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INTRODUCTION

Systematic desensitization, developed by Joseph Wolpe in the early 1950's, is the most widely used form of behavior therapy. The efficacy of this treatment was conceptualized by Wolpe (1958) as learned or conditioned inhibition based upon reciprocal inhibition. The behavioral prediction was succinctly stated by Wolpe (1958) in his basic principle:

> "If a response antagonistic to anxiety can be made to occur in the presence of anxiety-evoking stimuli so that it is accompanied by a complete or partial suppression of the anxiety responses, the bond between these stimuli and the anxiety responses will be weakened." (p. 71).

Wolpe's basic principle, or reciprocal inhibition principle as it is sometimes called, is operationally nothing more than a descriptive definition of counterconditioning. For example, the definitions of counterconditioning offered by Bandura (1969), "...eliciting activities that are incompatible with emotional responses in the presence of fear--or anxiety--arousing stimuli" (p. 424), and Kimble (1961), "Extinction under circumstances in which the response decrement is hastened by the reinforcement of a response which displaces the original conditioned response" (p. 478), constitute a more general statement of the reciprocal inhibition principle. Wolpe has in fact recognized the descriptive similarity, and uses the terms reciprocal inhibition and counterconditioning interchangeably, but has indicated some preference for the former due to his hypothesized neurological process accounting for the observed change in behavior. This

is unfortunate since Wolpe has apparently confused a procedure (counter-conditioning) with a construct (conditioned inhibition) which was in turn based upon an hypothesized neurophysiological explanation (reciprocal inhibition). The confusion over the terminology used in systematic desensitization is well documented (e.g., Evans & Wilson, 1968; Yates, 1970). The confusion of levels of discourse is not new to psychology, and as noted by Bandura (1969), the efficacy of a treatment based on a counter-conditioning principle is independent of the validity of Wolpe's neurophysiological explanation. A similar view was expressed by Spence (1956) with reference to intervening variable theory in general.

Thus, despite the fact that there is now good evidence for the effectiveness of systematic desensitization in reducing avoidance behavior (cf. Paul, 1969) there remains, as noted in the following review, the question as to whether this efficacy is correctly predicted by counter-conditioning. One of the most obvious implications of Wolpe's counter-conditioning principle is that a relationship (albeit unspecified) should exist between the amount of counterconditioning and the diminution of avoidance behavior, since counterconditioning is a learning process. This assumption seems to have completely escaped the attention of researchers in this area, with the exception of Melvin and Brown's (1964) seldom cited investigation involving the neutralization of an aversive light stimulus as a function of number of paired presentations with food.

With regard to human subjects, some researchers in attempting to assess the counterconditioning hypothesis have rendered their results uninterpretable by the omission of pre-test scores, e.g., Cooke (1968), or by the addition of another facet such as muscle tension, e.g., Wolpin and Raines (1966),

the use of extremely small Ns, e.g., Rachman (1965). Unambigous support for a counterconditioning hypothesis has been offered by Davison (1968). A similar picture emerges from infrahuman literature, in which the counterconditioning hypothesis has been investigated using a wide galaxy of experimental procedures, viz, CER (Gale, Strumfels, & Gale, 1966; Poppen, 1970); passive avoidance (Goldstein, 1969; Wilson & Dinsmoor, 1970); active avoidance (Julia Hall, 1955; Klein, 1969; Melvin & Brown, 1964; Nelson, 1966; Sermat & Shepard, 1956); Sidman avoidance (Gambrill, 1967). In a number of cases the reduction in avoidance behavior brought about by counterconditioning has been transient (Gale, et al., 1966; Gambrill, 1967; Melvin & Brown, 1964; Sermat & Shepard, 1959), or the interpretation of the results rendered somewhat dubious by the experimenter's failure to include untreated controls (Poppen, 1970; Wilson & Dinsmoor, 1970).

Moreover, as noted by Wilson and Dinsmoor (1970), the use of an active avoidance (crossing from the former shock to a safe side) to assess the effects of counterconditioning (typically feeding in the formerly shock side) is easily confounded by the fact that reduced latencies displayed by the counterconditioned subjects may be the result of secondary reinforcement set up by feeding in the shock area, a conclusion also reached by Melvin and Brown (1964), or crouching or freezing which results from shock and is obviously incompatible with active responding, or both. The latter hypothesis, as noted by Wilson and Dinsmoor (1970), was also expressed by Blanchard and Blanchard (1969) and Weiss, Krieckhaus and Conte (1968).

Hence, the purpose of the present study was to: (1) investigate the counterconditioning hypothesis, employing appropriate controls; (2) investigate its most obvious implication, i.e., that a relationship

(albeit unspecified) should exist between the degree of counterconditioning and the subsequent reduction of avoidance behavior; (3) compare the efficacy of implosion or response prevention (e.g., Baum, 1970; Stampfl & Levis, 1967), with varying degrees of counterconditioning, using a passive avoidance procedure.

Method

Subjects

The subjects were 70 male albino rats of the Sprague-Dawley strain, 124 days old at the beginning of the experiment, purchased from the Holtzman Company, and housed in individual cages. The subjects were randomly assigned to one of seven experimental groups, and to one non-shock control group. The animals were naive to shock but had been previously used in an appetitive runway study.

Apparatus

The apparatus was a wooden runway, 36 x 5 x 10.5 in. The interior was painted glossy black and was divided into safe and shock compartments by a clear plexiglas guillotine door. Each compartment was covered by a hinged wire screen top with a hook and eye fastener. The safe area was 8 in. long, whereas the shock area measured 28 in. The floor of the shock and safe area consisted of stainless steel bars, 7/8 in. in diameter and spaced 1 3/4 in. apart center to center. A retractable water bottle was mounted on the wall of the safe area opposite the guillotine door, 3 1/4 in. from the floor. One photocell was mounted in the shock area 1.5 in. above the grid and 19 1/4 in. from the guillotine door. Scrambled shock was administered by a Grason-Stadler shock generator (Model 700), and licking responses were recorded by a drinkometer. Masking noise was provided by a small ventilating fan.

Procedure

The experimental subjects were run in eight replications of eight subjects each; each replication took four days. Of these animals, six failed to acquire the passive avoidance task in one trial, and two failed to reach the lick criterion. These eight subjects were excluded, thereby reducing the N to eight in each of the seven experimental groups, and to six in the non-shock control. The animals in each replication were placed on a 23½ hour water deprivation schedule four days prior to the beginning of the replication and were maintained on this schedule throughout the replication. Lab chow was constantly available.

Phase I. Lick training and acquisition of passive avoidance. On the first day of the experiment animals were given 15 min. of confined free licking in the safe area, followed by 15 min. in a neutral home cage. The animals were then returned to the safe area for passive avoidance training. When the subjects left the safe area and entered the shock area the guillotine door was closed and a 15 sec., 3 ma (nominal setting) inescapable shock was administered. Following this passive avoidance trial, the subject was immediately returned to the safe area and was subjected to a five-min. passive avoidance test.

The criterion for successful passive avoidance was defined as a failure to leave the safe area within the 5 min. test period. Leaving the safe area was defined by the subject's hind paws touching the first bar in the shock area. Six animals failed to reach this criterion and were excluded. Upon termination of the passive avoidance trial, each animal was replaced in its home cage which also served as a carrying cage and returned to the colony room.

In order to minimize odor cues, the entire apparatus was wiped and the paper beneath it was changed between each animal. This procedure was continued throughout the entire experiment.

Phase II. Treatment and Test. This phase lasted for 3 days. The first day followed successful passive avoidance training by a 24-hour interval. Treatment and Test Days 1, 2, and 3 consisted of confined treatment for 10 min. on Day 1 and 7 min. on each of the two following days. Each treatment session was immediately followed by a 20 min. test period. The experimental treatments and their specifics were as follows:

1. Counterconditioning High (CCH). The subjects were confined to the safe area and allowed to reach a total of 1200 licks during each treatment session.
2. Countercondition Medium (CCM). The subjects were confined to the safe area and allowed to reach a total of 600 licks during each treatment session.
3. Counterconditioning Low (CCL). The subjects were confined to the safe area and allowed to reach a total of 300 licks during each treatment session.
4. Extinction. The subjects were confined in the safe area during each treatment session.
5. Implosion. The subjects were placed in the shock area in front of the closed guillotine door and confined there during each treatment session.
6. Untreated Control-Home Cage (UTC/HC). The subjects were transported from the colony room into the lab, and spent each treatment session in their home cages.

7. Untreated Control-Trash Can (UTC/TC). The subjects spent each treatment session in an empty covered trash can. The trash can, painted a metallic grey-pink, had a diameter of 13 in. at the top and tapered to 10 in. at the base.

8. Non-shock Controls (NSC). The subjects did not receive acquisition training and were divided into two groups, one of which received lick training and thereafter were treated identically to the CCM subjects; the other group did not receive lick training and were treated identically to the Extinction subjects. Since no differential effects resulted from this treatment, the results were combined.

Test. The rest for residual fear followed immediately after treatment. Each subject was removed from its respective treatment area and placed in the safe area facing the guillotine door. The guillotine door was raised and the following dependent measures were recorded.

1. Initial Exit Latency IEL. Initial exit latency was defined as that time spent in the safe area before both hind paws touched the third bar (3½ in.) within the shock area.

2. Photocell Latency (PCL). The time between the initial opening of the guillotine door and the subject's passing in front of the photocell was recorded as PCL.

3. Time on Bars (TOB). TOB refers to time the subject spent on the bars within the shock area. Recording began when the animal's IEL ended, as it cleared the third bar within the shock area, and continued throughout the test whenever the subject was in the shock area.

4. Number of Exits (NE). Number of exits was defined as the number of traversals from the safe to the shock area. Recording of this measure began with the first exit and continued throughout the test session.

IEL was recorded to the nearest second by a stopwatch; TOB and PCL to the nearest tenth of a minute by elapsed time indicators; NE was recorded by a mechanical counter. The recording devices for TOB and NE were manually activated by the experimenter. All time measures were converted to seconds. Animals which failed to exit from the safe area received a latency of 1200 seconds on both latency measures.

Results

Lick Data

An analysis of the number of cumulative licks during the 15 min. confinement period during Phase I revealed a nonsignificant treatment effect at 7, 10, and 15 min. (all dfs=6/49), Fs=.71, .90 and 1.03 respectively.

Between Group Differences

The means, per subject over three days, for each dependent variable are depicted in Table 1. Because of the unequal number of subjects in the no-shock group, and the obvious difference between this group and all others, these data are not included in the overall statistical analysis. A 7(Treatments) by 3(Days) analysis was used to evaluate each dependent variable. The results (all dfs=6/49) indicated a significant treatment effect: IEL ($F=4.54$, $p<.01$); NE ($F=2.77$, $p<.05$); PCL ($F=4.21$, $p<.01$); TOB ($F=2.95$, $p<.05$).

Although the number of repeated elements (Days) was small, the Day effect and the Day x Treatment interaction was evaluated using the Geisser and Greenhouse (1958) df correction procedure for possible nonequality of covariances. This procedure results in a critical value that is much larger than that for the usual uncorrected procedure. The Day effect (all corrected dfs=1/49 as opposed to the usual dfs of 2/98) was significant for IEL (F=77.18, p<.001); NE (F=63.99, p<.001); PCL (F=47.47; p<.001); TOB (F=49.66; p<.001). Furthermore, the Treatment x Day interaction (all dfs=6/49 as opposed to the usual dfs of 12/98) was significant for NE (F=2.41, p<.05); PCL (F=2.82, p<.05); TOB (F=2.64, p<.05); and of marginal significance for IEL (F=2.10, p<.10). Moreover, as depicted in Table 1, the Implosion, CCH, CCM, and UTC/HC groups showed a decided diminution of passive avoidance behavior relative to the CCL, Extinction and UTC/TC groups. Indeed, post hoc comparisons (Newman-Keuls) revealed that the difference between the two sets of groups was quite uniform and significant (p<.05) across most dependent variables. Subjects in the Implosion, CCH, CCM, and UTC/HC treatments showed significantly reduced IEL, PCL, and significantly greater TOB when compared with the CCL, Extinction, and UTC/TC treatments. Of considerable interest is the fact that Implosion, CCH, CCM, and UTC/HC were equall effective whereas CCL, Extinction and UTC/TC were equally ineffective. Results similar to those above were also evident for the dependent variable NE; the only exception was the Implosion-CCL and CCL-UTC/HC comparisons which proved to be nonsignificant with this measure.

Discussion

Since counterconditioning is a learning phenomenon one would expect its efficacy to depend upon the number of counterconditioning trials.

The results were in partial accord with this hypothesis; subjects in the CCH and CCM conditions displayed a sharp decrement in passive avoidance behavior when compared with the CCL subjects. However, the relationship was decidedly non-linear in that the CCH and CCM subjects did not differ significantly from each other. Also, the behavior of the CCH and CCM subjects did not differ from the Implosion and UTC/HC groups.

The diminution in passive avoidance behavior shown by the Implosion subjects far exceeded that shown by the CCL and Extinction subjects, but did not differ significantly from the CCH and CCM groups. These data highlight a very critical point, namely, that results of investigations involving comparison of the effectiveness of systematic desensitization with other treatment procedures, such as implosion and extinction (e.g., Davison, 1968; Rachman, 1966; Willis & Edwards, 1969) may lead to erroneous conclusions concerning the relative effectiveness of various treatments due to the lack of specification of the amount of counterconditioning, thereby obfuscating an area already beset with confusion. For instance, a restricted comparison of the CCL, Extinction and UTC/TC groups might prompt one to conclude erroneously that counterconditioning is relatively ineffective in eliminating passive avoidance behavior and not superior to extinction procedures.

The literature abounds with explanations of avoidance reduction following counterconditioning. The most noteworthy of these are: the maximal habituation hypothesis by Lader and Mathews (1968); Wilson and Davison's conceptualization that the counterconditioning procedure serves as a vehicle or facilitator for exposure to aversive stimuli (1971); and Wolpe's original use of conditioned inhibition (1952, 1958).

The maximal habituation hypothesis as espoused by Lader and Mathews (1968) embraces the idea that relaxation brought about by counterconditioning serves to lower the subject's arousal level and thereby facilitates habituation (which they view as functionally equivalent to extinction). This assumption has received empirical support from a number of divergent sources. First, there is an inverse relationship between arousal level, as measured by spontaneous skin fluctuations, and habituation to neutral stimuli (e.g., Lader, 1967; but see Van Egeren, 1971). More recently, Lang, Melamed and Hart (1970) have shown that rapid heart rate habituation predicted success of systematic desensitization. Second, early animal data (e.g., Perin, 1942; Williams, 1938) clearly demonstrated that resistance to extinction was positively related to drive level during extinctión. The fact that drive-inducing operations such as water/food deprivation are linked with physiological arousal has been documented by, among others, Belanger and Feldman (1962) whose data clearly showed that heart rate was a monotonic function of water deprivation. In addition, Rimm, Kennedy, Miller and Tchida (1971) subjected snake phobic subjects to one of five procedures designed to either increase or decrease arousal. Individuals subjected to arousal inducing operations (snake threat, frustration, shock threat) showed significantly greater snake avoidance on a subsequent behavior test than those who underwent arousal reducing operations (deep muscle relaxation, soft background music). Interestingly enough, an individual means comparison revealed that the relaxation vs soft background music condition was just shy of significance, whereas the control subjects who spent the interim period in an extremely comfortable easy chair evidenced a significantly greater snake-fear reduction than either the snake threat

or the frustration condition, but did not differ significantly from any other treatment mean. Similar differences were indicated by GSR change and peak scores. Of considerable theoretical significance is the fact that the arousal inducing or reducing operations took place in the absence of the phobic object.

Pretest gentling following the establishment of an approach-avoidance conflict was found by Altman and Hommel (1969) to effect a significant increment in running speeds, when compared with non-gentled controls. In conjunction with this analysis, Chapman and Feather (1971), employing a signal detection technique, reported that subjects undergoing muscle relaxation experienced significantly enhanced sensitivity to phobic imagery when compared with non-relaxed controls, among subjects suffering from public-speaking phobias. It appears then that a lowering of arousal may increase the saliency of the aversive cues, thus allowing for maximum exposure.

However, exposure alone may be a very potent variable. For instance, Vodde and Gilner (1971), employing a standardized filmed hierarchy for the purpose of equalizing exposure, assigned rat phobic students to one of five conditions: (1) muscle relaxation: the subjects received relaxation congruent with viewing the relevant hierarchy; (2) incentive: the subjects were paid to view the relevant hierarchy; (3) instructions: the subjects were instructed to view the relevant hierarchy; (4) muscle tension: the subjects experienced muscle tension while viewing the relevant hierarchy; (5) control: the subjects viewed an irrelevant hierarchy. Post-hoc comparisons following the behavioral avoidance test revealed that the relaxation, incentive, and instruction groups showed a similarly significant reduction in avoidance behavior.

In a recent review of infrahuman literature bearing on systematic desensitization, Wilson and Davison (1971) suggested that counterconditioning procedures expedite avoidance elimination by facilitating exposure to aversive stimuli. This conclusion was, in the main, based upon two interlocking lines of investigation. First, the data gleaned from the response prevention (flooding) literature (e.g., Baum, 1970; Polin, 1959) which clearly showed that forced inescapable exposure to aversive stimuli does lead to avoidance elimination; second, the results of a series of studies by Nelson (1966, 1967), which revealed that counterconditioning procedures have no effect on avoidance behavior unless they increase the subject's exposure to the aversive stimuli (but see Wilson & Dinsmoor, 1970).

Nelson's work is quite germane and merits additional attention. In his 1966 investigation (Experiment 1) Nelson replicated Lane's 1954 study with added controls. Lane had found, using an active avoidance task, that rats confined without food crouched at the escape door while the fed subjects roamed around the compartment alternating between the food dish which was placed in the center of the compartment and crouching near the escape door. Nelson fortuitously controlled for the possibility of increased exposure by placing his subjects directly over the food dish, a procedure which reinforced staying in that area. The test for residual fear revealed that the fed subjects showed significantly more fear than those that were confined. Experiment 2 (1966) lent further support to the interpretation that exposure and not counterconditioning accounted for fear reduction. Again using an active avoidance task, Nelson then added the following interpolated treatments between fear conditioning and the test for residual fear: (1) incentive: food was placed in the shock compartment and the

subjects were allowed to move from the safe compartment to the shock compartment; (2) no incentive: no food was present in the shock compartment but the subjects were allowed freedom of movement; (3) incentive control: the subjects were individually yoked for exposure with the incentive group, yet no food was present in the shock compartment; (4) no incentive control: the subjects were individually yoked for exposure with the no incentive group. The incentive group spent significantly more time in the fear compartment and made significantly more entries than the no incentive group. Also, the test for residual fear indicated that the incentive group showed significantly less fear than the no incentive group. It is also worth noting that the incentive group over the first 30 of 60 extinction trials showed less fear than the incentive controls. The author honestly noted that "this difference may reflect a counterconditioning effect, one which is secondary to the effect of food on exposure (p. 213)." Related data were also offered by Nelson (1967, Experiment 1). After acquiring an active avoidance response, subjects either drugged (chlorpromazine) or undrugged were placed in the fear compartment. The subjects which received high and medium doses (10 and 5 mg respectively) of chlorpromazine spent significantly more time in the fear side (all subjects were allowed freedom to cross into the safe side) than those which either received no drug or a low (1.25 mg) amount. Moreover, the escape speeds for residual fear indicated that the non-drugged and 1.25 mg groups were significantly more fearful than the 5 mg and 10 mg groups, which did not differ significantly from each other. Further support was offered by Experiment 2, which involved forced exposure, either drugged (5 mg) or undrugged, to the aversive stimuli. The results indicated no differential effect as to drugged or nondrugged conditions. The impact of

these results was lessened only by the author's failure to include untreated controls (1966, Experiment 2; 1967, Experiments 1 & 2) and the possible confounding which may result from active avoidance paradigms.

Strangely enough, Wolpe himself (1958) may have offered some incidental evidence favoring exposure as a critical factor in eliminating avoidance behavior. He noted, " ...anxiety reactions were much more rapidly eliminated when the pellets were tossed at widely distributed points than if they were confined to the food box" (p. 57). However, the explanatory burden rested on the Hullian concept of conditioned inhibition. Hull (1943) postulated the development of two forms of inhibition: an innate, fatigue-component reactive inhibition (I_R) and a learned component, conditioned inhibition (S^IR). The former is clearly conceived of and defined as a negative drive state while the latter is considered a negative habit. The reduction of this negative drive is reinforcing; the cessation of activity is conditioned to cues associated with its reduction; therefore, cues associated with the reduction of I_R come to evoke S^IR, the response of not responding. The early development of reactive and conditioned inhibition was acknowledged by Hull (1943) and others (e.g., Logan, 1959; Spence, 1966) to represent largely a blueprint and not a complete edifice. Consequently, there are a number of difficulties with the mechanisms which have not been resolved (for example, see Jones, 1958).

Nevertheless, Wolpe (1952, 1958) in a massive and aesthetically rude extrapolation assumed that conditioned inhibition (the negative habit of responding in a non-fearful manner) developed on the basis of reciprocal inhibition since anxiety generated too little reactive inhibition. Hence, the evocation of an antagonistic response in the presence of anxiety-

provoking cues (the procedure of counterconditioning) was purported to have neurophysiological consequences (reciprocal inhibition) which laid the basis for learned or conditioned inhibition. The inhibition was said to be learned in that with repetition--the evocation and subsequent dampening of the anxiety response--more reciprocal inhibition and therefore more conditioned inhibition was said to accrue, weakening the anxiety potential of the stimuli.

Also, it is well worth remembering that there was relatively little extinction data available when Hull formulated the concept of conditioned inhibition. Moreover, the emergence of the concept was based on two simple types of learning: instrumental discrete trial conditioning and classical conditioning. In both procedures, there is a deliberate and systematic attempt on the part of the experimenter to minimize the occurrence of competing responses and to limit the investigation to the strengthening of a single response, one that enjoyed a relatively high position in the initial response hierarchy.

On the other hand, response competition involves a deliberate and systematic attempt to introduce response competition and perforce to pit two responses in conflict (Spence, 1956). This present analog[1] of phobic behavior, passive avoidance conditioning, would seem to involve response competition. Consequently, the concept of conditioned inhibition is, by virtue of Wolpe's extrapolation, overburdened. The problems are manyfold: one, a clear explication of Wolpe's basic principle is that the link between the dominant response (not responding in passive avoidance, or responding in active avoidance) and the stimuli evoking it is weakened. How is this accomplished? Also, it is implicit in Wolpe's reciprocal

inhibition principle that the accrual of conditioned inhibition was dependent upon the development of reciprocal inhibition, ergo upon the number of counterconditioning trials. From this one would have predicted a rank-ordering in the present study of the CCH, CCM, and CCL groups in relation to passive avoidance reduction. This was not the case. One might on a purely ad hoc basis assume that conditioned inhibition had reached asymptote. Yet, an "explanation" of this nature is hardly the hallmark of a well articulated intervening variable theory, one whose predictive and explanatory powers rest on deductive derivation. Finally, the dramatic reduction in passive avoidance evidenced by the UTC/HC group does not have a well-defined place in Wolpe's model.

This critique is meant neither to imply that conditioned inhibition is not demonstrable (see Bull & Overmier, 1968; Rescorla, 1969; Rescorla & LoLordo, 1965); nor should it be construed as a misplaced vituperation against the immese pragmatic worth of systematic desensitization; rather it is a suggestion that Wolpe's theoretical considerations are less than complete.

It appears, then, that the models proposed by Lader & Mathews (1968) and Wilson and Davison (1971) may serve as rival hypotheses in accounting for the results of this investigation and those previously cited (e.g., Rimm, et al., 1971). The maximal habituation hypothesis predicts that any operation which lowers arousal level will lead to a reduction of avoidance behavior. The decided decrement in passive avoidance behavior shown by the UTC/HC, CCH, and CCM groups is in accord with this prediction. It is equally obvious that the Extinction and UTC/TC subjects underwent procedures that were likely to maintain or even raise their arousal level.

The variation in behavior of the UTC/HC and UTC/TC groups was not at all unlike the deep muscular relaxation group and the snake threat group in the 1971 investigation by Rimm, et al., or the gentled and non-gentled subjects in the 1969 study by Altman and Hommel. The greater but non-significant reduction in passive avoidance behavior displayed by the CCL group (which may be compared with Nelson's low chlorpromazine group) in comparison with the Extinction group could be indicative of an insufficient reduction in arousal.

On the other hand, non-punished exposure to the aversive stimuli (Rachman, 1968; Seligman, Maier & Greer, 1968; Wilson & Davison, 1971) may play a significant role. For example, the Implosion subjects, that experienced maximum exposure, showed passive avoidance reduction that was not unlike those subjects that experienced reduced exposure and lowered arousal.

The prediction resulting from a combination of these models is then obvious: the most rapid diminution in avoidance behavior should occur when an individual is subjected to an arousal-reducing operation and also experiences maximum non-punished exposure to the aversive stimuli.

References

Altman, H. B., & Hommel, L. S. The reduction of avoidance behavior as a function of gentling. Psychonomic Science, 1969, 14, 91-92.

Bandura, A. Principles of behavior modification. New York: Holt, 1969.

Baum, M. Extinction of avoidance responding through response prevention (flooding). Psychological Bulletin, 1970, 74, 276-284.

Belanger, D., & Feldman, S. M. Effects of water deprivation upon heart rate instrumental activity in the rat. Journal of Comparative and Physiological Psychology, 1962, 55, 220-225.

Blanchard, R. J., & Blanchard, D. C. Crouching as an index of fear. Journal of Comparative and Physiological Psychology, 1969, 67, 370-375.

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

Chapman, C. R., & Feather, B. W. Sensitivity to phobic imagery: a sensory decision theory analysis. Behaviour Research and Therapy, 1971, 9, 161-168.

Cooke, G. Evaluation of the efficacy of the components of reciprocal inhibition psychotherapy. Journal of Abnormal Psychology, 1968, 73, 464-467.

Davison, G. C. Systematic desensitization as a counterconditioning process. Journal of Abnormal Psychology, 1968, 73, 91-99.

Evans, I., & Wilson, G. T. Note on the terminological confusion surrounding systematic desensitization. Psychological Reports, 1968, 22, 187-191.

Gale, D. S., Sturmfels, G., & Gale, E. W. A comparison of reciprocal inhibition and experimental extinction in the therapeutic process. Behaviour Research and Therapy, 1966, 4, 149-156.

Gambrill, E. Effectiveness of the counterconditioning procedure in eliminating avoidance behavior. Behaviour Research and Therapy, 1967, 5, 263-273.

Goldstein, A. J. Separate effects of extinction, counterconditioning, and progressive approach in overcoming fear. Behaviour Research and Therapy, 1969, 7, 47-56.

Greenhouse, S. W. & Geisser. On methods in the analysis of profile data. Psychometrika, 1959, 24, 95-112. Cited in Winer, B. J. Statistical principles in experimental design. New York: McGraw-Hill, 1962.

Hall, J. C. Some conditions of anxiety extinction. Journal of Abnormal and Social Psychology, 1955, 51, 126-132.

Hull, C. L. Principles of behavior. New York: Appleton-Century-Crofts, 1943.

Jones, H. G. The status of inhibition in Hull's system: A theoretical revision. Psychological Review, 1958, 65, 179-182.

Kimble, G. A. Hilgard and Marquis' conditioning and learning. New York: Appleton-Century-Crofts, 1961.

Klein, B. Counterconditioning and fear reduction in the rat. Psychonomic Science, 1969, 17, 150-151.

Lader, M. H. Palmar conductance measures in anxiety and phobic states. Journal of Psychosomatic Research, 1967, 11, 271-281.

Lader, M. H. & Mathews, A. M. A physiological model of phobic anxiety and desensitization. Behaviour Research and Therapy, 1968, 6, 411-412.

Lane, B. R. The reduction of anxiety under three experimental conditions. Unpublished doctoral dissertation. Columbia University, 1954. Cited in Nelson, F. Effects of chlorpromazine on fear extinction. Journal of Comparative and Physiological Psychology, 1969, 69, 682-687.

Lang, P. J., Melamed, B. G., & Hart, J. A psychophysiological analysis of fear modification using an automated desensitization procedure. Journal of Abnormal Psychology, 1970, 76, 220-234.

Logan, F. A. The Hull-Spence approach. In S. Koch (Ed.), Psychology: a study of a science. Vol. 2. General systematic formulations, learning, and special processes. New York: McGraw-Hill, 1959, 293-358.

Melvin, K., & Brown, J. Neutralization of an aversive light stimulus as a function of number of paired presentations with food. Journal of Comparative and Physiological Psychology, 1964, 58, 350-353.

Nelson, F. Effects of two counterconditioning procedures on the extinction of fear. Journal of Comparative and Physiological Psychology, 1966, 62, 208-213.

Nelson, F. Effects of chlorpromazine on fear extinction. Journal of Comparative and Physiological Psychology, 1969, 69, 682-687.

Paul, G. L. Outcome of systematic desensitization. II. Controlled investigations of individual treatment, technique, and current status. In C. M. Franks (Ed.), Behavior therapy: Appraisal and status. New York: McGraw-Hill, 1969.

Perin, C. T. Behavior potentiality as a joint function of the amount of training and the degree of hunger at the time of extinction. Journal of Experimental Psychology, 1942, 30, 93-113.

Polin, A. T. The effects of flooding and physical suppression as extinction techniques on an avoidance locomotor response. Journal of Psychology, 1959, 47, 235-245.

Poppen, R. Counterconditioning of conditioned suppression in rats. Psychological Reports, 1970, 27, 659-671.

Rachman, S. Studies in desensitization: I. The separate effects of relaxation and desensitization. Behaviour Research and Therapy, 1965, 3, 245-252.

Rachman, S. Studies in desensitization: II. Flooding. Behaviour Research and Therapy, 1966, 4, 1-6.

Rachman, S. The role of muscular relaxation in desensitization therapy. Behaviour Research and Therapy, 1968, 6, 159-166.

Rescorla, R. A. Conditioned inhibition of fear resulting from negative CS-US contingencies. Journal of Comparative and Physiological Psychology, 1969, 67, 504-509.

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

Rimm, D. C., Kennedy, T. D., Miller, H. L., & Tchida, G. R. Experimentally manipulated drive level and avoidance behavior. Journal of Abnormal Psychology, 1971, 78, 43-48.

Seligman, M. E. P., Maier, S. E., & Geer, J. The alleviation of learned helplessness in the dog. Journal of Abnormal Psychology, 1968, 73, 256-262.

Sermat, V., & Shepard, A. H. The effects of a feeding procedure on persistent avoidance responses in rats. Journal of Comparative and Physiological Psychology, 1959, 52, 206-211.

Spence, K. W. Behavior theory and conditioning. New Haven: Yale University Press, 1956.

Spence, K. W. Cognitive and drive factors in the extinction of the conditioned eye blink in human subjects. Psychological Review, 1966, 73, 445-458.

Stampfl, T. G., & Levis, D. J. Essentials of implosive therapy: A learning-theory-based psychodynamic behavioral therapy. Journal of Abnormal Psychology, 1967, 72, 496-503.

Van Egeren, L. F. (In press) Psychophysiology of systematic desensitization: the habituation model. Journal of Behavior Therapy & Experimental Psychiatry. Cited in Van Egren, L. F. Psychophysiological aspects of systematic desensitization. Some outstanding issues. Behaviour Research and Therapy, 1971, 9, 65-77.

Vodde, T. W., & Gilner, F. H. The effects of exposure to fear stimuli on fear reduction. Behaviour Research and Therapy, 1971, 9, 169-176.

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.

Williams, S. B. Resistance to extinction as a function of the number of reinforcements. Journal of Experimental Psychology, 1938, 23, 506-522.

Willis, R. W., & Edwards, J. A. A study of the comparative effectiveness of systematic desensitization and implosive therapy. Behaviour Research and Therapy, 1969, 7, 387-395.

Wilson, E. H., & Dinsmoor, J. A. Effect of feeding on "fear" as measured by passive avoidance in rats. Journal of Comparative and Physiological Psychology, 1961, 54, 275-278.

Wilson, G. T., & Davison, G. C. Processes of fear reduction in systematic desensitization: Animal studies. Psychological Bulletin, 1971, 66 1-14.

Wolpe, J. Experimental neurosis as a learned behavior. British Journal of Psychology, 1952, 43, 243-268.

Wolpe, J. Psychotherapy by reciprocal inhibition. Stanford, California: Stanford University Press, 1958.

Wolpin, M., & Raines, J. Visual imagery, expected roles and extinction as possible factors in reducing fear and avoidance behavior. Behaviour Research and Therapy, 1966, 4 25-38.

Yates, A. J. Behavior therapy. New York: John Wiley & Sons, 1970.

TABLE 1

Means Per Subject over Three Days for Four Dependent Measures (Times Shown in Seconds)

Group	IEL	NE	PCL	TOB
Implosion	1654.24	17.24	2283.75	486.75
CCH	1670.24	18.74	2460.75	579.00
CCM	1773.25	23.62	2198.25	555.75
CCL	2722.25	9.12	3285.75	165.75
Extinction	2944.24	5.50	3321.00	127.50
UTC/HC	1983.37	17.00	2344.25	609.00
UTC/TC	3269.24	2.87	3551.25	39.75
NSC[a]	12.16	56.98	15.00	2123.00

a(n=6)

Footnotes

[1]Furthermore, this analog contains elements of an instrumental discrete trial and a free-operant procedure. Hullian learning theory is clearly applicable only to instrumental discrete trial procedures (Spence, 1956).

APPENDIX A

PROSPECTUS

Appendix A

AN ANALYSIS OF WOLPE'S RECIPROCAL INHIBITION PRINCIPLE

Systematic desensitization, developed by Joseph Wolpe in the early 1950's, is the most widely used form of behavior therapy. The therapy, which on a purely operational level may be described as the deconditioning of maladaptive behaviors, evolved following a series of animal studies in which Wolpe administered inescapable shock to food deprived cats. The effects were quite uniform across all Ss, viz., "(1) Resistance to being put into the experimental case; (2) signs of anxiety when inside the cage (muscular tension and mydriasis were invariable); (3) refusal to eat meat pellets anywhere in the experimental cage even after one, two or three days' starvation." (Wolpe, 1958, p. 52).

The conditioned fear was overcome by exposing and feeding the animals in situations which gradually approximated the chamber in which the fear was originally conditioned. Movement up the hierarchy was not instituted until all signs of anxiety in the current situation were eliminated. The treatment was uniformly successful.

Wolpe concluded that the anxiety responses (e.g., whining, urinating) were inhibited by eating. The inhibition was learned or conditioned, in that with repetition (pairing of the least to most potent anxiety evoking stimuli with eating) more conditioned inhibition accrued and the anxiety-evoking potential of the stimuli was progressively diminished.

Wolpe further assumed that this diminution was best conceptualized as conditioned inhibition based upon reciprocal inhibition. The term reciprocal

inhibition was first introduced by Sherrington (1906) in reference to the inhibition of one spinal reflex by the evocation of another, e.g., whenever the flexor muscles of the arm contract, the extensor muscles relax, or vice versa. The concept was originally used by Wolpe as the neurological process producing the diminution in fear shown by his cats. The behavioral prediction was succinctly stated by Wolpe (1958) in his basic principle:

> "If a response antagonistic to anxiety can be made to occur in the presence of anxiety-evoking stimuli so that it is accompanied by a complete or partial suppression of the anxiety responses, the bond between these stimuli and the anxiety responses will be weakened. (p. 71)."

In a word, a competing, alternative, incompatible or antagonistic response is made to interfere with the response that one wishes to abolish. The basic assumption is that the performance of one response prevents the performance of another behavior. Wolpe's basic principle or reciprocal inhibition principle as it is sometimes called, is nothing more than a descriptive definition of counterconditioning as seen by the definitions offered by Bandura (1969), "...eliciting activities that are incompatible with emotional responses in the presence of fear--or anxiety--arousing stimuli" (p. 424); Kimble, "Extinction under circumstances in which the response decrement is hastened by the reinforcement of a response which displaces the original conditioned response (Kimble, 1961, p. 478)."; Yates, "The terms reciprocal inhibition and counterconditioning are equivalent terms referring to procedures that strengthen alternative (or new) stimuli to which maladaptive responses are attached (1970, p. 65)." Wolpe has in fact recognized the similarity, and uses the terms reciprocal

inhibition and counterconditioning interchangeably, but has indicated some preference for the former in view of his inferences about the neurological process accounting for the observed change in behavior.

In the early 1950's Wolpe combined the procedure from his animal studies into a treatment package for human anxiety which he called systematic desensitization, in which imaginal representations of the anxiety provoking stimuli were substituted for actual, in vivo, exposure, and Jacobson's (1938) training in deep muscle relaxation replaced eating as the major incompatible response. The typical procedure is as follows: in preliminary sessions the client is given training in deep muscle relaxation and constructs an anxiety hierarchy under the tutelage of the therapist. During actual treatment, the client while relaxed is asked to visualize the least potent item in the hierarchy of emotion arousing stimuli. If any anxiety is experienced the client signals, is instructed to drop the scene and relaxation is perforce reinstated; the scene is repeatedly presented until it ceases to evoke anxiety. The process is then repeated with a scene which is slightly more disturbing, until the entire hierarchy has been neutralized.

It is noteworthy that the procedure is in no way standardized for such variables as duration and number of relaxation training sessions, duration of scene presentation, criteria for progression within the hierarchy (Paul, 1969). In addition, the reader will remember that Wolpe's cats were subjected to a forced counterconditioned procedure, entailing a prevention of the avoidance response, whereas the client is instructed to signal if the image evokes anxiety and to drop the image, thereby performing an escape response. A similar conclusion was reached in a recent paper by Wilson and Davison (1971). This point will merit further attention later in this paper.

Although deep muscle relaxation represents the most ubiquitous antagonistic response, others are employed and an attempt to enumerate them would be somewhat akin to listing the paradigms of learning or the galaxy of positive reinforcers; the anxiety inhibitors are peculiar to specific fears so that, for example, if an individual is overapologetic or afraid to ask for what is rightly his, he is encouraged to assert himself and express what he really wants. The assertive responses are said to reciprocally inhibit the submissive responses (since one can hardly display submission and assertion in a simultaneous fashion). The repetition of assertion responses is said to bring about the progressive conditioned inhibition of submissive behaviors (Wolpe, 1958). Other incompatible responses include sexual responses (e.g., Wolpe & Lazarus, 1966); relaxation afforded by the therapeutic setting (Rachman, 1968); appetising foods (Jones, 1924); pleasant imagery (Lazarus & Abramovitz, 1962); drugs (Friedman, 1968); the presence of a mother surrogate (Harlow, 1959).

In summary, Wolpe considers three sets of variables essential for achieving consistent results:

(1) the competing response (in most cases deep muscle relaxation) must serve as an anxiety inhibitor:

(2) anxiety to low hierarchy items must be dissipated before one proceeds to the next item on the hierarchy;

(3) temporal contiguity between antagonistic response and aversive stimuli.

As will be shown later, Wolpe's therapeutic method has enjoyed an amazing amount of success (cf Paul, 1969); yet the theoretical explanation concerning the acquisition of anxiety and subsequent alleviation are largely unsupported.

For instance, the notion of a learned inhibition of responding is, of course, reminiscent of Hull's (1943) conceptualization of factors affecting response decrement. Hull postulated the development of two forms of inhibitions: an innate, fatigue-procedure reactive inhibition (I_R) and conditioned inhibition (S^IR). The former is clearly conceived of an defined as a negative drive state while the latter is considered a negative habit. The reduction of this negative drive is reinforcing; this cessation of activity is conditioned to cues associated with its reduction; therefore, cues associated with the reduction of I_R come to evoke S^IR, the active response of not responding.

Furthermore, in accord with most theories of psychopathology Wolpe (1958) adopted the position that anxiety was the major causal agent, and results from sympathetic nervous system overdominance. Incompatible responses brought about by counterconditioning elicit parasympathetic nervous system responses which supposedly reciprocally inhibit the sympathetic nervous system responses which supposedly reciprocally inhibit the sympathetic nervous system dominance and the role of muscular relaxation has come under recent attack, (e.g., Davison, 1966, 1968). So as not to confuse levels of discourse one must understand that the efficacy of the treatment based on a counterconditioning principle is independent of the validity of Wolpe's neurophysiological speculation (Bandura, 1969).

Despite the fact that there is now good evidence for the effectiveness of systematic desensitization (cf Paul, 1969) there remains, as noted in the following review, the significant question as to whether this efficacy is correctly predicted by counterconditioning; moreover, the most obvious implications of Wolpe's counterconditioning principle, viz, that a

relationship (albeit unspecified) should exist between the amount of counterconditioning and the diminution of fear, has completely escaped attention.

With regard to human subjects, some researchers in attempting to assess the counterconditioning hypothesis have rendered their results uninterpretable by the omission of pre-test scores, e.g., Cooke (1968), or by the addition of another facet such as muscle tension, e.g., Wolpin and Raines (1966), the use of extremely small Ns, e.g., Rachman, 1965. Unambigous support for a counterconditioning hypothesis has been offered by Davison (1968). A similar picture emerges from infrahuman literature, in which the counterconditioning hypothesis has been investigated using a wide galaxy of experimental procedures, viz, CER (Gale, Strumfels, & Gale, 1966; Poppen, 1970); passive avoidance (Goldstein, 1969; Wilson & Dinsmoor, 1970); active avoidance (Julia Hall, 1955; Klein, 1969; Melvin & Brown, 1964; Nelson, 1966; Sermat & Shepard, 1959); Sidman avoidance (Gambrill, 1967). In a number of cases the reduction in avoidance behavior brought about by counterconditioning has been transient (Gale, et al., 1966; Gambrill, 1967; Melvin & Brown, 1964; Sermat & Shepard, 1959), or the interpretation of the results rendered somewhat dubious by the experimenter's failure to include untreated controls (Poppen, 1970; Wilson & Dinsmoor, 1970).

Moreover, as noted by Wilson and Dinsmoor (1970), the use of an active avoidance (crossing from the former shock to safe side) to assess the effects of counterconditioning (typically feeding in the formerly shock side) is easily confounded by the fact that reduced latencies displayed by the counterconditioned subjects may be the result of secondary reinforcement

set up by feeding in the shock area, a similar conclusion also reached by Melvin and Brown (1964), or crouching or freezing which results from shock and is obviously incompatible with active responding, or both. The latter hypothesis, as noted by Wilson and Dinsmoor (1970), was also expressed by Blanchard and Blanchard (1969) and Weiss, Kriechaus and Conte (1968).

Hence, the purpose of the present study was to: (1) investigate the counterconditioning hypothesis, employing appropriate controls: (2) investigate its most obvious implication, i.e., that a relationship (albeit unspecified) should exist between the degree of counterconditioning and the subsequent reduction of avoidance behavior; (3) compare the efficacy of implosion or response prevention (e.g., Baum, 1970; Stampfl & Levis, 1968), with varying degrees of counterconditioning, using a passive avoidance procedure.

References

Bandura, A. Principles of behavior modification. New York: Holt, 1969.

Baum, M. Extinction of avoidance responding through response prevention (flooding). Psychological Bulletin, 1970, 74, 276-284.

Blanchard, R. J. & Blanchard, D. C. Crouching as an index of fear. Journal of Comparative and Physiological Psychology, 1969, 67, 370-375.

Cooke, G. Evaluation of the efficacy of the components of reciprocal inhibition psychotherapy. Journal of Abnormal Psychology, 1968, 73, 464-467.

Davison, G. C. Anxiety under total curarerization: implications for the role of muscular relaxation in the desensitization of neurotic fears. Journal of Nervous and Mental Disease, 1966, 143, 443-448.

Davison, G. C. Systematic desensitization as a counterconditioning process. Journal of Abnormal Psychology, 1968, 73, 91-99.

Evans, I., & Wilson, G. T. Note on the terminological confusion surrounding systematic desensitization. Psychological Reports, 1968, 22, 187-191.

Friedman, D. E. Treatment of a case of dog phobia in a deaf mute by behavior therapy. Behaviour Research and Therapy, 1966, 4, 141.

Gale, D. S., Sturmfels, G., & Gale, E. W. A comparison of reciprocal inhibition and experimental extinction in the therapeutic process. Behaviour Research and Therapy, 1966, 4, 149-156.

Gambrill, E. Effectiveness of the counterconditioning procedure in eliminating avoidance behavior. Behaviour Research and Therapy, 1967, 5, 263-273.

Goldstein, A. J. Separate effects of extinction, counterconditioning, and progressive approach in overcoming fear. Behaviour Research and Therapy, 1969, 8, 47-56.

Hall, J. C. Some conditions of anxiety extinction. Journal of Abnormal and Social Psychology, 1955, 51, 126-132.

Harlow, H. F. Love in infant monkeys. Scientific American reprint, 1959, June.

Hull, C. L. Principles of Behavior. New York: Appleton-Century-Crofts, 1943.

Jacobson, E. Progressive relaxation. Chicago, University of Chicago Press, 1938, Cited in Wolpe, J. Psychotherapy by reciprocal inhibition. Stanford, California: Stanford University Press, 1958.

Jones, M. C. The elimination of children's fear. Journal of Experimental Psychology, 1924, 7, 382-390.

Kimble, G. A. Hilgard and Marquis' conditioning and learning. New York: Appleton-Century-Crofts, 1961.

Klein, B. Counterconditioning and fear reduction in the rat. Psychonomic Science, 1969, 17, 150-151.

Lazarus, A. A., & Abramovitz, A. The use of "emotive imagery" in the treatment of children's phobias. Journal of Mental Science, 1962, 108, 191-195. Cited in C. M. Franks (Ed.), Behavior therapy: Appraisal and status. New York: McGraw-Hill, 1969.

Melvin, K., & Brown, J. Neutralization of an aversive light stimulus as a function of number of paired presentations with food. Journal of Comparative and Physiological Psychology, 1964, 58, 350-353.

Nelson, F. Effects of two counterconditioning procedures on the extinction of fear. Journal of Comparative and Physiological Psychology, 1966, 62, 208-213.

Paul, G. L. Outcome of systematic desensitization. II. Controlled investigations of individual treatment, technique variations, and current status. In C. M. Franks (Ed.), Behaviour therapy: Appraisal and status. New York: McGraw-Hill, 1969.

Poppen, R. Counterconditioning of conditioned suppression in rats. Psychological Reports, 1970, 27, 659-671.

Rachman, S. Studies in desensitization: I. The separate effects of relaxation and desensitization. Behaviour Research and Therapy, 1965, 3, 245-252.

Rachman, S. The role of muscular relaxation in desensitization therapy. Behaviour Research and Therapy, 1958, 6, 159-166.

Sermat, V., & Shepard, A. H. The effects of a feeding procedure on persistent avoidance responses in rats. Journal of Comparative and Physiological Psychology, 1959, 52, 206-211.

Sherrington, C. S. Integrative action of the nervous system. New Haven: Yale University Press, 1960. Cited in Wolpe, J. Psychotherapy by reciprocal inhibition. Stanford, California: Stanford University Press, 1958.

Stampfl, T. G., & Levis, D. J. Essentials of implosive therapy: a learning-theory-based psychodynamic behavioral therapy. Journal of Abnormal Psychology, 1967, 72, 496-503.

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.

Wilson, E. H., & Dinsmoor, J. A. Effect of feeding on "fear" as measured by passive avoidance in rats. Journal of Comparative and Physiological Psychology, 1961, 54, 275-278.

Wolpe, J. Psychotherapy by reciprocal inhibition. Stanford, California: Stanford University Press, 1958.

Wolpe, J., & Lazarus, A. A. Behavior therapy techniques: a guide to the treatment of neuroses. London: Pergamon Press, 1966. Cited in C. M. Franks, (Ed.), Behavior therapy: Appraisal and status. New York: McGraw-Hill, 1969.

Wolpin, M. & Raines, J. Visual imagery, expected roles and extinction as possible factors in reducing fear and avoidance behavior. Behaviour Research and Therapy, 1966, 4, 25-38.

APPENDIX B

ANOVAS

IEL

Source	df	MS	F
A (Treatments)	6	1210269.62	4.54**
S/A (Error)	49	266348.64	
B (Days)	2	5067148.69	88.18***
A x B	12	138495.60	2.10 NS
B x S/a (Error)	98	65651.68	

NE

Source	df	MS	F
A (Treatments)	6	156.62	2.77*
S/A (Error)	49	56.50	
B (Days)	2	777.21	63.99***
A x B	12	29.38	2.41 *
B x S/a (Error)	98	12.14	

PCL

Source	df	MS	F
A (Treatments)	6	897668.54	4.21**
S/A (Error)	49	212924.76	
B (Days)	2	3345238.56	47.47***
A x B	12	199305.52	2.82*
B x S/a (Error)	98	70462.72	

TOB

Source	df	MS	F
A (Treatments)	6	159299.85	2.95 *
S/A Error)	49	53871.67	
B (Days)	2	642758.35	49.66***
A x B	12	34203.60	2.64*
B x S/a (Error)	98	12942.14	

*$p < .05$

**$p < .01$

***$p < .001$

Lick Data Seven Minutes

Source	df	MS	F
A(Group)	6	73691.39	.71 NS
Error	49	103189.84	

Lick Data Ten Minutes

Source	df	MS	F
A(Group)	6	2439413.85	.90 NS
Error	49	2703575.07	

Lick Data Fifteen Minutes

Source	df	MS	F
A(Group)	6	453827115.12	1.03 NS
Error	49	440304261.00	

APPENDIX C

TABLES

Cumulative Lick Data, Seven, Ten, and Fifteen Minute Intervals, Expressed in Means per Treatment Group

Tmt	7 min.	10 min.	15 min.
Implosion	1122.86	1604.25	1900.75
CCH	1105.62	1234.66	1693.75
CCM	945.24	1333.25	1613.37
CCL	893.74	1167.00	1611.87
Extinction	1064.13	1474.12	1652.12
UTC/HC	1069.00	1331.62	1816.87
UTC/TC	912.36	1187.50	1495.75

Means and Standard Deviations for IEL Across Three Days

Tmt	Day 1 $\bar{X}$	Day 1 SD	Day 2 $\bar{X}$	Day 2 SD	Day 3 $\bar{X}$	Day 3 SD	Cum $\bar{X}$	Cum SD
Implosion	1032.12	365.44	516.25	533.80	105.87	67.58	1654.24	966.82
CCH	979.12	379.73	490.00	452.71	201.12	232.08	1670.24	1064.52
CCM	1048.25	353.20	485.50	474.11	239.50	392.48	1773.25	1219.79
CCL	1118.25	231.22	972.50	365.14	631.50	464.83	2722.25	1061.19
Extinction	1200.00	0.00	1012.87	311.97	731.37	400.15	2944.24	712.12
UTC/HC	877.75	380.99	655.75	583.08	449.87	473.60	1983.37	1437.67
UTC/TC	1200.00	0.00	1184.12	44.90	885.12	286.57	3269.24	331.47
NSC	4.50	4.42	3.16	1.67	4.50	2.42	12.16	18.51

Means and Standard Deviations for NE Across Three Days

Tmt	Day 1 $\bar{X}$	Day 1 SD	Day 2 $\bar{X}$	Day 2 SD	Day 3 $\bar{X}$	Day 3 SD	Cum $\bar{X}$	Cum SD
Implosion	0.62	1.40	5.62	4.98	11.00	7.46	17.24	13.84
CCH	0.87	1.35	5.50	6.27	12.37	6.50	18.74	14.12
CCM	2.37	5.92	7.50	7.55	13.75	8.77	23.62	22.24
CCL	0.75	1.48	1.75	2.91	6.62	8.21	9.12	12.60
Extinction	0.00	0.00	0.50	0.53	5.00	5.01	5.50	5.54
UTC/HC	2.75	3.69	6.50	7.19	7.75	7.06	17.00	17.94
UTC/TC	0.00	0.00	0.25	0.70	2.62	3.29	2.87	3.99
NSC	18.66	13.19	19.16	4.38	19.16	2.41	56.98	9.98

Means and Standard Deviations for PCL Across Three Days

Tmt	Day 1 $\bar{X}$	Day 1 SD	Day 2 $\bar{X}$	Day 2 SD	Day 3 $\bar{X}$	Day 3 SD	Cum $\bar{X}$	Cum SD
Implosion	1200.00	0.00	705.00	540.60	378.75	509.53	2283.75	1050.13
CCH	1200.00	0.00	767.25	427.27	493.50	479.61	2460.75	906.88
CCM	1077.00	347.89	779.25	503.61	342.00	385.84	2198.25	1237.34
CCL	1200.00	0.00	1181.25	53.03	904.50	424.64	3285.75	477.67
Extinction	1200.00	0.00	1200.00	0.00	921.00	393.31	3321.00	393.31
UTC/HC	1098.50	141.14	682.50	554.16	563.25	552.33	2344.25	1247.63
UTC/TC	1200.00	0.00	1200.00	0.00	1151.25	137.88	3551.25	137.88
NSC	6.00	5.42	4.00	1.41	5.00	3.31	15.00	10.14

Means and Standard Deviations for TOB Across Three Days

Tmt	Day 1 $\bar{X}$	Day 1 SD	Day 2 $\bar{X}$	Day 2 SD	Day 3 $\bar{X}$	Day 3 SD	Cum $\bar{X}$	Cum SD
Implosion	6.00	14.69	166.50	190.02	314.25	241.23	486.75	445.04
CCH	14.25	25.64	170.25	194.89	394.50	262.56	579.00	483.09
CCM	39.75	107.61	180.00	191.43	336.00	184.68	555.75	483.72
CCL	4.50	12.72	24.75	46.85	136.50	232.94	165.75	292.51
Extinction	0.00	0.00	4.50	5.31	123.00	157.83	127.50	163.14
UTC/HC	72.00	112.88	246.75	266.02	290.25	324.42	609.00	703.32
UTC/TC	0.00	0.00	1.50	4.24	38.25	59.04	39.75	63.28
NSC	721.00	330.90	709.00	142.77	693.00	100.38	2123.00	574.05