, K.S. An analysis of the influence of alcohol on experimental neurosis in cats. Psychosomatic Medicine, 1946, 8, 36-52.

McAllister, W.R. & McAllister, D.E. Post conditioning delay and intensity of shock as factors in the measurement of acquired fear. Journal of Experimental Psychology, 1962, 64, 110-116. (a)

McAllister, W.R. & McAllister, D.E. Role of the CS and of the apparatus cues in the measurement of acquired fear. Psychological Reports, 1962, 11, 749-756. (b)

McAllister, W.R. & McAllister, D.E. Increase over time in the stimulus generalization of acquired fear. Journal of Experimental Psychology, 1963, 65, 576-582.

McAllister, W.R. & McAllister, D.E. Variables influencing the conditioning and the measurement of acquired fear. In W.F. Prokasy (Ed.) Classical Conditioning: A Symposium. New York, Appleton Century Crofts, 1965, 172-191.

McAllister, W.R. & McAllister, D.E. Forgetting of acquired fear. Journal of Comparative and Physiological Psychology, 1968, 65, 352-355.

McMurray, G.A. & Jacques, L.B. The effects of drugs on a conditioned avoidance response. Canadian Journal of Psychology, 1959, 13, 186-192.

Miller, N.E. Studies of fear as an acquirable drive: Fear as motivation and fear reduction as reinforcement in the learning of new responses. Journal of Experimental Psychology, 1948, 38, 89-101.

Miller, N.E. Learnable drives and rewards. In S.S. Stevens (Ed.) Handbook of Experimental Psychology, New York, Wiley, 1951, 435-472.

Miller, N.E. Effects of drugs on motivation: The value of using a variety of measures. Annals of the New York Academy of Science, 1956, 65, 318-333.

Miller, N.E. Learning resistance to pain and fear: Effects of overlearning, exposure and reward exposure in context. Journal of Experimental Psychology, 1960, 60, 137-145.

Miller, N.E. Some recent studies on conflict behavior and drugs. American Psychologist, 1961, 16, 12-24.

Miller, N.E. & Barry, H. Motivational effects of drugs: Methods which illustrate some general problems in psychopharmacology. Psychopharmacologia, 1960, 1, 169-199.

Mowrer, O.H. Learning theory and Behavior. New York, Wiley, 1960.

Mowrer, O.H. A stimulus-response analysis of anxiety and its role as a reinforcing agent. In G.A. Kimble (Ed.) Foundations of Conditioning and Learning, New York, Appleton Century Crofts, 1967, 660-667.

Mullin, A.D. & Morgenson, G.H. Effects of fear conditioning on avoidance learning. Psychological Reports, 1963, 13, 707-710.

Overmier, J.B. Interference with avoidance behavior: Failure to escape traumatic shock. Journal of Experimental Psychology, 1968, 78, 340-343.

Overmier, J.B. & Leaf, R.C. Effects of discriminative Pavlovian fear conditioning upon previous or subsequently acquired avoidance responding. Journal of Comparative and Physiological Psychology, 1965, 60, 213-217.

Overmier, J.B. & Seligman, M.E.P. Effects of inescapable shock on subsequent escape and avoidance responding. Journal of Comparative and Physiological Psychology, 1967, 63, 28-33.

Overton, D.A. State dependent learning produced by depressants and atropine like drugs. Psychopharmacologia, 1966, 10, 6-31.

Overton, D.A. High Education, Psychology Today, 1969, November, 48-51.

Pawlowski, A.A., Denenberg, V.H. & Zarrow, M. Prolonged alcohol consumption in the rat; acquisition and extinction of an escape response. Quarterly Journal of Studies in Alcohol, 1961, 22, 232-240.

Perkins, C.C. & Weyant, R.G. The interval between training and test trials as a determiner of the slope of the generalization gradient. Journal of Comparative and Physiological Psychology, 1958, 51, 596-600.

Powell, B.J., Kamano, D.K. & Martin, L.K. Multiple factors affecting volitional consumption of alcohol in the Abrams Wistar rat. Quarterly Journal of Studies in Alcohol, 1966, 27, 7-15.

Rescorla, R.A. Predictability and number of pairings in Pavlovian fear conditioning. Psychonomic Science, 1966, 4, 383-384.

Rescorla, R.A. Inhibition of delay in Pavlovian fear conditioning. Journal of Comparative and Physiological Psychology, 1967, 64, 114-120.

Rescorla, R.A. Pavlovian conditioned fear in Sidman avoidance learning. Journal of Comparative and Physiological Psychology, 1968, 65, 55-60.

Rescorla, R.A. & LoLordo, V.M. Inhibitions of avoidance behavior. Journal of Comparative and Physiological Psychology, 1965, 59, 406-412.

Rescorla, R.A. & Solomon, R.L. Two process learning theory: relationships between Pavlovian and instrumental learning. Psychological Review, 1967, 74, 151-182.

Scarborough, B.B. Lasting effects of alcohol on the reduction of anxiety in rats. Journal of Genetic Psychology, 1957, 91, 173-179.

Seligman, M.E.P. & Maier, S.F. Failure to escape traumatic shock. Journal of Experimental Psychology, 1967, 74, 1-9.

Slotnick, B.M. Effects of fear conditioning on subsequent acquisition of an avoidance response. Psychonomic Science, 1968, 13, 159-160.

Smart, R.G. Effects of alcohol on conflict and avoidance behavior. Quarterly Journal of Studies in Alcohol, 1965, 26, 187-205.

Smith, J.A., Cohen, P.S. & Turner, L.M. Short term interference of inescapable shock upon acquisition of subsequent escape-avoidance responding. Proceedings of the 76th Annual Convention of the American Psychological Association, 1968, 3, 145-146.

Solomon, R.L. & Turner, L.H. Discriminative classical conditioning in dogs paralyzed by curare can later control discriminative avoidance responses in the normal state. Psychological Review, 1962, 69, 202-219.

Soloman, R.L. & Wynne, L.C. Traumatic avoidance learning: The principle of anxiety conservation and partial irreversability. Psychological Review, 1954, 61, 353-385.

Stewart, D.J. & Anisman, H. An automated one-way avoidance apparatus. Perceptual and Motor Skills, 1970, 31, 545-546.

Stewart, J. Differential responses based on the physiological consequences of pharmacological agents. Psychopharmacologia, 1962, 3, 132-138.

Thomas, D.R. & Lopez, L.J. The effects of delayed testing on generalization slope. Journal of Comparative and Physiological Psychology, 1962, 55, 541-544.

Thompson, C.P., Sachson, S.M. & Higgins, R.C. Distribution of intertrial responses in shuttle box avoidance conditioning. Journal of Comparative and Physiological Psychology, 1969, 69, 563-572.

Thor, D.H., Weisman, M.H. & Boshka, S.C. Preparations of alcohol solutions for behavioral research. Quarterly Journal of Studies in Alcohol, 1967, 28, 342-345.

Wallgren, H. & Savolainen, S. The effect of ethyl alcohol on a conditioned avoidance response in rats. Acta Pharmacologia et Toxicologia, 1962, 19, 59-67.

Walters, G.C. Frequency and intensity of preshock experience as determinants of fearfullness in approach avoidance conflict. Canadian Journal of Psychology, 1963, 17, 412-419.

Weisman, R.G. & Litner, J.S. Positive conditioned reinforcement of Sidman avoidance behavior in rats. Journal of Comparative and Physiological Psychology, 1969, 68, 597-603 (a).

Weisman, R.G. & Litner, J.S. The course of Pavlovian excitation and inhibition of fear in rats. Journal of Comparative and Physiological Psychology, 1969, 69, 667-672 (b).

Weiss, J.M., Krieckhaus, E.E. & Conte, R. Effects of fear conditioning on subsequent avoidance behavior and movement. Journal of Comparative and Physiological Psychology, 1968, 65, 413-421.

Winer, B.J. Statistical Principles in Experimental Design, New York, McGraw-Hill, 1962.

Zielenski, K. & Soltysik, S. The effects of pretraining on the acquisition and extinction of avoidance reflex. Acta Biologicae Experimentalis, 1964, 24, 73-87.

Appendix A

Table 1

Individual scores for trials to first avoidance (TFA), trials to three consecutive avoidances (TCA), trials to nine out of ten consecutive avoidances (NTA), reciprocal of latency over the first five (RL_1) and last five trials (RL_2), avoidances over blocks of thirty trials (BTT) and number of intertrial responses (ITR) for Ss in group PSS

Subject No.	Dependent variables								
	TFA	TCA	NTA	RL 1	RL 2	BTT 1	BTT 2	BTT 3	ITR
S_1	5	21	43	.201	.265	10	21	20	1
S_2	1	25	29	.204	.367	11	29	29	8
S_3	21	21	35	.135	.229	5	22	23	2
S_4	10	19	17	.176	.657	15	30	30	1
S_5	15	23	19	.148	.344	12	25	25	2
S_6	10	24	22	.156	.442	10	23	23	5
S_7	1	1	90	.520	.204	6	6	7	34
S_8	3	21	36	.209	.252	8	24	23	7
S_9	5	27	27	.210	.393	11	21	21	7
S_{10}	11	27	90	.168	.274	7	10	13	3
S_{11}	11	34	82	.177	.240	4	16	20	3
S_{12}	9	15	30	.163	.409	13	24	27	2

Appendix A

Table 2

Individual scores for trials to first avoidance (TFA), trials to three consecutive avoidances (TCA), trials to nine out of ten consecutive avoidances (NTA), reciprocal of latency over the first five (RL_1) and last five trials (RL_2), avoidances over blocks of thirty trials (BTT) and number of intertrial responses (ITR) for Ss in group PAS

Subject No.	Dependent variables								
	TFA	TCA	NTA	RL 1	RL 2	BTT 1	BTT 2	BTT 3	ITR
S_1	21	37	72	.152	.359	4	19	19	1
S_2	24	24	38	.140	.301	3	24	23	6
S_3	32	60	90	.063	.214	0	9	12	1
S_4	2	24	24	.170	.295	9	22	24	3
S_5	25	90	90	.158	.196	1	7	11	1
S_6	20	35	40	.109	.282	2	22	24	2
S_7	90	90	90	.134	.163	0	0	0	0
S_8	4	33	33	.187	.308	6	23	24	7
S_9	27	46	90	.099	.216	1	7	8	5
S_{10}	18	26	31	.143	.295	5	26	16	3
S_{11}	25	45	90	.147	.187	1	11	8	5
S_{12}	9	22	27	.143	.253	8	28	25	8

Appendix A

Table 3

Individual scores for trials to first avoidance (TFA), trials to three consecutive avoidances (TCA), trials to nine out of ten consecutive avoidances (NTA), reciprocal of latency over the first five (RL_1) and last five trials (RL_2), avoidances over blocks of thirty trials (BTT) and number of intertrial responses (ITR) for Ss in group PSA

Subject No.	Dependent variables								
	TFA	TCA	NTA	RL 1	RL 2	BTT 1	BTT 2	BTT 3	ITR
S_1	8	24	44	.176	.316	8	19	20	14
S_2	3	44	90	.162	.208	7	12	12	6
S_3	8	10	90	.138	.173	5	1	2	6
S_4	21	33	67	.155	.322	4	15	23	6
S_5	40	49	80	.165	.271	0	7	18	4
S_6	12	39	90	.181	.216	5	14	13	23
S_7	29	37	35	.159	.349	1	22	24	6
S_8	9	41	54	.164	.462	4	19	24	6
S_9	1	54	62	.246	.342	7	7	24	12
S_{10}	22	45	73	.148	.216	2	14	16	1
S_{11}	19	53	57	.117	.370	4	11	23	16
S_{12}	30	90	90	.150	.186	1	1	3	3

Appendix A

Table 4

Individual scores for trials to first avoidance (TFA), trials to three consecutive avoidances (TCA), trials to nine out of ten consecutive avoidances (NTA), reciprocal of latency over the first five (RL_1) and last five trials (RL_2), avoidances over blocks of thirty trials (BTT) and number of intertrial responses (ITR) for Ss in group PAA

Subject No.	Dependent variables								
	TFA	TCA	NTA	RL 1	RL 2	BTT 1	BTT 2	BTT 3	ITR
S_1	21	90	90	.156	.175	2	3	1	2
S_2	24	32	90	.157	.242	4	18	20	5
S_3	14	44	67	.154	.204	4	16	19	9
S_4	54	90	90	.066	.177	0	3	3	4
S_5	40	63	82	.156	.420	0	4	22	2
S_6	19	48	46	.157	.208	2	18	19	0
S_7	17	37	64	.160	.256	6	13	22	9
S_8	26	90	90	.130	.173	2	14	6	2
S_9	26	41	41	.181	.174	3	19	15	8
S_{10}	23	38	36	.151	.462	3	17	23	5
S_{11}	34	62	62	.167	.261	0	15	24	12
S_{12}	32	42	77	.158	.273	0	11	20	10

Appendix A

Table 5

Individual scores for trials to first avoidance (TFA), trials to three consecutive avoidances (TCA), trials to nine out of ten consecutive avoidances (NTA), reciprocal of latency over the first five (RL_1) and last five trials (RL_2), avoidances over blocks of thirty trials (BTT) and number of intertrial responses (ITR) for Ss in group NSS

Subject No.	Dependent variables								
	TFA	TCA	NTA	RL 1	RL 2	BTT 1	BTT 2	BTT 3	ITR
S_1	15	18	22	.171	.384	13	16	20	5
S_2	47	80	90	.155	.270	0	4	10	1
S_3	59	90	90	.178	.187	0	1	3	2
S_4	17	30	58	.179	.284	5	17	23	2
S_5	19	69	90	.151	.174	2	8	9	3
S_6	16	53	53	.156	.212	4	14	14	0
S_7	3	23	20	.165	.290	14	26	25	1
S_8	18	50	75	.149	.471	4	13	21	1
S_9	27	48	90	.082	.260	1	10	13	0
S_{10}	24	47	90	.154	.193	1	8	9	8
S_{11}	33	65	90	.146	.183	0	6	4	2
S_{12}	12	19	17	.169	.295	14	23	27	7

Appendix A

Table 6

Individual scores for trials to first avoidance (TFA), trials to three consecutive avoidances (TCA), trials to nine out of ten consecutive avoidances (NTA), reciprocal of latency over the first five (RL_1) and last five trials (RL_2), avoidances over blocks of thirty trials (BTT) and number of intertrial responses (ITR) for Ss in group NAS

Subject No.	Dependent variables								
	TFA	TCA	NTA	RL 1	RL 2	BTT 1	BTT 2	BTT 3	ITR
S_1	18	34	62	.170	.364	3	17	20	6
S_2	41	44	48	.184	.242	0	16	19	1
S_3	48	90	90	.111	.182	0	3	3	3
S_4	5	21	51	.176	.282	7	24	24	8
S_5	42	90	90	.154	.182	0	1	6	2
S_6	35	71	90	.136	.165	0	5	5	2
S_7	23	41	41	.167	.209	2	19	19	2
S_8	15	23	41	.158	.357	8	20	26	3
S_9	2	25	37	.163	.236	9	22	22	2
S_{10}	10	71	90	.163	.416	3	10	12	9
S_{11}	18	40	90	.107	.180	2	9	9	2
S_{12}	34	55	90	.158	.181	0	10	7	2

Appendix A

Table 7

Individual scores for trials to first avoidance (TFA), trials to three consecutive avoidances (TCA), trials to nine out of ten consecutive avoidances (NTA), reciprocal of latency over the first five (RL_1) and last five trials (RL_2), avoidances over blocks of thirty trials (BTT) and number of intertrial responses (ITR) for Ss in group NSA

Subject No.	Dependent variables								
	TFA	TCA	NTA	RL 1	RL 2	BTT 1	BTT 2	BTT 3	ITR
S_1	28	40	40	.173	.265	1	20	19	12
S_2	50	68	90	.158	.223	0	5	16	1
S_3	38	90	90	.171	.225	0	6	9	8
S_4	49	90	90	.116	.179	0	1	3	4
S_5	20	53	58	.132	.222	6	15	23	3
S_6	65	83	90	.168	.265	0	0	14	4
S_7	27	82	90	.157	.218	1	5	17	6
S_8	19	64	76	.162	.342	2	10	23	6
S_9	56	90	90	.158	.200	0	1	1	16
S_{10}	26	40	57	.161	.370	1	17	27	10
S_{11}	40	58	58	.151	.247	0	9	26	8
S_{12}	73	86	90	.166	.242	0	0	6	2

Appendix A

Table 8

Individual scores for trials to first avoidance (TFA), trials to three consecutive avoidances (TCA), trials to nine out of ten consecutive avoidances (NTA), reciprocal of latency over the first five (RL_1) and last five trials (RL_2), avoidances over blocks of thirty trials (BTT) and number of intertrial responses (ITR) for Ss in group NAA

Subject No.	Dependent variables								
	TFA	TCA	NTA	RL 1	RL 2	BTT 1	BTT 2	BTT 3	ITR
S_1	45	90	90	.158	.171	0	1	0	2
S_2	76	90	90	.168	.181	0	0	2	3
S_3	57	90	90	.166	.232	0	3	8	10
S_4	78	90	90	.125	.185	0	0	2	0
S_5	42	90	90	.113	.187	0	3	2	1
S_6	15	26	90	.168	.188	9	17	18	10
S_7	20	27	35	.163	.403	4	25	29	8
S_8	47	61	90	.158	.248	0	6	15	2
S_9	4	33	90	.191	.202	4	11	9	5
S_{10}	33	76	90	.137	.178	0	7	7	9
S_{11}	16	18	26	.148	.471	9	23	21	11
S_{12}	27	31	31	.169	.694	1	24	30	3

Appendix B

Table 1

Newman Keuls multiple comparisons between means for a prior training (P vs. N) x drug during prior training (A vs. S) interaction on trials to first avoidance

Treatments		PS	PA	NA	NS
	Totals	304	627	761	781
PS		---	323	457	477
PA			---	134	154
NA				---	20
NS					---

	r=2	r=3	r=4
$q_{.99}$ (r,88)	3.76	4.28	4.60
$\sqrt{nMS_{res}}\ q_{.99}$ (r,88)	302.34	344.15	369.88

	PS	PA	NA	NS
PS		**	**	**
PA				
NA				
NS				

** p .01

Appendix B

Table 2

Newman Keuls multiple comparisons between means for a prior training (P vs. N) x drug during prior training (A vs. S) interaction on trials to three consecutive avoidances

Treatments		PS	PA	NA	NS
	Totals	791	1186	1327	1435
PS	791	---	395	536	644
PA	1186		---	141	259
NA	1327			---	108
NS	1435				---

	r=2	r=3	r=4
$q_{.99}(r,88)$	3.76	4.28	4.60
$/\overline{nMS_{res}}\ q_{.99}(r,88)$	212.62	226.85	235.17

	PS	PA	NA	NS
PS		**	**	**
PA				
NA				
NS				

** p .01

Appendix B

Table 3

Newman Keuls multiple comparisons between means for a prior training (P vs. N) x drug during prior training (A vs. S) interaction on reciprocal of latency over the first five and last five trials

Treatments		PA	NS	NA	PS
	Totals	9.475	9.929	10.047	11.935
PA	9.475	---	.454	.572	2.460
NS	9.929		---	.118	2.006
NA	10.047			---	1.888
PS	11.935				---

	r=2	r=3	r=4
$q_{.99}$ (r,88)	3.76	4.28	4.60
$/\overline{nMSres}\ q_{.99}(r,88)$	1.425	1.622	1.743

	PA	NS	NA	PS
PA				**
NS				**
NA				**
PS				**

**p .01

Appendix B

Table 4

Newman Keuls multiple comparisons between means for a drug during avoidance training (A' vs. S') x blocks of trials interaction on number of avoidances over blocks of thirty trials

Treatments		A'1	S'1	A'2	A'3	S'2	S'3
	Totals	112	244	502	719	751	805
A'1	112	---	132	390	607	639	693
S'1	244		---	258	475	507	561
A'2	502			---	217	249	302
A'3	719				---	32	85
S'2	751					---	54
S'3	805						---

	r=2	r=3	r=4	r=5	r=6
$q_{.99}(r,176)$	3.70	4.20	4.50	4.71	4.87
$\sqrt{nMS_{res}}\, q_{.99}(r,176)$	109.96	124.82	133.74	139.98	144.73

	A'1	S'1	A'2	A'3	S'2	S'3
A'1		**	**	**	**	**
S'1			**	**	**	**
A'2				**	**	**
A'3						
S'2						
S'3						

** p .01

Appendix B

Table 5

Treatments	NA1	PA1	NS1	PS1	NS2	NA2	NA3	PA2	NS3	PA3	PS2	PS3
Totals	61	66	69	160	235	276	315	349	358	388	393	463
NA1 61	--	5	8	99	175	215	254	288	297	327	362	402
PA1 66		--	3	94	169	210	249	283	292	322	357	397
NS1 69			--	91	166	207	246	280	289	319	354	394
PS1 160				--	75	116	155	189	198	228	233	303
NS2 235					--	41	80	114	123	153	158	228
NA2 276						--	39	77	86	112	121	187
NA3 315							--	34	43	73	78	148
PA2 349								--	9	39	44	114
NS3 358									--	30	35	105
PA3 388										--	5	75
PS2 393											--	70
PS3 463												--

	r=2	r=3	r=4	r=5	r=6	r=7
$q_{.99}(r,176)$	3.70	4.20	4.50	4.71	4.87	5.01
$\sqrt{nMS\ res}\ q_{.99}$	77.77	88.28	94.59	99.00	102.36	105.31
	r=8	r=9	r=10	r=11	r=12	r=13
$q_{.99}(r,176)$	5.12	5.21	5.30	5.38	5.44	5.51
$\sqrt{nMS\ res}(r,176)$	107.62	109.51	111.40	113.08	114.34	115.82

	NA1	PA1	NS1	PS1	NS2	NA2	NA3	PA2	NS3	PA3	PS2	PS3
NA1				**	**	**	**	**	**	**	**	**
PA1				**	**	**	**	**	**	**	**	**
NS1				**	**	**	**	**	**	**	**	**
PS1						**	**	**	**	**	**	**
NS2							**	**	**	**	**	**
NA2										**	**	**
NA3												**
PA2												
NS3												
PA3												
PS2												
PS3												