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# The Effect of Inescapable Shock on Competitive Dominance in Rats

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# THE EFFECT OF INESCAPABLE SHOCK ON COMPETITIVE DOMINANCE IN RATS

by

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ABSTRACT

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Experimental examination of the generality of learned helplessness has previously been confined to treatment and tests employing aversive motivators, such as electric shock. In the present study, rats were used to evaluate the effect of inescapable shock on their performance in a water test of competitive dominance which employs no aversive motivator. The subjects were paired and pre-tested for competitive dominance. In the experimental groups one member of each pair was treated with inescapable shock and the pairs were then post-tested for competitive dominance either 48, 72, or 168 hours after treatment. The control subjects were pre- and post-tested with no treatment intervening. Competitive dominance ranks were assigned to subjects after each test. Rank differences from pre- to post-test were analyzed for treated and control subjects. Controls showed no shifts in dominance from pre- to post-test, while significant shifts toward subordination appeared in all three experimental groups. No treated subjects showed shifts toward dominance. Significant pre- to post-test differences in drinking time

were produced in all treatment groups, with the greatest difference at 72 hours after treatment. These results parallel those of Glazer and Weiss (1976) for escape time latencies at different times of post-treatment testing. The results of the present study, in contrast to those of Glazer and Weiss, cannot be accounted for by the principles of stimulus control. Instead, they support the claim of Maier and Seligman (1976) for considerable generality to the effect of learned helplessness, though the generality observed in this study is not explained by current principles of learning theory.

(60 pages)

# INTRODUCTION

Recently a distinction has been observed in the behavioral consequences of exposure to controllable vs. uncontrollable aversive events. Animals exposed to uncontrollable trauma in the form of inescapable shock will later fail to learn to escape or avoid shock. The principal theoretical account for this deficit in escape response acquisition is called Learned Helplessness (Maier & Seligman, 1976). It is both the first and, by now, the most elaborate theory to be advanced. Though several challenges to learned helplessness have appeared, the latest and most viable is Glazer and Weiss' (1976a,b) Learned Inactivity hypothesis. Learned helplessness is basically a cognitive account of the deficit, while learned inactivity is an S-R account. I will clarify this difference between the two theories in the following review, and summarize the characteristics of the research used to test them.

## Learned Helplessness

Since 1976, Maier, Seligman and their colleagues have been compiling evidence to support and elaborate their theory of learned helplessness (reviewed in Maier & Seligman, 1976). The theory derives from an observed deficit in escape and avoidance response acquisition in subjects pre-treated with unpredictable and inescapable shock. Initial research was with dogs (Overmier and Seligman, 1966;

Seligman & Maier, 1967) and has more recently employed rats, but other species have also been used (i.e. cats, fish and humans--reviewed in Maier & Seligman, 1976). In the nonhuman research, experimental subjects are typically pre-treated with inescapable shock and then tested for escape response acquisition with a signalled shock stimulus as the aversive motivator.

The learned helplessness theory states that subjects exposed to uncontrollable aversive events learn non-contingency between responding and reinforcement, or response-outcome independence (Maier & Seligman, 1976). They learn that they are helpless. Learning response-outcome independence is proposed as a new aspect in learning theory (Maier & Testa, 1975) and is distinguished from learning that responses and reinforcement are either positively or negatively correlated. Learned helplessness requires that the subject learn non-correlation between responding and reinforcement (Maier & Seligman, 1976).

Maier and Seligman consider that learning response-outcome independence produces three distinct deficits, each with its own behavioral characteristics. They argue for a cognitive deficit, a motivational deficit, and an emotional deficit resulting from exposure to uncontrollable aversive events (Maier & Seligman, 1967). The cognitive deficit is also referred to as "cognitive" or "associative" interference. It is explained as follows: Having learned non-contingency between responding and reinforcement, the subject then fails to learn a contingency when he encounters

it--when he accidentally performs the escape response and terminates the shock. Prior learning of noncontingency interferes with subsequent learning of a contingency between responding and reinforcement. The evidence for this consequence of inescapable shock treatment is either relatively constant or increasing mean latencies to escape over trials during post-treatment escape testing, and the fact that during testing occasional performances of the escape responses were not systematically repeated (Maier & Seligman, 1976).

The second behavioral consequence in support of learned helplessness is hypothesized to be a motivational deficit. Having learned that they are helpless, subjects then fail to even initiate responses in an attempt to escape. These responses are required in order for the animal to finally produce the escape response by chance and thus encounter the contingency between responding and shock termination. Beyond long latencies to escape, evidence for this effect derives from the experimenters' anecdotal reports of the over-all inactivity of treated subjects compared to unshocked or escapably shocked controls (Maier & Seligman, 1976).

The third aspect is an emotional deficit. Inescapably shocked subjects show an emotional imbalance after treatment, and will show signs of depression or anxiety. Evidence for this effect ranges across a variety of studies, most conducted outside the learned helplessness literature (Maier & Seligman, 1976). Jay Weiss (1968) used physiological measures to demonstrate that inescapable shock

is a more severe stressor than escapable shock. Inescapably shocked rats showed greater weight loss, decreased appetite and increased urination and defication, as well as increased stomach ulceration in comparison to both inescapably shocked and unshocked controls. From this and other evidence, Maier and Seligman (1976) conclude that there exists an emotional disruption or deficit as an effect of exposure to uncontrollable aversive events.

The learned helplessness theory posits that it is the uncontrollability of the aversive event that produces learned helplessness, and that within unspecified limits the specific parameters of inescapable shock treatment are unimportant (Maier & Seligman, 1976). Maier, Albin and Testa (1973) have provided at best a cursory and narrow examination of the parameters of the shock treatment. Maier has generally used a 1mA, 5-second long unsignalled shock delivered on a variable time 60-second schedule over one hour (60 shocks) as his treatment procedure for rats. The shock is delivered through tail electrodes while the rat is restrained in a plexiglass tube. Seligman (Seligman & Beagley, 1975) uses a different treatment procedure for rats. Inescapably shocked rats are yoked to escapably shocked subjects, and shock is delivered through a pin electrode in the subject's back so that he completes a circuit between the pin and the floor grid on which he stands. The shock duration is a maximum of 10 seconds, and is of 1mA intensity. Shock is delivered unsignalled for 80 trials on a VT 60" schedule. Thus treatment lasts approximately 80 minutes. These differences in parameters of shock treatment appear to generate no differences in results.

Maier and Seligman claim considerable generality for the effects of inescapable shock treatment. For example, wide species generality has been demonstrated. The response-outcome independence supposedly learned during treatment is transferred to tests using aversive motivators other than shock (Rosellini & Seligman, 1975). There is some evidence that experiencing uncontrollable aversive events will affect nonaversively motivated behaviors, such as human problem-solving behavior (Hiroto & Seligman, 1975). But such evidence is sparse, and one may question whether the effect is produced by learning response-outcome independence per se, or some other effect of the procedure. One must also question on what basis such transfer is made, since it is not encompassed by learning principles of response or stimulus generalization. Experimental examination of trans-situational generality of the effect of shock treatment is as yet sparse and has usually not assessed time course or durability of the effect.

A recent exception to this observation (Anderson, Crowell, Koehn & Lupo, 1976) has added a dimension to the generality of exposure to inescapable shock. In this study, Anderson used two levels of shock intensity, 1.25mA and 4.0mA, and a different treatment and test procedure than that used by proponents of either learned helplessness or learned inactivity. They examined the effect of inescapable shock on rats' open field exploration. Their treatment consisted of five sessions, on consecutive days, of individual exposure to three minutes of constantly on unsignalled inescapable scrambled grid shock. Subjects were first tested one week after treatment.

Each test was three minutes of free ranging in an open field. Nine post-treatment tests were run in blocks of one each day for four days, five days off, a four-day block of tests, then five days off again, then a final single test. The high-shock subjects showed the greatest effects on open-field behavior, but the effect dissipated over test blocks to below that of the low-shock group. The low-shock group showed a smaller effect but it was constant across tests, meaning it did not dissipate with time. The effect for the low-shock group compared with non-shocked controls, however consistent, only approached statistical significance. Shocked subjects showed less grid lines crossed, less rearing, more wall hugging and more defication than unshocked controls in the open field.

Recent experimental attention has focused on the time course of the escape response acquisition deficit produced by inescapable shock (Hannum, Rosellini & Seligman, 1976) and no upper limit has been found. However, these experimenters treated very young rats and tested them last when the subjects were six months of age. Whether the same time course would hold for subjects treated when older, or for different species remains to be demonstrated. Maier has reported that the effect is transitory in mongrel dogs obtained from the dog pound (apparent 24 hours after treatment but not 48 hours after), but that kennel raised beagles will show deficits which will last at least a week, though later tests were not made (Maier and Seligman, 1976).

Procedures to immunize against, or reverse the effect of inescapable shock have centered exclusively on subsequent escape response acquisition (Williams & Maier, 1977; Seligman, Rosellini & Kozak, 1975). No attention has as yet been directed toward the effects of immunization or "therapy" (reversal) procedures on other behaviors that have been shown to be affected by inescapable shock. Immunization treatment has consisted of pre-training an escape response to shock, then treating with inescapable shock, and finally testing for performance of the same response (Seligman, et al., 1975) or acquisition of a different response (Williams & Maier, 1977). Therapy for the deficit has consisted of forced shaping, referred to as "drag training" (Seligman, et al., 1975). Here the rat is physically dragged by the pin electrode in its back onto the lever for performance of each of the lever presses of the FR3 escape response. Though crude, this procedure has proven effective in removing the escape response deficit.

In summary, learned helplessness is a theory based primarily on an escape response deficit observed in subjects ranging from rats to man. Learned helplessness is thought to be the result of learning response-outcome independence and occurs after exposure to uncontrollable aversive events. Its proponents claim that the parameters of treatment are relatively unimportant; it is uncontrollability per se that is the essential aspect. The effect is thought to consist of three different distinct deficits; cognitive, motivational and emotional.

Learned Inactivity

Glazer and Weiss (1976a,b) have proposed that during shock treatment, instead of learning to be helpless, the subjects learn to be inactive. This is because their greater tendency for inactivity at the time of shock termination, which increases over trials, is paired with shock termination. This learned inactivity then interferes with subsequent escape response acquisition. Learned inactivity is thus essentially a competing response hypothesis, similar to other S-R accounts of the phenomenon. Learned inactivity identifies a characteristic of the competing response--inactivity. Other competing response hypotheses have identified the response itself. For example, Bracewell and Black (1974) hypothesized that freezing was conditioned during treatment because movement was explicitly punished. Anisman and Waller (1973) have also cited freezing, which is paired with shock termination, as a learned competing response which interferes with subsequent escape response acquisition.

Evidence in support of the learned inactivity hypothesis is presented in two experiments by Glazer and Weiss (1976a,b). The first experiment presents evidence against the lack of parametric constraints on the shock treatment proposed by Maier and Seligman (1976), and on their failure to impose any constraints or delineations on the time-course of the phenomenon. A second experiment demonstrates that a certain type of escape response (an inactive response) will in fact be acquired after inescapable shock treatment while other types, which

require active responding are not acquired. This is a prediction made specifically by the learned inactivity hypothesis and not by the learned helplessness hypothesis, which would predict that the type of escape response used would make no difference as long as it is learned and not elicited by shock, since learning response-outcome independence interferes with escape response acquisition. It is this last evidence which distinguishes Glazer and Weiss' account and makes it particularly damaging to the learned helplessness hypothesis.

In their first experiment, Glazer and Weiss (1976a) partially replicated Maier, Albin and Testa (1973). They used rats as subjects and the same treatment procedure as Maier, et al. (1973) except with a variable shock duration. Maier used a five-second duration, whereas Glazer and Weiss used 2, 3, 4, 5, and 6-second shock durations while concurrently controlling for overall duration of shock by varying session length of different treatment groups. They found the escape deficit was only produced after treatment with shock durations of five and six seconds, and the deficit was greater for subjects shocked for six seconds. The learned helplessness hypothesis does not account for these results, since it stipulates that lack of control over the aversive event is both necessary and sufficient to produce an escape response deficit, if the subject experiences sufficient trials to learn noncontingency between responding and reinforcement (Maier & Seligman, 1976).

Glazer and Weiss (1976a) also found a distinct time-course to the deficit. They post-tested different groups treated with six-second shocks either 30 minutes, 24 hours, 72 hours, or one week after treatment. No escape deficit was found in subjects tested 30 minutes after treatment, but the deficit was apparent 24 hours after treatment, at a maximum 72 hours after treatment, and had begun to decline one week after treatment. They offered no explanation of this time course. Proponents of learned helplessness have made no delineations of time course other than to indicate that the deficit is apparent 24 and 48 hours after treatment in most subjects, and is apparent in rats 90 days after treatment when treatment occurred just after weaning (Hannum, Rosellini & Seligman, 1977). This suggests that proponents of learned helplessness are perhaps overconfident in generalizing no limit to the effect of treatment from their quite limited experimental results.

In their second experiment, Glazer and Weiss (1976b) provide an elegant and strong test of learned helplessness vs. learned inactivity. They treated rats with six-second shock on a variable time 60-second schedule (VT60") and compared escape response acquisition for different types of escape responses. They compared acquisition of a low-activity nosing response with acquisition of the responses which have shown deficits in the learned helplessness literature--i.e., FR2 shuttle (Maier, Albin & Tests, 1973), FR3 lever press (Seligman & Beagley, 1975), and CRF barrier cross (Weiss and Glazer, 1975; Glazer and Weiss, 1976a).

The last three responses are classified by the authors as high-activity responses which would be incompatible with learned inactivity. These three responses all showed an escape response acquisition deficit during testing 72 hours after treatment, while the nosing response was acuired more quickly by the preshocked subjects than by unshocked controls. In the same study, Glazer and Weiss also demonstrated that nosing is actually acquired more quickly by yoked inescapably shocked subjects than by the escapably shocked subjects to which they are yoked. The learned inactivity hypothesis would predict exactly these results, since inactivity would compete only with acquisition of active responses but would actually facilitate acquisition of an inactive response. Learned helplessness, on the other hand, would predict no difference in response acquisition since the learning of noncontingency during treatment would interfere with acquisition of any escape response during testing. Learned helplessness proponents might counter that nosing responses are elicited by shock, but the learning curve (declining latencies over trials) demonstrated by these subjects definitely indicated acquisition and no prior learning of noncontingency between responding and reinforcement.

Learned inactivity thus accounts for the behavioral deficits which have been used by learned helplessness proponents to support both the associative interference effect or the cognitive deficit, and the motivational deficit aspects of their hypothesis. Subjects both fail

to show declining latencies to escape even when they do often eventually escape, and also fail to initiate active responding in an attempt to escape because learned inactivity acquired during treatment is competing with such responses.

Learned inactivity is proposed only to account for deficits in the acquisition of active escape responses after treatment with inescapable shock. But this hypoethesis cannot account for trans-situational generality of the effect of inescapable shock treatment like that observed by Anderson, et al. (1976) on the open-field exploration of rats. Glazer and Weiss do not assert that only inactivity is learned during treatment. But what other effect of treatment could account for effects on behaviors not motivated by shock? How widespread is such generality? What sorts of other behaviors are affected by exposure to inescapable shock and what kind of effect does this treatment produce? We do not know on what basis transfer between treatment and test in Anderson's study (1976) could be achieved, since treatment and test conditions and environments are so dissimilar. Perhaps the rats are learning inactivity during treatment, since Anderson's procedure fulfills the minimum shock duration requirement outlined by Glazer and Weiss (1976a). But there must be some other change effected during treatment which produces differences in a range of behaviors not connected with escape from shock. In another study concerning generality of the effect of shock treatment, Rosellini and Seligman (1975) found that treated rats behaved differently than

unshocked controls in a goal box during extinction--they showed longer latencies to escape the goal box. There is little similarity between tasks, environments, or reinforcers involved with a food goal box and a shock chamber. So it appears that principles of generalization cannot account for such transfer.

The treatment parameters used by Glazer and Weiss (1976a,b) and Maier, et al. (1973) have not specifically been tested for trans-situational generality. In the present study this is done with regard to a class of behaviors as yet unexamined for generality of the effect of shock treatment. It is asked here if inescapable shock treatment will effect changes in a social behavior--competitive dominance within pairs of rats. We are interested in what effect shock will have and whether the magnitude of the effect will parallel, by any chance, that found by Glazer and Weiss (1976b), i.e., peak at 72 hours after treatment.

Since Glazer and Weiss' learned inactivity hypothesis predicts no generality of treatment to social behaviors, I have partially replicated their treatment procedures and testing times to determine whether such generality of effect might be produced from these procedures. A competing response hypothesis cannot account for generality beyond what might be explained by learning principles of generalization. If such "unexplained" generality of shock treatment on competitive dominance is produced, this would indicate that something more than learned inactivity is affected by treatment.

In this study then, pairs of rats are tested for shifts in competitive dominance from pre to post-test after exposure of one member of the pair to inescapable shock. Before continuing further with aspects of the present study, I will briefly discuss relevant issues from research on competitive dominance in rats.

## Competitive Dominance

In general, research on dominance has been characterized by two problems. First, dominance measures have been both unstable across tests and difficult to define. And second, laboratory (competitive) measures of dominance have not correlated well with ethological measures (Syme, 1974). Because of these problems, research on dominance in rats has not yielded a unidimensional concept of dominance, i.e., "that there is one basic social order through which all of a group's resources are regulated" (Syme, 1974). So there is some confusion in the literature regarding just what dominance is. In this study, I do not attempt to answer such questions, but merely use the competitive dominance model as a convenient means of examining the effect of a treatment on a form of social responding. Competitive dominance has not typically been used in this way (Syme, Pollard, Syme & Reid, 1974). Test-retest designs have been confined to looking for procedures which will produce stability or correlations between one measure of dominance and another (i.e., aggressive orders, competitive orders, and grooming orders).

Dominance involves competition between two or more animals over a variety of resources such as food, water, territory, proximity to a receptive member of the opposite sex, etc. In some cases dominance will be established between a pair of animals with regard to at least one resource. One animal will emerge as dominant according to operationally defined criteria, and the other will be identified as subordinate. In other cases, clear dominance will not emerge as the pair continues to compete for the resource. At any given time, degree of deprivation may vary for different resources, and it is likely that different degrees of deprivation result in different dominance relations. An animal that is subordinate to another in a water competition situation after 24 hours of water deprivation, may well not be so after 48 hours of deprivation, or after 72 hours. I'm suggesting that as the subordinate animal becomes more deprived for a life-sustaining resource, i.e., as the resource becomes more valuable to him, he may not remain subordinate. Analogously, an animal that is deprived more for territory than for food may be subordinate in a food competition situation, but may actively compete for and gain territory. This might explain both the frequent failures to obtain test-retest reliability (stability) and the failure of ethological and laboratory measures to correlate. There is some experimental evidence that levels of deprivation will not affect test stability, but the levels of deprivation examined in this study were an insufficient sample (Rushkin & Corman, 1971). In the present study I don't directly test this question. Such an hypothesis has merely directed the formation of procedures used to establish competitive dominance

relations between pairs of rats that will be stable across tests over time.

Competitive dominance (for food or water) is the standard laboratory measure of dominance in rats (Syme, 1974). In a review of social dominance Van Kreveld (1970, cited in Syme, 1974) defines dominance as a "priority of access to an approach situation or away from an avoidance situation that one animal has over another." In this study, priority of access to water after a consistent level of water deprivation is used to determine dominance.

In order to test the effect of inescapable shock on competitive dominance in rats, a measure of dominance is required within animals which is stable over time. This has not been reported in the literature for rats to date. One of the objectives of this experiment was to derive a procedure which would yield a stable dominance measure against which the effects of exposure to inescapable shock can be assessed.

If a stable dominance relationship can be produced across a number of tests by controlling the level of deprivation and by pair-housing the animals, then this result alone would suggest further examination of the deprivation hypothesis of dominance stability. A stable dominance relation from these procedures would suggest that dominance is in part a function of deprivation level for the resource used.

The working hypothesis of this study is that inescapable shock produces a disruptive and degenerative effect on a variety of responses,

such that animals exposed to inescapable shock will show subsequent dominance shifts toward subordination. I consider that learned inactivity is an inadequate account of the effect of inescapable shock treatment, because it is too restricted and accounts only for the effect of treatment on subsequent escape response acquisition. I have, therefore, been specifically interested in testing for different degrees or magnitudes of effect of shock treatment on competitive dominance as a function of time between treatment and post-test. Glazer and Weiss (1976a) found deficits in escape response acquisition were greatest 72 hours after shock treatment, and less at 24 hours and one week after treatment, respectively. Finding differences in competitive dominance after inescapable shock treatment parallel to those for escape response acquisition, indicates that the same time course holds for a generalized effect of treatment as for the specific escape response deficit. So the first aspect of my working hypothesis has been that treating rats with inescapable shock will effect a shift toward subordination in their competitive dominance behavior. The second aspect is that the magnitude of this shift will parallel the magnitude of escape response acquisition deficits found by Glazer and Weiss (1976a) for different post-treatment test times.

It should be noted briefly that a pre-test, treatment, post-test design is to be used with subjects paired with the same animals throughout. Pretesting can be considered analogous to the pretraining used by Hannum, Rossellini and Seligman (1976), and by Williams and Maier

(1977) to prevent an effect of inescapable shock treatment on subsequent escape response acquisition. They have called this pre-training procedure "immunization." In these two studies, the immunization procedure was effective in preventing the escape response deficit or learned helplessness. Their results would argue against a finding of significant shifts in dominance from pre-test to post-test. The learned helplessness hypothesis, itself, however, does not directly argue against a dominance shift. The learned inactivity hypothesis, on the other hand, does not predict it since a general effect on competitive dominance is not within the range of this hypothesis' focus.

## METHODS

### Subjects

Forty male Sprague-Dawley rats purchased from Simonsen Laboratories were used. They were from six to seven months old when experimental procedures began, and had a mean weight of 582 grams with a range of 456-624 grams.

### Apparatus

The subjects were individually housed on standard rodent racks in hanging nine by six by six inch metal cages. These cages have sheet metal sides and backs with wire mesh fronts and floors. During pair-housing (see procedure) subjects occupied double-wide (nine by 13.5 by six inch) metal cages of the same construction and design. Both single

and double-wide cages were fitted with a single water bottle and tube when the subjects were not on water deprivation.

Competitive dominance testing was done in a 10 by 12 by 12 inch (h) glass box with one plywood wall and a particle board ceiling. An AC 120V, 6W house light was mounted in the center of the ceiling and was on continuously while subjects were in the chamber. An oval hole one and one-half inches high and one inch wide was centered in the plywood wall three inches from the floor. This hole allowed access by only one rat at a time to a water bottle drinking tube mounted behind the wall. The end of this tube was centered with regard to the hole and recessed one-half inch behind the back surface of the wall. The glass floor of the chamber was covered by approximately one and one-half inch of wood shavings.

An earlier study indicated that the recessed drinking tube was necessary to prevent shared drinking (both animals drinking simultaneously from a single water tube).

A one-half inch plywood panel between the oval access hole and the drinking tube could be removed or inserted manually to allow or block access to the drinking tube.

Shock treatment was given in a seven by eight by eight inch (h) floor grid shock delivery chamber. The chamber had plexiglass walls and roof with metal front and back walls. The floor was a grid of three-eighth inch metal rods through which scrambled shock could be delivered. A response lever and two stimulus lights in the front

metal wall were disconnected during treatment. The chamber was illuminated by a house light while occupied. Shock was delivered to the chamber by means of a Coulborn Instruments shock scrambler.

Procedure

Subjects were individually housed while learning to drink from the recessed drinking tube in the test chamber. Each was water deprived 24 hours prior to the first exposure to the drink chamber, and during the two days of shaping received water only in the drink chamber. Shaping of this behavior consisted of placing the animals individually in the drink chamber for one hour on two consecutive days. The experimenter determined how much water was consumed after each animal was removed from the chamber. A criterion of at least a .5 centimeter drop in water level (approximately 1.5 ounces) on either of the two sessions was required. Subjects not meeting criterion (all did) in the two sessions would have been dropped from the experiment.

An advantage of the foregoing procedure was that all subjects received equal time for adaptation to the chamber during the two hours of shaping. This insured that chamber exploring behavior was minimized for all subjects during the subsequent timed competitive dominance tests. The preliminary study also indicated that 24 hours of water deprivation was not stringent enough to preclude considerable chamber exploring when a ten-minute shaping procedure was used. In a timed test, these competing behaviors precluded an adequate assessment of competitive dominance.

The present study employed a small group design composed of an untreated control group and three experimental groups. Members of the experimental groups received the same shock treatment, but groups differed on the basis of time between treatment and post-test.

This design required that a stable dominance relation exist between paired subjects prior to treatment and that this stable relation be demonstrated in the untreated control group. To achieve such stability, subjects were pair-housed for eight consecutive days and tested twice for competitive dominance during this time, first on day five, and again on day eight of pair housing. Preliminary work indicated that dominance relationships between pairs of rats who met only in the drink chamber were not stable over time. Such subjects expended a good deal of the test time engaging in social responses. These responses gave no clear indication of dominance and competed with drinking and displacing one another at the tube access.

It was determined that pair-housing would allow social responses to largely dissipate and that a dominance relationship could be established which would then merely be tested for in the drink chamber rather than established there, per se. Since the first competitive drinking test was each pair's first experience with competing for access to a stimulus for which they had been deprived, this test was used to help establish a dominance hierarchy within each pair, and stability for all pairs between the first and second pre-tests was not expected.

Subjects were randomly assigned into pairs which then remained constant throughout the experiment. Five pairs were assigned to each

of four groups. Data for treatment effects were obtained from only one member of each pair, yielding five actual subjects per group.

Table 1 presents a flow chart of the test and treatment sequences for the four groups. The sequence runs from pair-housing to the post-test. All subjects were pair-housed for eight consecutive days. On day four they were placed on water deprivation and given the first competitive dominance test on day five, and a second test on day eight. Dominance from this point (day eight) through subsequent tests was stable in the control group. Hence the measure of dominance was considered adequate.

After completion of the second dominance test, subjects were returned to individual cages and no longer pair-housed. Shock treatment was administered two days after the second test. Since untreated animals might have injured or killed the treated subjects had they been pair-housed following treatment, the pairs were separated after the second test. It was of interest to examine the effects of treatment on competitive dominance after experimental subjects had fully recovered from the immediate and gross physiological trauma of treatment. Subjects were to encounter one another in a third paired test for the first time since the second test, and following treatment for one member of the pair. Such a procedure allowed for any shift in dominance to occur during the test itself, while the subjects were under observation.

The first experimental group, E-48, was tested 48 hours after treatment. The second group, E-72, was post-tested 72 hours after

Table 1

Testing and Treatment Sequence

| Control | Day | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Housing | P | P | P | P | P | P | P | P/S | S | S | S | S | | | | | |
| | Treatment | | | | on dep | T-1 | | on dep | T-2 | | | on dep | T-3 | | | | | |
| | | | | | | | | | | | | | | | | | | |
| E-48 | Day | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | | | | | |
| | Housing | P | P | P | P | P | P | P | P/S | S | S | S | S | | | | | |
| | Treatment | | | | on dep | T-1 | | on dep | T-2 | | | on dep | T-3 | | | | | |
| | | | | | | | | | | | | | | | | | | |
| E-72 | Day | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | | | | |
| | Housing | P | P | P | P | P | P | P | P/S | S | S | S | S | S | | | | |
| | Treatment | | | | on dep | T-1 | | on dep | T-2 | | Tr | | on dep | T-3 | | | | |
| | | | | | | | | | | | | | | | | | | |
| E-168 | Day | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 |
| | Housing | P | P | P | P | P | P | P | P/S | S | S | S | S | S | S | S | S | S |
| | Treatment | | | | on dep | T-1 | | on dep | T-2 | | Tr | | | | | | on dep | T-3 |

Groups: Control, E-48, E-72, and E-168

Symbols: P--pair housed
S--singly housed
P/S--pair housed before the test, singly housed after
on dep--put on water deprivation
T-1 through 3--competitive dominance tests
Tr--shock treatment

treatment. The third group, E-168, was tested one week (168 hours) after treatment. The testing schedule for the control group paralleled that of the 48-hour treatment group.

Competitive dominance drink tests lasted five minutes. As water-deprived subjects drink in this situation they approach satiation. Their motivation to drink and to compete for access to the recessed tube declines as they drink. Previous work indicated that 24-hour deprived rats in their home cages would drink steadily for four to six minutes without moving from the water tube. The drink test time of five minutes, was, therefore, selected as providing sufficient time to assess dominance between two deprived animals without allowing the animals to satiate during the test.

Dominance was defined by a combination of two measures, neither of which was considered adequate to determine dominance when used alone. These measures were total drinking time and number of displacements, and were scored by an observer for each subject in the pair as the test proceeded.

Drink time was recorded whenever a subject had his nose in the water tube access hole. Cumulative running timers were activated by the observer depressing a separate switch for each subject. Occasionally a subject would have his nose in the access hole without drinking, but this occurred rarely. Nose-in-the-hole indicated control of access to the water, and in a competitive dominance test with limited access to the resource, control of access is more at issue than how much water

is consumed (Van Kreveld, 1970). Since pausing was insignificant, nose-in-the-hole time is referred to as drink time rather than a more unwieldy and potentially more complicated control-of-access time.

Displacements were scored whenever one subject removed the other from the access hole. Displacements were occasionally caused simply by the approach of the non-drinking subject. Hence the following definition and criteria were used for scoring a displacement. A displacement consisted of one subject physically removing (not simply replacing) another from the access hole. The displacer had to:

1. either make physical contact with the subject having access control, or his head had to approach to within one centimeter of the head or shoulders of the drinking subject who had then to remove his head from the hole at this approach or contact, and
2. either the displacer had to start drinking or the displaced subject had to remain with his nose out of the access hole for a minimum of ten seconds.

This definition is required in order to distinguish displacements from simple alternation drinking between subjects, and from pauses and unsuccessful attempts at displacement. Displacements were scored (recorded) for the displacer.

When they occurred, displacements were used to determine the dominance ranking of the subjects in a pair. Subjects were scored as dominant if they displaced but were not themselves displaced. If both

subjects displaced each other at least once, they were scored as contested. If a subject was displaced, but didn't himself displace, he was then scored as subordinate. When no displacement occurred, subjects were scored as dominant, contested, or subordinate on the basis of drink times. An animal was scored as dominant if his drink time was at least twice as great as his pair-mate's. The subordinate subject, therefore, had to have a time one-half of his mate's, or less. Times which fell between these ratios resulted in the subject being assigned contested scores.

Ranked scores were assigned to these designations as follows: dominance = 1, contested = 2, and subordinate = 3. Each subject was given a dominance score from each competitive drink test. It was then possible to compute shifts in dominance between tests for each subject. Different scores were obtained by subtracting the ranked score on a later test from the ranked score of an earlier test for each subject. Table 2 gives an example of possible difference scores and what they would indicate about a dominance shift.

Table 2

Possible Difference Scores

| Score | Dominance Shift Indicated |
|---|---|
| 0 | No shift--no difference between tests. |
| -1 | A shift from dominant to contested, or from contested to subordinate (1→2 or 2→3). |
| -2 | A shift from dominant on the first test to subordinate on the second test (1→3). |
| +1 | A shift from contested to dominant (2→1). |

Only data from treated rats and analogous control subjects was used in the rank shifts analysis. Since treated subjects were either ranked dominant (1) or contested (2), the above table includes all possible difference scores (shifts from ranked scores of either 1 or 2). Difference scores for shifts from subordinate to other higher ranks (differences scores of +1 or +2) are not included because these subjects were not treated. However, it should be noted that any shift of one pair member's score always involves a corresponding inverse shift in the dominance score of the other member of the pair. This shift would be of the same magnitude as the first pair member's, but in the opposite direction. For example, if a dominant animal had become subordinate in the next test, then his formerly subordinate pair mate would have become dominant. Dominance in this study is examined as a dyadic behavior, and the inverse shifts in dominance ranks between one test and another are a function of the way dominance is defined and ranked with only three possible ranked scores.

At the start of the five-minute drink tests both subjects being tested were simultaneously placed in the test chamber. The only room illumination was from the chamber's house light. The subjects were distinguished from each other by a red ink mark placed on top of the head between the ears of one subject.

The water access hole was covered for two minutes to allow adaptation. The cover was removed by the experimenter when, after at least two minutes, both subjects' front quarters were a minumum of four

inches from the water access hole. The observer sat to the side of the chamber and operated timer switches to record each subject's drink times. A second set of switches was available for a second observer to use for reliability checks. The observer(s) also tallied displacements for both subjects. A timer in view of the observer(s) indicated the adaptation time and automatically signalled the end of the timed drink test by turning off the chamber light. Following testing, the subjects were returned to their home cages and given free access to water until deprivation was again instated for the next test.

A 24-hour deprivation schedule was used for the tests because preliminary evidence indicated that more competition and less clear dominant-subordinate relations resulted if the subjects were more deprived. Animals which were subordinate after only one day of water deprivation began contesting dominance (displacing) after only two consecutive days on a 23-hour water deprivation schedule.

Observer reliability. Reliability was calculated at least once for each of the four tests and once for each group. The reliability checks involved the use of two observers scoring both drink time and displacements for both subjects in a test. Two observers scored nearly 59 percent of all tests. From among these, tests were randomly selected for calculation of reliability coefficients. The reliability coefficient for drinking time was .979, with a range of .96 to 1.00. The reliability of drink time was calculated for each subject by dividing the shorter time in seconds by the longer time recorded, and then averaging between

the two subjects for the pair's individual test reliability coefficient. The same method was used for displacements. The greater number was divided by the lesser number recorded for each subject, and then the average of the subjects taken for the pair's coefficient for that test. These coefficients indicate the adequacy of both the definitions and the recording procedures used.

Shock treatments. The treated subjects were individually exposed to one hour of unsignalled and inescapable intermittent shock. The shocks were 1mA in intensity and six seconds in duration. Shocks were programmed to occur on an average of once every minute (VT1').

Only one member of each pair in the treatment groups received treatment. In pairs where dominance was established by the end of the second competitive test, the dominant subject received treatment. One member of each pair where dominance remained contested was randomly selected for treatment.

## RESULTS

Exposure to inescapable shock produced very consistent decreases in drinking time for treated subjects in all of the treatment groups, while drink time showed a slight mean increase from pre to post-test in the control group. Displacements showed a parallel result. In considering data for displacement, it is important to keep in mind the fact that displacements depend to a certain extent on drinking time--that is, a subject must be drinking in order to be displaced. If treatment reduces

the drinking time of a subject, then it may also reduce the number of displacement performed by the treated subject's pair-mate. This was found to be the case for displacement data from pairs where both subjects were ranked as contested in the test prior to treatment.

Raw data for drinking time and displacements for each pair and all tests in the study are presented in Table 3. The data are organized by groups. Within groups data for pairs for both dependent measures is juxtaposed. The subjects marked by asterisks are treated subjects or their analogues in the control group (subjects whose data was selected for analysis by the same procedure as subjects were selected for treatment in the experimental groups). In the pre and post-test columns for each group, the drinking time is presented in seconds, and the displacements performed by that subject in that test is in parentheses. In the Post-Pre difference column the difference, pre-test subtracted from post-test, is presented for both drink time and displacements. Below each group, mean times for both treated subjects and untreated subjects as separate groups is presented for both the pre-test and post-test. The plus and minus figures at the end of the mean time rows are the mean post-pre differences for treated and untreated subjects. The pre-test and post-test mean drinking times for each group are represented in Figure 1.

Dominance ranks were assigned to all subjects for each test on the basis of the data presented in Table 3. Altogether, in seven tests no displacements occurred, and time alone had to be used to determine dominance. In only four tests from a total of 40, displacements did not agree with time designations of dominance. As mentioned earlier,

Table 3

Drink Times and Displacements**

| Controls Pair | Subject | Pre-test | Post-test | Post-Pre Dif. |
|---|---|---|---|---|
| 1 | 8* | 177.9 (4)[1] | 117.9 (4) | -60.0 (0) |
| | 7 | 115.8 (0) | 43.7 (0) | -72.1 (0) |
| 2 | 9* | 136.7 (32) | 160.7 (43) | +24.0 (+11) |
| | 10 | 127.9 (32) | 111.0 (41) | -88.1 (+9) |
| 3 | 11* | 244.6 (0)[2] | 197.6 (2) | -47.0 (+2) |
| | 12 | 0 (0) | 84.0 (0) | +84.0 (0) |
| 4 | 25* | 113.4 (22) | 189.0 (9) | +75.6 (-13) |
| | 26 | 139.2 (25) | 105.0 (10) | -34.2 (-15) |
| 5 | 35* | 24.2 (2)[1] | 59.5 (2) | +35.3 (0) |
| | 36 | 184.0 (4) | 98.4 (2) | -85.6 (-2) |
| Mean | * | 139.4 | 131.9 | +5.6 (0) |
| Time | no* | 113.4 | 101.8 | -39.2 (-1.6) |

| 78 Hour Pair | Subject | Pre-test | Post-test | Post-Pre Dif. |
|---|---|---|---|---|
| 11 | 16* | 134.2 (8) | 10.2 (0) | -124.0 (-8) |
| | 15 | 140.4 (8) | 120.9 (4) | -19.5 (-4) |
| 12 | 17* | 181.8 (11) | 56.1 (0)[2] | -125.7 (-11) |
| | 18 | 102.9 (11) | 201.9 (0) | +99.0 (-11) |
| 13 | 19* | 221.3 (0)[2] | 126.1 (0) | -95.2 (0) |
| | 20 | 24.1 (0) | 41.1 (2) | +17.0 (+2) |
| 14 | 33* | 103.4 (5)[1] | 10.9 (3) | -92.5 (-2) |
| | 34 | 151.8 (0) | 148.1 (1) | -3.7 (+1) |
| 15 | 31* | 187.2 (5) | 107.2 (5) | -80.0 (0) |
| | 32 | 101.3 (3) | 67.9 (1) | -33.4 (-2) |
| Mean | * | 165.6 | 62.1 | -103.4 (-4.2) |
| Time | no* | 104.1 | 116.0 | +11.88(-2.8) |

| 48 Hour Pair | Subject | Pre-test | Post-test | Post-Pre Dif. |
|---|---|---|---|---|
| 6 | 1* | 197.7 (1) | 144.5 (2) | -53.2 (+1) |
| | 2 | 51.1 (0) | 60.5 (3) | +914 (+3) |
| 7 | 3* | 168.3 (6) | 41.9 (0)[2] | -126.4 (-6) |
| | 4 | 101.4 (5) | 85.2 (0) | -16.2 (-5) |
| 8 | 6* | 124.7 (0)[2] | 83.7 (5) | -41.0 (+5) |
| | 5 | 32.1 (0) | 92.9 (2) | +60.8 (+2) |
| 9 | 28* | 80.2 (11) | 46.0 (0)[2] | -33.4 (-11) |
| | 27 | 135.1 (11) | 157.8 (0) | +22.7 (-11) |
| 10 | 29* | 103.8 (9) | 65.4 (0) | -38.4 (-9) |
| | 30 | 160.2 (10) | 174.0 (2) | +13.8 (-8) |
| Mean | * | 135.0 | 76.5 | -58.4 (-4.4) |
| Time | no* | 96.0 | 114.1 | +18.1 (-3.6) |

| 1 Week Pair | Subject | Pre-test | Post-test | Post-Pre Dif. |
|---|---|---|---|---|
| 16 | 14* | 121.3 (13) | 30.1 (0)[2] | -91.2 (-13) |
| | 13 | 109.4 (12) | 158.8 (0) | =49.4 (-12) |
| 17 | 21* | 149.2 (2) | 163.9 (2) | +14.7 (0) |
| | 22 | 87.7 (0) | 120 0 (1) | +32.3 (+1) |
| 18 | 24* | 159.0 (3) | 93.4 (3) | -65.6 (0) |
| | 23 | 52.4 (0) | 108.7 (3) | +56.3 (+3) |
| 19 | 38* | 151.8 (8) | 162.9 (3) | +11.1 (-5) |
| | 37 | 103.4 (7) | 122.9 (4) | +19.5 (-3) |
| 20 | 39* | 85.9 (2) | 0 (0)[2] | -85.9 (-2) |
| | 40 | 97.8 (0) | 163.0 (0) | +65.2 (0) |
| Mean | * | 133.4 | 90.1 | -43.4 (-4) |
| Time | no* | 90.2 | 134.7 | +44.5 (-2.2) |

* Treated subjects.

** Drink times are reported in seconds, with number of displacements in parentheses.

displacements were always used to indicate dominance/subordination designations when they occurred.

Dominance ranks for treated subjects for the pre and post-test, and rank differences and rank differences from pre to post-test for these subjects (actual differences column) are presented in Table 4. In this table actual differences are compared with potential negative differences. A negative difference indicates a shift toward subordination. The ranks for subjects in the control group were taken from the asterisked subjects in Table 3--subjects selected for comparative analysis by the same method as subjects were selected for treatment in the experimental groups.

In the actual difference column in Table 4 we see that there were no differences between pre and post-test for the control group subjects. In the 48-hour group negative shifts or shifts toward subordination were consistently produced in all treated subjects. These shifts were from contested to subordinate, where a maximum shift of -1 was all that was possible, and from dominant to contested, where a shift of -1 was observed but a shift of -2 (from dominant to subordinate) was possible. In both the 72-hour and the one-week (168 hour) groups the treated subjects showed dominance shifts. In each group a -2 degree actual shift was produced once (three such shifts were possible in the 72-hour group, and two were possible in the one-week group), and one subject in each group showed no shift from pre to post-test.

Total actual shifts observed for each group can be compared with total potential negative shifts. No group produced the potential shift.

Table 4

Dominance Ranks and Shifts from Pre to Post Test

for Treated Subjects

| Controls Pair | Subj. | Pre-test Rank | Post-test Rank | Actual Difference | Potential Neg. Difference |
|---|---|---|---|---|---|
| 1 | 8 | 1 | 1 | 0 | -2 |
| 2 | 9 | 2 | 2 | 0 | -1 |
| 3 | 11 | 1 | 1 | 0 | -2 |
| 4 | 25 | 2 | 2 | 0 | -1 |
| 5 | 35 | 2 | 2 | 0 | -1 |
| Totals | | | | 0 | -7 |
| 48 Hrs. | | | | | |
| Pair | Subj. | | | | |
| 6 | 1 | 1 | 2 | -1 | -2 |
| 7 | 3 | 2 | 3 | -1 | -1 |
| 8 | 6 | 1 | 2 | -1 | -2 |
| 9 | 28 | 2 | 3 | -1 | -1 |
| 10 | 29 | 2 | 3 | -1 | -1 |
| Totals | | | | -5 | -7 |
| 72 Hrs. | | | | | |
| Pair | Subj. | | | | |
| 11 | 16 | 2 | 3 | -1 | -1 |
| 12 | 17 | 2 | 3 | -1 | -1 |
| 13 | 19 | 1 | 3 | -2 | -2 |
| 14 | 33 | 1 | 2 | -1 | -2 |
| 15 | 31 | 2 | 2 | 0 | -1 |
| Totals | | | | -5 | -7 |
| 1 Week | | | | | |
| Pair | Subj. | | | | |
| 16 | 14 | 2 | 3 | -1 | -1 |
| 17 | 21 | 1 | 2 | -1 | -2 |
| 18 | 24 | 1 | 2 | -1 | -2 |
| 19 | 38 | 2 | 2 | 0 | -1 |
| 20 | 39 | 1 | 3 | -2 | -2 |
| Totals | | | | -5 | -8 |

Ranks:
1--dominant
2--contested
3--subordinate

Total potential negative dominance shifts of -7 were possible for the control group, and for the 48-hour and 72-hour groups, and a potential of -8 was possible for the one-week group. No positive shifts (toward dominance) were found in either the control group or in any of the three treatment groups. Such shifts were possible when the treated subject's pre-test rank was a 2, or contested.

Table 4 clearly indicates that dominance shifts were produced in all three experimental groups. The difference between pre and post-test ranks indicates that these shifts were in the hypothesized direction. They were shifts toward subordination. However, no experimental group showed the maximum negative rank shift possible for that group. The sum of -5 for each experimental group's actual rank difference indicates no difference between experimental groups according to this analysis (by rank differences).

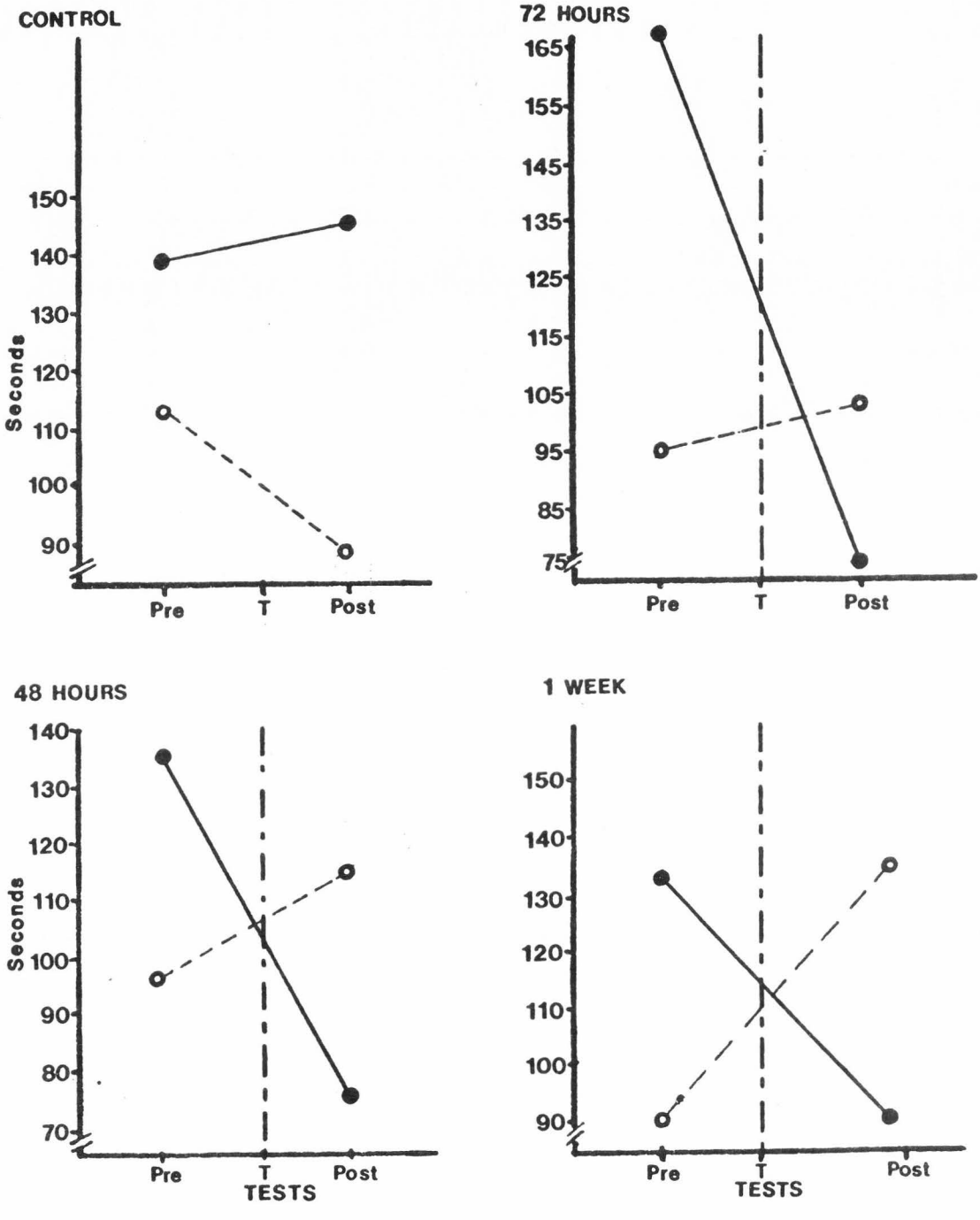
A non-parametric analysis using chi square for a dichotomous variable of change/no-change was recommended for analysis of the rank differences from pre to post-test (Dr. Donald Sisson, personal communication, 1978). Analysis of variance of rank differences was precluded by the small number of ranks (3) and the frequency of one-degree differences (see Table 4). A chi square test of independence employing Yates' correction for cell expectancies of five or less yielded a significant result for the variables change (change, no-change) vs. group (control, E-48, E-72, and E-168), $X^2 = 8.12$, $p. < .05$, d.f. = 3. No significant result was produced for change (maximum shift, not maximum shift) vs. groups. This lack of signi-

ficance precluded further statistical analysis of differences between treatment groups with regard to rank differences from pre to post-test.

In addition to producing dominance rank shifts in treated subjects, the inescapable shock treatment also produced differences in the mean drinking time of treated subjects from pre to post-test in each experimental group. With a separate graph for each group in the study, Figure 1 represents mean differences for drink time from pre-test to post-test for treated subjects compared with untreated subjects. In the control group drink time for subjects analogous to treated subjects in the experimental group increased slightly from pre-test to post-test, while it decreased for untreated analogues. In other words, in the control group differences between "treated" and "untreated" subjects diverged from pre-test to post-test when no shock treatment intervened.

In each of the experimental groups the treated subjects' mean drink time falls substantially from pre-test to post-test, while the untreated subject's time increased. That is, in these groups differences between treated and untreated subjects' drink times reversed from pre-test to post-test. Since time is represented on the ordinate at a fixed rate of 10 seconds per centimeter, the degree of difference can be directly compared in the four figures. It is clear that the decline for treated subjects is greatest in the 72-hour group, and less in the 48-hour and one-week groups, respectively. Reference to Table 3 indicates that the decline in the drinking time in the treatment groups

Figure 1: Mean drinking time in each group for treated and untreated subjects on pre and post-treatment tests. Control subjects were not treated.




● Treated subjects or their analogues in the control group

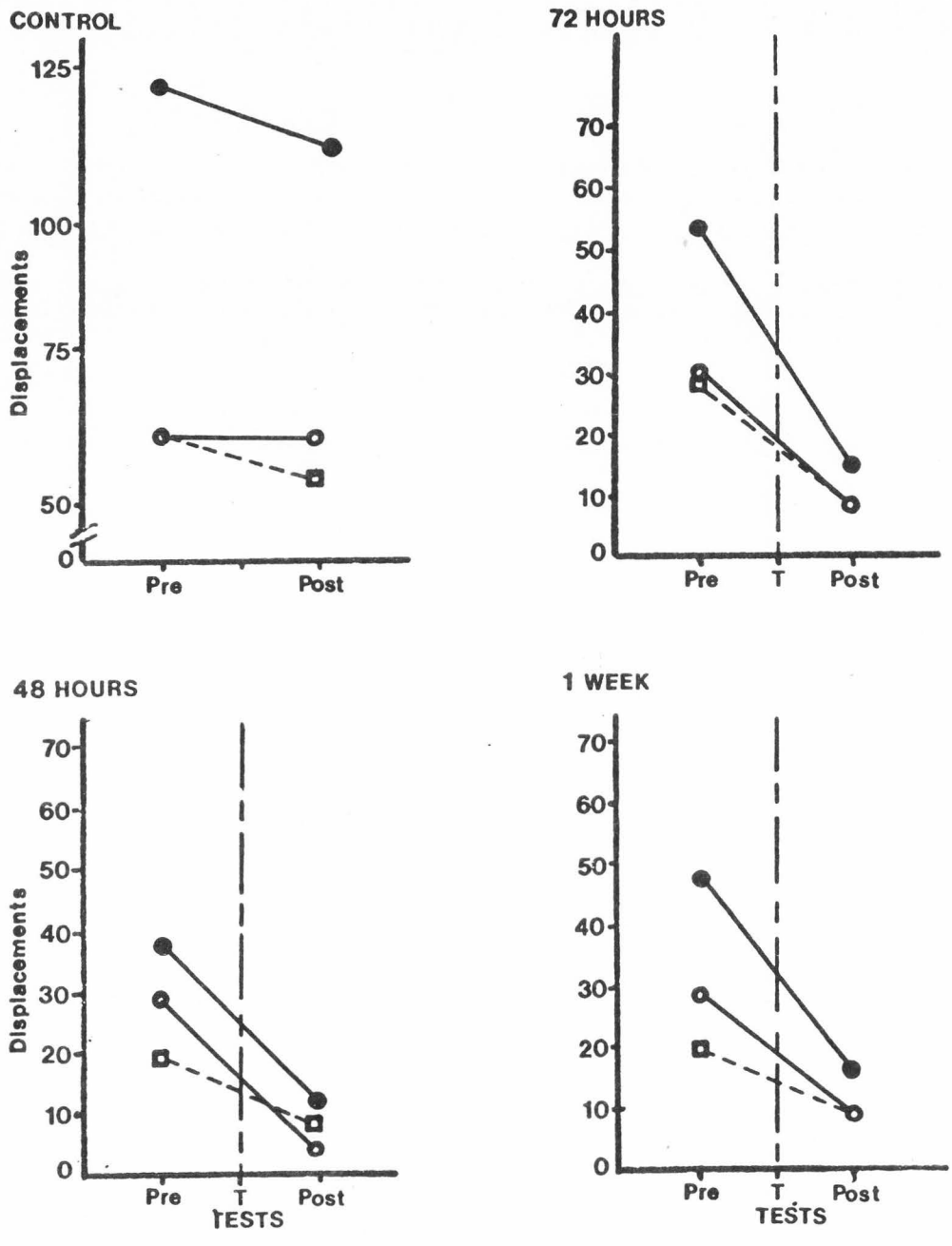
○ Untreated subjects

is more than a mean effect. This is clear in the Post-Pre Differences column, looking at differences for the treated (asterisked) subjects.

The general effect of treatment on displacements is represented in Figure 2. Here total displacements for each group are shown against totals for treated subjects and for untreated subjects. This indicates the contribution that treated subjects make to the decline observed in all groups in number of displacements from pre-test to post-test in comparison to untreated subjects. As in Figure 1, ordinate intervals are constant across the four figures (10 displacements per centimeter), so the figures may be directly compared. The decrease in total displacement is greater in the treatment groups than in the control group. In the treatment groups both treated and untreated subjects contribute to this decline, while in the control group the decline is due to the "untreated" subjects, while the "treated" subjects remained constant in total displacements produced.

The mean effects represented in Figure 2 are not as consistent over subjects as the drink time differences were for treated subjects. There is more within-group variability for this measure, as is evident in Table 3. Also, the number of displacements tends for most subjects to be few, so that occasional frequent displacing by pairs in tests tends to greatly increase within-group variability. I have already pointed out that displacements depend on drinking time, so that the decline in the untreated subjects' total displacements in each of the treatment groups is partly accounted for by the treated subjects'

Figure 2: Displacement totals for the four groups from the pre and post-treatment tests. Total displacements for all subjects are shown against totals for treated subjects and for untreated subjects.




● Total displacements for the group

o Total for treated subjects

□ Total for untreated subjects

decreased drink time in the post tests; the untreated subjects have less occasion to displace. I think it is appropriate to view this measure as useful for determining dominance ranks, and as reliable in this respect considering the stability shown by the control subjects in the rank differences presented in Table 4. But the dependency of this measure on drink time, and the fact that it is across types of subjects (untreated subjects' displacements depend on treated subjects' drink time) led me to suspect that no significant effects of the treatment would be found in an analysis of variance for this variable.

Analysis of Variance for Treated
and for Untreated Subjects

In order to independently assess the effect of treating only one member of each pair on the behavior of each pair member, separate analyses of variance were done for treated and untreated subjects on both dependent measures, drink time and displacements. A two-way analysis of variance was used for both analyses, 4(groups) X 2(tests).

For treated subjects the following significant differences were found:

1. On drink time there was a significant main effect of Tests, $F = 25.13$, $P = .000127$ for d.f. - 1/16. The Groups X Tests interaction was also significant, with $F = 5.09$, $p. < .05$ for d.f. = 3/16.
2. On displacements the main effect for Tests approached significance with $F = 4.27$, $P = .055$. The critical value for

p. $<$.05 is 4.49 for d.f. = 1/16. No other significant effects were found for this variable.

For the untreated subjects differences merely approached significance. For drink time only the main effect for Tests approached significance with F = 3.34, d.f. 1/16. For dispalcements, again, only the main effect for Tests approached significance, F = 3.12.

An initial analysis of variance of all individual data, with subjects nested within pairs, which were nested within groups, and tests treated as a split plot, indicated a significant main effect of Tests, F = 6.595, p. $<$.05, while the Groups X Tests interaction approached the .05 level, F = 3.016, d.f. = 3/16, P = .0606. The critical value for p $<$.05 is 3.24. We see in the separate two-way analyses for treated and untreated subjects that the significance found in the earlier three-way analysis resides in the treated subjects.

Analyses of Variance on Pair Data

Differences within each pair on the pre-tests and post-tests for both drink time and displacements were calculated by subtracting the untreated subject's measure from the treated subject's. Thus, the untreated subjects' drink time on the pre-test was subtracted from the treated subject's to obtain a pair time-difference score for that test. These were calculated for both pre and post-tests and for both displacements and drink times.

A two-way analysis of variance (Groups X Tests) on these within-pair differences on the two dependent variables yielded the following significant differences:

For drink time differences within pairs the following results were obtained:

1. A significant main effect for Tests, $F = 15.306$, $P = .00124$ d.f. = 1/16.
2. A significant Groups X Tests interaction, $F = 4.074$, $p < .05$, d.f. = 3/16.

For displacement differences within pairs the Groups X Tests interaction was significant, $F = 4.24$, $p < .05$, while main effects were not significant.

In general this analysis indicates that the effect of treating one pair member can be viewed appropriately as an effect on a relationship within the pair. This is made clear by Table 5, which follows, showing mean within-pair differences overall and for each group from pre-test to post-test.

In Table 5 we see an increase in the control group and a decrease in treatment groups from pre-test to post-test for both drink time differences and displacement differences within pairs. The increase in drink time and displacement differences from pre to post-test in the control group indicates the differences widened between pair members. The decrease in drink time from a positive to a negative value in the three treatment groups means the treated subjects went from drinking longer than their pair mates during the pre-test to drinking for less time in the post-test. For displacements the difference in the control group indicates that the analogues of the treated subjects went from

Table 5

Mean Within-Pair Differences for

Drink Time and Displacements

| Groups | Mean Drink Time Differences | | Mean Displacement Differences | |
|---|---|---|---|---|
| | Pre-test | Post-test | Pre-test | Post-test |
| All Groups* | 43.675 | -19.005 | not significant | |
| Controls** | 25.98 | 58.1 | -.2 | 1.4 |
| E-48 | 43.94 | -37.62 | .2 | 0 |
| E-72 | 61.48 | -51.88 | 1.4 | 0 |
| E-168 | 43.3 | -44.62 | 1.8 | 0 |

* Main effect for tests

** Interaction effect for groups x tests

displacing slightly less than their pair mates during the pre-test to displacing more in the post-test. The shift from positive difference values in the pre-test to zeroes in the post-test for all treatment groups indicates that treated subjects went from displacing more frequently in the pre-test to displacing only as often as their pair mates on the post-test. Frequently this was a matter of neither subject displacing at all on the post-test, as can be seen in Table 3.

An analysis of variance was also run on time and displacement ratios within pairs, but no significant results were obtained.

Analysis of Variance on Treated Subjects' Difference Scores

To simplify the analysis, the treated subjects' drink times and displacements during the pre and post-tests were reduced to difference scores by subtracting the pre-test result from the post-test for each dependent variable (these scores are listed, together with the untreated subject's scores, in the Post-Pre Differences columns in Table 3). This analysis yielded a significant $F = 5.09$, $p < .05$ for d.f. = 3/16 for drink time differences. No significant results were found for displacements.

Since our working hypothesis was that the effect of shock treatment would follow the time-course found by Glazer and Weiss (1976a), and be at a maximum 72 hours after treatment, and less at 48 hours and one week, respectively, orthogonal comparisons were made between group means

from this analysis. Group means for treatment groups E-48 and E-72 were both significantly different from the control group's mean, $F = 5.17$, $p$ .05 for $X_1 - X_2$ (controls compared to E-48) and $F + 14.98$, $p = .00136$ for $X_1 - X_3$ (controls compared to E-72). The difference between controls and treatment group E-168 was not significant, $F = 3.02$. The only other significant difference was between E-72 and E-168, with $F = 4.55$, $p$ .05. E-72 was not significantly different from E-48, $F = 2.55$, nor, of course, was E-48 significantly different from E-168. Degrees of freedom for the above tests were 1/16. The mean differences for drink times are given in Table 3.

This analysis allows the interpretation that the effect of shock treatment was greatest at 72 hours after treatment, but indicates no significant difference between the 48-hour and one-week treatment groups. The effect of treatment is also consistent over groups, as in the rank differences analysis, producing consistent declines in drink time from treated subjects in the post-test.

## DISCUSSION

The overall shifts in dominance ranks and reductions in drink time and total displacements observed in subjects treated with inescapable shock indicate a generalized effect of treatment on social competitive responding. The various hypotheses advanced to account for response deficits observed in subjects after exposure to inescapable shock have in most cases focused on deficits in escape response acquisition. In the present study it was found that the effect of shock on competitive

dominance was to produce a shift in treated subjects toward subordination and corresponding shifts toward dominance in their untreated pair-mates. These changes were assessed against the performance of untreated controls, which showed stability of dominance ranks from pre-test to post-test. The stability of controls demonstrated the effectiveness of the procedure used to achieve stable dominance relationships in paired rats, i.e. pair housing, use of a consistent and moderate level of water deprivation prior to testing, and a competitive test duration of five minutes. Which, if any, of these measures was crucial was not assessed in this study, since the objective was simply to produce a stable relationship against which the effects of shock treatment could be assessed.

The magnitude of the effect of shock treatment on drink time did vary as a function of time of post-test. These differences between groups closely paralleled the time course observed by Glazer and Weiss (1976a,b) for escape response acquisition deficits. The reductions in drink time from pre-test to post-test were greatest 72 hours after treatment, and less 48 hours and one week after treatment. It is interesting to find a generalized effect that so closely parallels Glazer and Weiss' results. Their hypothesis of learned inactivity would not predict these parallel results since it relies on principles of stimulus control to account for the deficits they observed in escape behavior (responding was controlled by a shock stimulus motivator). The transfer observed in the present study is unusual in that it cannot be accounted for by learning theory principles of stimulus control. There is no

basis for the transfer to occur, since the shock chamber and drink chamber are very different environments, and the shock and water stimuli afford very different contexts.

In their analysis of their results, Glazer and Weiss (1976a) gave no explanation of the time course they observed for the effect of shock treatment. Similarly no adequate explanation can be offered here. But indications from this study are that the effect of treatment is transitory, with evidence of some recovery of responding in subjects tested one week after treatment. In groups tested 48 hours and 72 hours after being treated, the effect was not only to reduce drink time for the treated subjects and increase it for their untreated pair-mates, but to reduce overall within-group variability. Though there was still an overall mean reduction in drink time for the one-week post-test group, the within-group variability was more like that seen in the control group. That is why the statistical analysis indicated no significant differences between the control group and the one-week group.

The fact that the treated subjects showed some recovery (though it is more in terms of recovered within-group variability than recovered drink time on a mean or an individual basis) one week after treatment suggests a possible transitory physiological basis for the effects of treatment. If the effect were due to learning, we might expect a more enduring change in behavhior, since this is how learning is defined. Weiss, Glazer and associates have experimentally

explored this avenue of explanation with what appears now to be little profit (Weiss & Glazer, 1975; Wiess, Glazer, Pohorecky, Brick & Miller, 1975; Glazer, Weiss, Pohorecky & Miller, 1975). They used treatment parameters and experimental procedures very different from those of learned helplessness proponents (Maier & Seligman, 1976). But the possibility of a physiological account which precludes learning has by no means been exhausted, and further examination is warrented, especially in view of their more recent findings (Glazer & Weiss, 1976a,b). The unusual transfer observed in the present study from a shock treatment situation to a competitive dominance test might be better accounted for by a physiological explanation, since learning theory principles offer no explanation.

In summary, the learned inactivity hypothesis (Glazer & Weiss, 1976a,b) cannot account for either the general effect of shock treatment on competitive responding observed in the present study, or the time course of the effect of treatment. It is an S-R or stimulus control hypothesis and would expect transfer to occur only where some controlling stimulus could be identified. The learned inactivity proponents intended their hypothesis simply to be an alternative and more plausible account of the escape response deficit observed in subjects after inescapable shock treatment, only, and they suggest that there may well be other effects of this treatment. However, the general effects of inescapable shock treatment may be of more interest than the specific ones, and the utility of the learned inactivity hypothesis appears very constrained.

Learned helplessness proponents might offer the following account of the time course found in the present study and by Glazer and Weiss (1976a). They have found that escape response deficits are transitory only when experienced subjects or mature subjects of unknown history are used--dogs from the city pound (Maier & Seligman, 1976; Maier & Testa, 1975). Maier has called this effect "pro-active interference" or "associative interference"--previous learning of contingency between responding and reinforcement, even though in other contexts, causes the subject to quickly overcome an escape response acquisition deficit (Maier & Testa). The drawback to this interpretation is that it has not been sufficiently examined in their published work--typically the time course of the deficit is either not treated or only superficially examined (Seligman, Rosellini & Kozak, 1975; Seligman & Groves, 1970). This interpretation would predict that subjects treated when very young would show enduring deficits, and this is in fact what Hannum, Rosellini and Seligman (1975) found when they treated rats just after weaning and tested them 90 days later.

The subjects used in the present study were mature rats 5 to 7 months old, and were probably housed since weaning in group cages in the laboratory from which they were purchased. Being group housed would have given them ample opportunity to learn various contingencies between responding and reinforcement. Glazer and Weiss (1976a,b) used 90-day old rats, and they do not report the housing conditions

of their subjects prior to their being used experimentally. If they were group housed, this pre-experimental condition might account for the time course they observed, showing a peak deficit in escape responding at 72 hours after treatment, and some recovery one week after treatment.

Maier's account of pro-active interference (Maier & Testa, 1975) has led to therapy and immunization procedures used to eliminate or prevent escape response deficits after treatment with inescapable shock (Williams & Maier, 1977; Seligman, Rosellini & Kozak, 1975) which were reviewed earlier. The immunization procedure in these studies is to pre-train rats to escape shock, then expose them to inescapable shock and test for subsequent escape response acquisition. Subjects pre-trained to escape showed no deficits in escape response acquisition.

This leads to the only difficulty that the learned helplessness hypothesis has with the results found in this study. Insofar as pre-exposing subjects to each other and to the test environment in a pre-test is analogous to pre-training subjects to escape, using a pre-test/post-test design would predict that inescapable shock would have no effect on post-test performance. The pre-test should prevent an effect of treatment by immunizing the subjects. Immunization research would suggest that inescapable shock would have no effect on established social responses or any other established responses.

The results of this study clearly show that pre-testing the subjects did not prevent an effect of treatment. This suggests that

social responses may be affected differently than response acquisition (e.g., escape responding or problem solving) by exposure to inescapable shock. Learned helplessness has been proposed as an account of depression (Seligman, 1975). In this respect, learned helplessness has been both a convincing and useful model, since therapy and immunization procedures are readily suggested and could generally be characterized as procedures to train competence or correlations between responding and reinforcement. But to date efforts to prevent or reverse the effects of exposure to response-independent aversive events have focused on deficits in response acquisition. Social responding, when it evidences certain types of changes, is an important member of the nexis of responses which index depression. If social responding, or just certain types of social responses, are not affected in the same way as the learning of new responses, then efforts to develop therapy and immunization programs for depression based on the learned helplessness model might fall short in the important area of social behavior. It would be of interest, then, to examine the effects of the therapy and immunization procedures developed by Seligman, Rosellini and Kozak (1975) and Williams and Maier (1977) for their effects on a variety of social behaviors. I suggest that the results of the present study indicate that issues of behavioral covariance are relevant in the development of depression therapies from the learned helplessness model.

Maier and Seligman have hypothesized three areas of deficits as a function of exposure to inescapable shock--associative, motivational,

and emotional. An informal assessment of response changes in treated subjects observed during the competitive dominance post-test leads to the conclusion that the consistent effect of treatment was to disrupt responding in a very general way. Treated subjects seemed simply not to stay on task, whether they were engaged in grooming, drinking, exploring the chamber or engaging in social responses. Exposure to shock seemed to affect the duration of a variety of types of responding rather than to lower the activity level of the subjects, or produce changes that would indicate they were applying a principle of response-outcome independence to their responding. This was an informal evaluation made independently by the observers in this study. It is not clear how such a disruption of durations of behaviors would fit into the learned helplessness model, if at all.

Future work should examine whether inescapable shock produces frequent response interruptions or a general disruption of on-task behavior, and whether this might be a better account of the effect of exposure to inescapable shock. Shock treatments of different intensities might produce different ways of disrupting responding so as to inhibit learning. These could only superficially have the same effect, getting there by very different means.

The effect of deprivation level on competitive dominance should also be examined. Preliminary work for the present study indicated that clear dominance - subordinate relationships emerged from pairs of rats tested after 24 hours of water deprivation, but that this degenerated to vigorous competition for water after only two days on a 23½ hour water deprivation schedule.

Most importantly, the issue of behavioral covariance should be addressed with regard to the therapy and immunization procedures developed by the proponents of learned helplessness. If the effect of exposure to uncontrollable aversive events is widely generalized--as the results of the present study indicate--and the effect of therapy or immunization procedures is specific to subsequent learning, then these procedures will be inadequate.

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