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DISINHIBITION OF THE RABBIT'S CONDITIONED NICTITATING MEMBRANE RESPONSE

A Dissertation Presented

By

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Dedication

To my grandfather, Horace, and my parents, Horace, Jr. and Lygia: without whose collective guidance and support I would not have accomplished what I have.

Abstract

Disinhibition, the disruptive effect of a novel stimulus presentation upon an inhibitory process, was investigated utilizing the rabbit's conditioned nictitating membrane response (NMR). In Experiment 1 it was shown that the presentation of a novel back shock stimulus can produce CR magnitude increases during extinction of the NMR. In Experiment 2 it was shown, however, that an extinguished stimulus does not become a conditioned inhibitor. In Experiments 3-6 attempts were made to disrupt a conditioned inhibitor. Following failures in all such attempts, it was concluded that disinhibition might be better conceptualized as a disruption of attentional, rather than associative, processes. The results were discussed in relation to the theories of the inhibitory process posed by Pavlov (1927), Hull (1943), and Rescorla and Wagner (1972).

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INTRODUCTION

A: CONDITIONED INHIBITION

1: Pavlov's work

The investigation of conditioned inhibition (CI) was instigated by Pavlov in Russia around the turn of the century (Pavlov, 1927). For Pavlov, inhibition was a tendency on the part of an organism to not make a conditioned response (CR). Pavlov regarded such a tendency as biologically adaptive as it was a method through which the organism could save an unnecessary expenditure of energy (Pavlov, 1927, p. 106). Thus, for Pavlov the response tendencies of an organism, as well as its central nervous system,[1] were in constant state of conflict between the opposing processes of inhibition and excitation.

Pavlov differentiated between two primary types of inhibition—external and internal. External inhibition occurs when a novel stimulus is presented simultaneously with a previously established excitatory conditioned stimulus (CS+). The CS+, however, rather than eliciting the CR as it normally would is followed by no CR. This form of inhibition can be contrasted with internal inhibition, which Pavlov essentially regarded as being produced by systematic nonreinforcement of a CS. For example, if a CS+ is systematically followed by an absence of the unconditioned stimulus (UCS), it undergoes extinction

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[1] Pavlov's speculative account of cortical function is not, however, of concern in this paper.

and gradually the CS loses its ability to elicit a CR. Pavlov attributed this gradual disappearance of the CR to the development of an underlying inhibitory process. Thus, as the CS was consistently not reinforced--not followed by the UCS--the CS gradually acquired inhibitory properties as a function of the nonreinforcement and, finally, the inhibitory properties of the CS exceeded the previously established excitatory strength of the CS and the CS no longer produced a CR.

In support of his notion that extinction was due to active inhibitory process, Pavlov (1927, p. 87) cited the phenomenon of disinhibition. Pavlov demonstrated that if a novel stimulus (or the UCS) presentation preceded the presentation of an extinguished CS, the CS was followed by a sudden recurrence of the CR (Pavlov, p. 65). The next few presentations of the CS were again followed by a CR before it gradually disappeared and was no longer elicited by the CS. The occurrence of disinhibition, then, was labelled "inhibition of inhibition" and constituted support for his contention that extinction is due to an active inhibitory process being overlaid upon an existing excitatory process. When the inhibitory process is somehow disrupted, in this case by the presentation of a novel stimulus, the underlying excitatory process momentarily regains control of the organism and a CR is suddenly once again elicited by the CS. The gradual disappearance of the CR is then attributed to the reestablishment of the inhibitory process due to continued nonreinforcement of the CS.

Finally, Pavlov noted the seemingly paradoxical effects of a novel stimulus presentation. If the stimulus was presented prior to (or concurrent with) an excitatory stimulus, the underlying inhibitory process was exposed and the result was external inhibition. If, on the other hand, the novel stimulus was presented prior to the presentation of an inhibitory stimulus, the existing excitatory process was laid bare and disinhibition occurred.

2: Recent criteria for conditioned inhibition

Recent work on both the empirical and theoretical fronts has led to a number of generally agreed upon criteria for distinguishing between CI and other situations in which a CR does not occur. Since inhibition is regarded as a response tendency opposite that of excitation, the existence of an active inhibitory process implies that response strength has been reduced, in some sense, below zero. Yet, the nonoccurrence of a CR might be due to a lack of excitation--a response strength of zero--rather than an active inhibitory process. To differentiate between these two possible alternatives, a "retardation" test (Rescorla, 1969) is usually used. This test consists of comparing the excitatory acquisition rate of a suspected inhibitor with the acquisition rate of a stimulus which has zero response strength in some appropriate control group. For example, Marchant and Moore (1974), using a method known as Pavlovian Conditioned Inhibition (PCI), produced an inhibitor in the rabbit nictitating membrane response (NMR) preparation by pairing a light stimulus with the UCS, but not reinforcing the light if it were presented simultaneously with a tone. These experimental animals gradually made the appropriate discrimination, and it was suspected that the tone had become inhibitory. To test this notion, the acquisition rate to the tone--now reinforced--of this group was compared with a group (among others) of subjects for whom the tone was a novel stimulus. It was found that the acquisition rate of the experimental group was retarded relative to the "normal" (zero response strength) acquisition rate, providing justification for the assumption that the response strength to the tone in the experimental group lay below zero.

Even though the response strength of a stimulus lies below zero, it is possible that this is due to some nonassociative factors such as attention, fatigue, etc. Since we are concerned with conditioned inhibitors, we should also want some method of attempting to ensure that the inhibitory strength of a stimulus is not due to any of these nonassociative properties. Therefore, Marchant and Moore employed additional groups to control for experience in the apparatus, possible handling effects, experience with stimuli, etc. Although such groups may control for a number of factors, it does not seem likely that they are sufficient to control for attentional factors. That is, a retardation effect might be due to an attentional effect; if for some reason the inhibitory training procedure caused the animal to "filter out," "ignore," etc. the suspected inhibitor a retardation-like effect would be expected, since an animal actively ignoring a stimulus might be expected to learn to respond to it quite slowly.

To control, then, for attentional effects, a "summation test" (Rescorla, 1969) is generally employed. This test consists of pairing the suspected inhibitor with a stimulus known to be excitatory, in extinction. If the stimulus is, in fact, inhibitory, then the response rate to this compound should be less than the response rate to the excitatory stimulus by itself. If, however, the animal is ignoring the stimulus, one would expect the addition of such a stimulus to have no effect on response rate.[2]

Marchant, Mis and Moore (1972) subjected the tone stimulus from the previously mentioned light-tone compound CS- to a summation test. They found that, when the tone was paired with a previously excitatory conditioned white noise stimulus, the compound was responded to far less often in extinction than the white noise by itself. The tone also met a number of secondary criteria of inhibition suggested by Rescorla. In conclusion, then, when a light-tone CS- is contrasted with a light CS+, the tone acquires active inhibitory properties.

B: DISINHIBITION

The phenomenon of disinhibition has received spradic attention since Pavlov's pioneering work. A thorough literature review (see Appendix A) indicates that when disinhibition has been demonstrated, it has almost always been in a paradigm that has not been shown to meet the previously mentioned criteria delineated by Rescorla (1969).

For example, Razran (1939) had human _S_s acquire a salivatory response to a flashing light, then extinguished the response. During

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[2] Incidentally, exactly this attention-like effect seems to be the cause of so-called "latent inhibition" (Lubow and Moore, 1959; Solomon, _et al._, 1974).

the extinction trials, a buzzer preceding some of the trials produced an increment in response amplitude. This amplitude increase, specific to trials preceded by the novel stimulus, was considered to be a demonstration of disinhibition. Similarly, Hearst, Franklin and Mueller (1974) conditioned and extinguished a key peck response with pigeons utilizing a discrete-trial paradigm with a 25-second intertrial interval (ITI). Following five consecutive trials on which no pecks occurred, the ITI was switched to 5 seconds and key pecking was reinstated in most of the birds.

Each of these studies, however, utilized extinction as the procedure presumed to produce CI. A number of recent studies (Cousins, 1972; Henderson, 1973; Reberg, 1972; Weisman and Litner, 1969)[3] have demonstrated that an extinguished CS does not seem to become inhibitory. Instead, the CS seems to remain slightly excitatory or becomes neutral (controls zero response strength).

Although extinction situations are not the only ones that have been used to test for disinhibition, none of the other paradigms utilized have been demonstrated to produce a conditioned inhibitory stimulus. Thus, novel stimuli have been superimposed during an FI scallop (Singh and Wickens, 1968), conditioned suppression (Brimer, 1972), suppression produced by punishment (Brimer, 1972), etc. While disinhibition was demonstrated, none of these methods of reducing

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[3] Henderson (1973) utilized a CS which had suppressive effects of its own in the CER. Hence, he found that extinction merely produced a return to the initially suppressed baseline, rather than absolute neutrality.

response rate, however, have ever been shown to satisfy the criteria for demonstrating active inhibition outlined by Rescorla (1969) and Hearst (1972).

What seems to be the only study of disinhibition in a preparation presumed to produce an active inhibitory stimulus (PCI) was reported by Hunter (1938). In a series of studies using finger withdrawal avoidance, Hunter conditioned Ss to respond to one stimulus, but not to respond when that stimulus was compounded with another. A buzzer presentation to which the unconditioned startle, and subsequent finger withdrawal, response had been habituated was used as the disinhibitor. For example, in one experiment Ss were trained to differentiate between the reinforced presentation of a single light and the nonreinforced presentation of two lights. Following establishment of the discrimination,the startle response to the buzzer was habituated by presenting it during the 59 sec ITI and preceding both trial types. The buzzer was then presented simultaneously with the CS- compound and six of the seven Ss responded to the CS-. Similar experiments were performed using either visual presentation of the word "no" preceding a single light or, the phrase "Don't lift your finger" delivered via earphones preceding the phrase "Lift your finger" (CS+), as the CS-. Similar results were obtained in each experiment with 4/6 and 5/8 Ss showing disinhibition on the buzzer trial.

The disinhibitory effect demonstrated, however, is confounded by a failure to control certain independent variables. First, the subjects were generally not naive, since many of the subjects were used in more than one experiment. Of the 21 Ss used in the three experiments described above, only three were experimentally naive. A more serious problem, however, was the use of the buzzer as the disinhibiting stimulus. The buzzer produces the withdrawal response as an unconditioned effect. Despite the habituation of this startle response over trials, the habituation was done during the ITI and one might reasonably expect that the presentation of the buzzer in compound with CS- for the first time might produce dishabituation. Finally, no unstimulated control group was included to ensure that the "disinhibited" responses were not merely random variations in performance during the ongoing differentiation.

Thus, the only PCI study in the literature which purports to demonstrate disinhibition resulted in an increase in the probability of a CR. This disinhibitory effect, however, seems to have been actually caused by dishabituation of an habituated response.

Given the fact that there seems to be no compelling demonstration of disinhibition of a known inhibitory stimulus in the literature since Pavlov, the following series of experiments were conducted in an attempt to examine a number of issues. First, can disinhibition be demonstrated in extinction of the rabbit NMR? If so, does extinction of the NMR produce an actively inhibitory CS, or does extinction in this preparation produce results analogous to those in the other preparations mentioned? Finally, if disinhibition can be demonstrated in extinction, will the same set of parameters produce disinhibition in the PCI paradigm used by Marchant and Moore (1974), when the novel stimulus precedes an inhibitory stimulus?

Should it prove possible to reliably obtain disinhibition of an inhibitory stimulus, disinhibition should then function, along with summation and retardation results, as another method for attempting to quantify the "depth" of inhibition. Additionally, a successful demonstration of disinhibition would imply that either Pavlov's (1927) or Hull's (1943) models of the inhibitory process may be more appropriate than the recently developed Rescorla-Wagner model (Wagner and Rescorla, 1972). This point will be elaborated on during a later section.

GENERAL METHOD

Since subjects, apparatus, and parametric variables are held constant throughout the following experiments, a description of these follows:

Subjects. All Ss were experimentally naive New Zealand albino rabbits (Oryctolagus Cuniculus) weighing approximately 2 kg and obtained from a local supplier.

Apparatus. A detailed description of the apparatus is available elsewhere (e.g. Marchant, Mis and Moore, 1972). Briefly, up to four animals were run concurrently in individually ventilated and soundproofed, fireproof file drawers. Each rabbit was restrained in a plexiglas box like those described by Gormezano (1966). A "minitorque" potentiometer mounted on S's head was connected by a small metal hook and silk thread to a nylon loop sutured into the nictitating membrane of the right eye. Movement of the membrane produced a dc signal which was amplified and recorded on a Grass 5D oscillograph. A CR was defined as a pen deflection of 1 mm (corresponding to an extension of the membrane of less than 1 mm) during the 500 msec CS-UCS interval or within 550 msec following the onset of a nonreinforced CS.

The CSs were a pure tone (T) of 1,200 Hz and 85 db delivered via a speaker located directly in front of the rabbit's head and the onset of two 6v incandescent lights (L) mounted on either side of the speaker behind translucent white plastic screens. A 70 db white noise stimulus was constantly present to serve as masking noise. The UCS was an ac shock of 2 ma and 50 msec duration delivered through two stainless-steel wound clips (Clay-Adams 9 mm) applied to the infraorbital region of the right eye.

The back shock (B) stimulus, which was used as the disinhibiting novel stimulus, was a brief dc shock generated by a Variac (see Tintner and Moore, 1974) and delivered to S via 1.5 in. stainless steel safety pins inserted into the skin in the rabbit's back. The pins were inserted approximately 2 in. apart, parallel to the spine and approximately 5 in. behind the animal's neck. Pins were generally inserted on the day prior to disinhibition testing to avoid the possible complications of infection. Pin insertion had no noticeable effect on the rabbit's behavior. The B stimulus was 10v delivered 5 sec prior to the onset of a CS.[4] The ITI was a constant 30 sec and there were 100 trials per day, except during extra acquisition sessions.

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[4] B was presented during the ITI by E utilizing a stopwatch and a make-break switch.

EXPERIMENT 1

The purpose of Experiment 1 was to attempt to demonstrate disinhibition of the rabit NMR during extinction.

Procedure. Ss were 20 albino rabbits, sutured and habituated to the apparatus for approximately 15 minutes on the first day. Ss were then given six daily sessions of excitatory conditioning to an LT simultaneous compound. There then followed two days of extinction during which B preceded 25 (N=6) or 27 (N=4) of the 100 trials.[5] Ss were divided into experimental and control groups on the last day of acquisition with the two groups matched on the basis of total number of CRs made in acquisition. The function of the control animals was to ensure that any pattern of responding evidenced by experimental animals, such as an increase in response magnitude on trials following B presentation, was not merely random fluctuation. Hence, control Ss were treated exactly like experimental Ss except that the Variac was not turned on.

Finally, in this and other experiments which involved control Ss, the animals were assigned unsystematically to a different file cabinet drawer each day throughout the experiment. This was done to ensure that any rearranging of box order due to matching of groups would not constitute a novel event for the rabbit.

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[5] Because of the exploratory nature of this research, the latter four Ss received 50 L and 50 T trials with nine Bs preceding five of the L and four of the T trials on the first day of extinction. This resulted in rapid extinction, presumably due to generalization decrement, and only the second day's data, when only the compound was presented, are presented.

Results. Except for one animal, all Ss responded on 90% or better of the trials on Day 2 of acquisition. The last rabbit, however, attained a response level at 98% CRs on Day 3. The mean percentage CRs for the experimental group over six days was 89.2%, while for the control group it was 89.4%. While the two groups did not differ in acquisition, they did, however, differ in their performance during extinction. Magnitude measures, defined as amplitudes including those of 0 mm (see Gormezano and Moore, 1970, p. 126), were taken for each group on the trial preceded by B (Trial N) and on the trials before (N-1) and after (N+1). It was then ascertained for each animal whether magnitude increased or decreased on each possible comparison between trials N and N-1. Additionally, magnitude change was recorded for all possible comparisons between trials N-1 and N+1.[6] An "Up" (N-(N-1)>0), "Down" (N-(N-1)<0) difference score was then calculated for each animal for each day of extinction. Since the normal response strength decrease in extinction would be represented by more Downs than Ups, a tendency in the opposite direction in the experimental group would constitute evidence of disinhibition. As may be seen in Table 1, the control animals consistently show a tendency for CR magnitude to decrease over both days of extinction. In contrast, however, the experimental Ss exhibit a tendency in the opposite direction; that is, CR magnitude increases significantly more often in the experimental group than in

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[6] This form of comparison follows the traditional conceptualization of disinhibition as an increment in amplitude or magnitude of the CR following the presentation of the novel stimulus (e.g., Pavlov, 1927; Razran, 1939).

Table 1: Mean difference score for the Up/Down magnitude comparison over two days of testing in extinction. A positive value indicates magnitude was increasing more often than decreasing.

	Comparison	Group E	Group C	N	t-value
Day 1	N-1 vs N	3.3	-3.7	10	2.58, 18 df*
	N-1 vs N+1	1.4	-3.2	10	2.86, 18 df*
Day 2	N-1 vs N	2.2	-1.8	6	2.04, 10 df**
	N-1 vs N+1	1.5	-3.7	6	2.02, 10 df**

* $P<.02$, All t-tests are two-tailed

** $.05<P<.10$

Note: Because of the exploratory nature of this research, four <u>S</u>s received B only nine times on Day 1. The data from these <u>S</u>s were therefore added combined with those of the other six animals for Day 1. Since the four <u>S</u>s were not given a third day of testing, no data were available from them for Day 2.

the control group over both comparisons on Day 1. On Day 2, the difference between groups does not quite attain significance for either comparison.

In contrast to these differences between groups, Ss in the control group made an average of 81.8 CRs on Day 1 of extinction, while the mean for Group E was only 59.0. This difference, although large, is not significant (t=1.655, 18d.f., .20>P>.10).[7] On Day 2, the means were 68.1 and 68.3 for Groups E and C, respectively.

Discussion. The results indicate that a novel B stimulus presented to experimental Ss preceding by 5 seconds some trials on the first day of extinction caused a significant magnitude increase on those trials and the following trials relative to the performance of non-shocked control Ss. There was also a tendency for this effect to continue on the second day of extinction. The failure to obtain significant differences on Day 2 of extinction may have been due to the decreased number of Ss from which data were available or to adaptation to the B on the part of the experimental Ss. Thus disinhibition was demonstrated during extinction of the rabbit's conditioned NMR.

Moreover, this magnitude increase was fairly specific to trials in close temporal proximity to the B, as the experimental group showed more rapid extinction than did the control group. This finding that the effect of the novel stimulus decayed over time is compatible with

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[7] This difference in means is due in part to the four Ss for whom Day 2's data were used. Due perhaps to the generalization decrement experienced on Day 1, the mean CRs for these four experimental Ss was 25.5, while for the control group it was 63.25.

those of Pavlov, who reported that "In all the experiments which have just been described the restoration of the extinguished reflexes lasts only for a few minutes depending on the duration of the extra stimulus and its after-effect." (Pavlov, 1927, p. 65)

EXPERIMENT 2

On the basis of the data collected in Experiment 1, it is concluded the disinhibition was demonstrated in extinction of the rabbits' NMR. The effect was produced by the presentation of a novel back shock stimulus, which resulted in a magnitude increase relative to non-shocked control animals. Furthermore, the increase was fairly specific to the trial following the B presentation and the succeeding trial; there was not an overall elevation of responding in the experimental group.

Having demonstrated disinhibition of an extinguished CS, the next experiment was performed in an attempt to ascertain whether an extinguished CS could produce an inhibitory summation effect. Since the previously cited studies that demonstrated extinction does not seem to produce an inhibitory CS were all conducted with rats in an avoidance or Conditioned Emotional Response (CER) paradigm, it was felt that a replication of this effect in another preparation would be profitable.

Additionally, an attempt was made to gather more data regarding the disinhibitory effect of the B stimulus. The design entailed conditioning the stimulus to be used as the excitatory stimulus (L) in the summation test, conditioning and extinguishing another CS (T), and then combining the two in summation testing. One group of Ss (Group E) received B prior to some LT trials during summation testing, while the control group (Group C) received no B presentations.

Procedure. Sixteen Ss were sutured and habituated to the apparatus on Day 1. Subjects then received 2 sessions of conditioning to L (Stage 1). Additional trials in 2 sessions ranging from 100 to 175 trials were run on Day 2 for Ss who were slow to condition. Stage 2 consisted of 3 days of acquisition to T. Extinction to T was then run for 2 days (Stage 3). Ten "reminder" reinforced L trials were given to all Ss at the end of the second day's session to ensure L still elicited a high response rate prior to summation testing. At the end of Stage 3-Day 2 the data from two Ss were discarded; one animal died and the other had failed to give more than one CR to L in Stage 1. The remaining 14 Ss were divided into two equal groups, matched on the basis of percentage of CRs made on Stage 3-Day 2, to ensure an equal amount of extinction occurred in each group. Stage 4 then consisted of 3 days of summation testing. Fifty L and 50 LT trials were presented in an unsystematic order each day; for Group E, 24 of the 50 LT trials were preceded by B. The Variac was turned off when Group C was run, so they received no back shocks.

Results. Most Ss gave CRs at least 50% of the time to L on Stage 1-Day 2. Five Ss were given additional training, to ensure responding occurred during summation testing, until they attained a response level of at least 25% CRs for the session. In Stage 2 all Ss were responding to T at at least the 80% level by Day 3. On Stage 3-Day 2, Group E animals gave a total of 147 CRs ($\bar{x} = 21.0$), while Ss in Group C gave a total of 145 CRs ($\bar{x} = 20.8$) to the tone.

The results of Stages 2 and 3 indicate acquisition and extinction to T occurred. In fact, extinction to T was virtually complete by the end of Stage 3-Day 2, as evidenced by the fact that during the last 20 trials of that session, the Ss gave an average of 3.5 CRs. All rabbits responded to at least 50% ($\bar{x}$ = 88%) of the L trials given at the end of Stage 3-Day 2, just prior to summation testing.

An analysis of variance with one between-subject variable (Groups) and two within-subject variables (Days and Trial-type) was performed on the % CR data from Stage 4 (summation testing). The results are summarized in Table 2. It can be seen that both the effects of Days and Trial-type are significant, while the Group E versus Group C effect approached significance. The F values for all interactions were less than 1. Examination of the group means contained in Table 3 shows that the Days effect is merely an extinction effect. The Trial-type effect is due to the fact that both groups responded more often on LT trials than on L trials, indicating an excitatory summation effect. Also, there was a strong tendency for Group E to respond more often than Group C, although this trend was not significant.

Finally, an Up/Down difference score, like that used in Experiment 1, was calculated for each animal. In this case, however, the magnitude comparison was between the LT trial preceded by B and the most recent LT trial.[8] The means for Groups E and C on Day 1 were

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[8] On two occasions the most recent LT trial was preceded by B and these trials were not used in this analysis.

Table 2: Summary of an analysis of variance for stage 4 (summation test) data. G = Group (E or C), D = Days and T = Trial-type (L or LT).

Source of Variance	df	Sum Squares	Mean Squares	F
Total	83	66,527.24	--	--
Between S	13	32,375.24	--	--
G	1	7,131.86	7,131.86	3.39*
S/G	12	25,243.38	2,103.62	--
Within S	70	34,152.00	--	--
D	2	7,182.31	3,591.16	4.45**
GD	2	14.36	7.18	F<1
SD/A	24	18,810.33	783.76	--
T	1	2,952.43	2,952.43	16.28***
GT	1	0.53	0.53	F<1
ST/G	12	2,176.05	181.34	--
DT	2	95.21	46.61	F<1
GDT	2	23.68	11.84	F<1
S/GDT	24	2,897.10	120.13	--

* .05<P<.10

** P<.025

*** P<.005

Table 3: Mean percentage CRs for each trial type over three days of summation testing. L = light and LT = light-tone compound.

	Day					
	1		2		3	
	L	LT	L	LT	L	LT
Group E	57	66	33	48	39	51
Group C	40	48	16	29	18	33

-2.57 and -3.57. This difference, while far from significant (t=.508), implies that B presentations had a slight disinhibitory effect, reducing the generally downward trend in response magnitude in Group E. The difference scores for Days 2 and 3 were virtually identical for the two groups.

Discussion. The major finding of this experiment was that the extinguished tonal CS was found to control an excitatory, rather than inhibitory, response tendency. This occurred despite the fact that the Ss were responding to the tone only 20% of the time during the last session of extinction. This result is compatible with previous findings (e.g., Weisman and Litner, 1969). Another interesting result was the strong tendency for Group E to respond more often during extinction than did Group C. This is the opposite of the tendency found in Experiment 1 and may merely represent random fluctuation. Alternatively, the ceiling effect on the part of the control group in Experiment 1 (81% CRs on Day 1 of extinction) was not present in Experiment 2 and this may have contributed to the difference between experiments.[9]

Finally, the response magnitude comparison between Groups E and C, although far from significant, is at least in the same direction as in Experiment 1. This is encouraging since the measure used in

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[9] Also, matching of E and C Ss in Experiment 2 was done on the basis of performance in extinction, rather than in acquisition, as was done in Experiment 1. Presumably, equating groups on the basis of prior extinction, performance should be less sensitive to random fluctuation.

Experiment 2 was not identical to that used in Experiment 1. That is, in Experiment 1, it was found that a B presentation affected not only the next trial, but also the trial after that. This latter trial was often the "N-1" trial for the magnitude comparisons made in Experiment 2, which would be expected to produce a strong bias against finding CR magnitude increases in Group E.

EXPERIMENT 3

It has been demonstrated that disinhibition can occur during extinction of the rabbit's NMR (Experiment 1), but the extinction of the NMR does not seem to produce an inhibitory stimulus (Experiment 2). These facts, as well as the fact that no demonstration of disinhibition since Pavlov has involved the disinhibition of a truly inhibitory stimulus, raises the possibility that so-called disinhibition is not actually "inhibition of inhibition." Instead, it seems more parsimonious to assume another process is occurring.

Specifically, recent evidence has shown that response deficits which occur during extinction are due partly to a decrease in excitatory strength (Reberg, 1972; Experiment 2 above) and in part due to a lack of attention to the stimulus (Cousins, 1972). If attentional deficits are occurring, the presentation of a novel stimulus in extinction may serve merely to cause an attention-shift. Such a shift might result in a brief increase in or reinstatement of responding, as well as a temporary reinstatement of attention.

Such a formulation is advantageous in that it does not necessitate assuming that an extinguished stimulus--which has been shown to not be inhibitory--is inhibitory. Additionally, if it is assumed that attentional phenomena may be less robust and more labile than associational phenomena, an attentional account of disinhibition is not only compatible with the existing literature but also might account for some of the anomalies within that literature.[10] That is, failures to obtain disinhibition are not uncommon even in carefully controlled studies (e.g. Gormezano, 1958) and in replications of successful demonstrations (e.g. Boakes and Halliday, 1975; Wolach and Ferraro, 1969). If non-associative attentional processes are rather labile,[11] it might be anticipated that parametric details may be crucial for the generation of the phenomenon, and, hence, that failure might not be uncommon.

Such a theoretical account would, however, not predict that disinhibition of a known inhibitory stimulus should occur. Since no such demonstration currently exists in the literature, Experiment 3 constituted a first attempt to disinhibit such a stimulus—the one used by Marchant and Moore (1974) in their demonstration of conditioned inhibition. It was assumed that the parameters which resulted in disinhibition in Experiment 1 would maximize the probability of obtaining the phenomenon in another paradigm, and, hence, lend some credence to any failures to obtain disinhibition.

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[10] An attentional account of disinhibition additionally seems preferable to that offered by Denny, 1971. Denny accounts for disinhibition in the context of Elicitation theory by positing that any event which makes the extinction situation more like the conditions under which acquisition occurs should make it more likely the CS will elicit the CR. It is not clear, however, how presentation of a novel B in these experiments would make conditions more like those of acquisition. Although B is an electrical stimulus, it is in an entirely different location and of greatly different intensity than the eyeshock UCS. (Subjectively, the B stimulus produces a slight tingling, if anything.)

[11] Tentative evidence for the inconsistent nature of disinhibition in extinction may be found in Experiment 1. Comparison of the data from individual Ss reveals that some Ss show robust disinhibition, while others fail to show the effect at all.

The following experiment, then, consisted of training Ss to L reinforced contrasted with an LT compound which was not reinforced. Following eight days of such training, which previous work (Marchant and Moore, 1974) indicated should be enough to produce an inhibitory T stimulus, the novel B was introduced during CI training. Twenty-four B presentations were delivered 5 seconds prior to some of the LT presentations during each of the next 4 sessions to the experimental group, while the control group received no B presentations. The conditions for the two groups were then reversed for the next two sessions, so that each group served as a control for the other. It was expected that, should disinhibition occur, it would be evidenced by a CR magnitude, or percentage CR, increment to those LT trials preceded by B relative to normal LT trials.

Procedure. Eight Ss were sutured and habituated to the apparatus for 15 minutes prior to the first session. Days 1-8 consisted of 50 reinforced L presentations unsystematically alternated with 50 nonreinforced LT presentations. At the end of Day 8, Ss were divided into two groups of four animals each, based on their performance that day. CI training continued for the next 6 days, but with one difference: B presentations preceded 24 of the LT trials for one group (Group B-0), but not the other group (Group 0-B). Conditions were then reversed for the two groups so that Group 0-B received B for the last two days, while Group B-0 did not.

Results. During Day 8 of CI training, the Ss in each of the two groups had a mean CS+/CS- difference score of 4%, which was a smaller mean difference than had occurred on previous days. For example, the mean difference on Day 5 was 13% (see Appendix B). These relatively low difference scores indicate the L, LT discrimination is not an easy one for rabbits to make. However, six out of the eight animals (three in each group) showed a greater mean CR magnitude to L than to LT (see Appendix B). As can be seen in Table 4, discrimination performance for both groups improved somewhat to approximately a 10% CS+/CS- difference during the next 6 days and was essentially the same for both groups. Similarly, Up/Down magnitude differences for each group may be seen in Table 5. Examination of this table reveals what seem to be essentially random variations. Thus, the effect of B presentations seemed to have no effect on CI performance.

Discussion. The major finding of this experiment was that the introduction of B as a novel stimulus during CI training produced no systematic effect on CI performance. Unlike the results obtained in Experiment 1, the B presentations seemed to have no systematic effect on individual S's CR magnitude. The magnitude comparison is, however, essentially quite conservative, as it was in Experiment 2. In Experiment 2, though, there was a disruption of performance--an overall increase in responding. An analogous disruption in this experiment would be increased responding to CS- on the part of the group being shocked relative to the non-shocked controls. Such an effect was not present in this experiment. Overall, then, despite the fact that B parameters were the same as those used in the first two experiments, no evidence of disinhibition was obtained in this experiment.

Table 4: Mean percentage CRs to CS+ (light) and CS- (light-tone) during conditioned inhibition training with mean difference (diff.) scores for each day.

		Day					
		9	10	11	12	13	14
Group B-0	CS+	100.0	100.0	100.0	100.0	99.5	98.0
	CS-	94.5	88.5	94.0	91.5	89.0	86.5
	Diff.	5.5	11.5	6.0	8.5	10.5	11.5
Group 0-B	CS+	97.5	94.0	98.0	94.5	95.5	97.5
	CS-	91.0	83.0	82.5	87.0	81.0	88.5
	Diff.	6.5	11.0	15.5	9.5	14.5	9.0

Note: Group B-0 received back shock on Days 9-12, Group 0-B on Days 13-14.

Table 5: Mean Up/Down over days of conditioned inhibition training.

	Day					
	9	10	11	12	13	14
Group B-0	1	3.25	.5	-2	4	-5
Group 0-B	-1	3.0	-.25	.75	-2.75	2.75

Note: Group B-0 received back shock on Days 9-12, Group 0-B on Days 13-14.

EXPERIMENT 4

Following the failure to obtain disinhibition in Experiment 3, the following experiment was performed, in part, to attempt to detect evidence of disinhibition during CI using a slightly different procedure. Since the L stimulus is excitatory and only the T stimulus becomes inhibitory in the PCI paradigm used here (see Marchant, Mis and Moore, 1972), it may possibly be more susceptible to disinhibition when presented alone, rather than when imbedded in the compound. Hence, B was presented prior to T alone presentations, as well as prior to LT presentations.

Additionally, evidence was sought that might allow for differentiation between Pavlov's (1927) and/or Hull's (1943) conceptualization of inhibition and that recently proposed by Rescorla and Wagner (1972). Essentially, the difference in theoretical positions is that disinhibition of inhibition is compatible with the first two theories, but not with the Rescorla-Wagner model. Thus, Pavlov's descriptive model states that excitation and inhibition exist simultaneously; hence, inhibition can be disrupted, leaving excitation. Hull's model allows for disinhibition in an analogous manner. Basically, excitation (reaction potential--${}_{S}E_{R}$) and conditioned inhibition (${}_{S}I_{R}$) summate with other variables in determining response strength (${}_{S}\bar{E}_{R}$--effective reaction potential). That is, ${}_{S}E_{R} - (I_{R} + {}_{S}I_{R}) = \bar{E}_{R}$, where I_{R} is reactive inhibition. Hence, a novel stimulus could disrupt ${}_{S}I_{R}$, through afferent interaction (essentially generalization decrement in this case), and produce disinhibition. (For Hull's derivation, see Appendix D.)

In contrast to Hull's system, in the Rescorla-Wagner model response strength (V--associative strength) is a function of the associative strength of all stimuli present on a trial. Thus, in these experiments, while the V value for T might be positive (excitatory) or negative (inhibitory) or zero (neutral), there is currently no provision in the model for a sudden change in V due to the presentation of B. The B stimulus should have no effect on V, as the value of V for a single stimulus is unitary rather than a combination of excitation and inhibition.

Given these considerations, a successful demonstration of the disinhibition of an inhibitory stimulus would be very damaging to the Rescorla-Wagner model, while lending credence to the formulations of Pavlov and Hull. Hence, to attempt to ensure both excitatory and inhibitory strength could be present for the T stimulus, as is postulated by Pavlov and Hull, Ss received excitatory conditioning to the tone prior to CI training.

Procedure. Eight rabbits were sutured and habituated to the apparatus prior to the first day of acquisition. Ss then received 3 days of conditioning to reinforced L and T stimuli; 50 trials per day of each. CI training was then given for 12 days. The seven Ss (one was sacrificed to the God of Futility on Day 10 of CI training) were divided into an experimental group (Group E) and a control group (Group C), matched on the basis of their performance on Day 12.

Testing of Group E began the next day with 40 "warmup" (regular CI) trials. During the next 60 trials, 5 of the LT trials were preceded by B (BLT trials). Additionally, 10 T trials were presented, 5 of which were preceded by B (BT), while 5 acted as "control" trials against which BT trials could be compared for possible CR magnitude increments. Group C was treated identically, but without B. This testing was continued for 2 additional days and then was given without the warmup for one day. Both groups were then given 3 days of normal CI training, followed by 3 more days of testing.[12]

<u>Results</u>. Mean percentage CRs were 42% and 75% to L and T respectively on the last day prior to CI training. These percentages were similar to those found by Marchant, Mis and Moore (1972). By Day 12 of CI, the CS+/CS- difference was 28% for Group C and 27.5% for Group E.

The mean percentage CRs during testing may be seen in Table 6. The data of primary interest are contained in the last two columns, which show the performance of Group E to T alone compared with BT trials. It can be seen that in all test situations response rate was higher when T was not preceded by B. The opposite generally was true for Group C. These results are the opposite of what would be expected should disinhibition have occurred. It should also be noted that, as can be seen in the first four columns, Group E's CI performance was somewhat disrupted after testing began. Examination of the next two

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[12] In an attempt to gather more data on the effect of B on the LT compound, T alone presentations were omitted on the first day of the last 3 test days.

Table 6: The probability of the occurrence of a CR to CS+ (light), CS- (light-tone), or tone alone over three days of testing, one day without "warmup" and three more days of testing. B indicates back shock preceded the trial type, B+1 indicates the trial following a back shock-preceded trial, and Warmup indicates the first 40 conditioned inhibition trials each day, while "last 60" indicates the last 60 trials, during which testing with back shock during conditioned inhibition training.

	Trial Type								
	Warmup		Last 60		CS- during last 60		Tone		
	CS+	CS-	CS+	CS-	B and B+1	Not B or B+1	B	B	
Group E	.97	.59	.98	.66	.72	.60	.21	.26	First 3 days
Group C	.89	.63	.95	.64	.62	.66	.18	.09	
Group E			.96	.63	.64	.61	.25	.30	Day 4 (without warmup)
Group C			.98	.91	.97	.93	.05	0	
Group E	.96	.53	.94	.57	.61	.55	.16	.09	Last 3 days
Group C	.98	.49	.97	.48	.42	.56	0	0	

Note: Three days of conditioned inhibition training occurred between Day 4 and the last three days of testing.

columns shows this disruption, i.e., increase in CR probability to LT, seems to be due to the effect of B. That is, Ss in Group E show an elevation in response rate on BLT trials, or on the following trial, relative to trials presumably not influenced by B. This effect was consistent throughout testing and was the opposite of the performance of Group C. Finally, although too little data were available for statistical comparison, an Up/Down difference measure showed that the CR magnitude of Ss in Group E tended to go up on trials preceded by B relative to Group C Ss.

Discussion. There were two results of interest in this experiment. The first was a small but consistent increase in CR probability and magnitude on BLT trials in Group E relative to Group C. Such data suggest a possible disinhibiting effect of B. This effect may have been obtained in this experiment and not Experiment 3 because of a "ceiling" effect in Experiment 3. That is, mean CS+/CS- difference prior to testing was only 4% in Experiment 3, whereas it was 28% in this experiment, due to the fact that Ss received 12 rather than 8 days of CI training.

Of greater interest, however, was the finding that, despite the effect of B on the compound, the shock had the opposite effect when presented prior to T alone. Since tone, rather than the LT compound, is the inhibitory stimulus, it would seem these data offer no evidence of disinhibition of an inhibitory stimulus. Instead, it seems more parsimonious to regard the effect of B as, once again, attentional in nature. If one posits that the B causes an attentional shift from T, to which S must attend if he is to discriminate, to L--an excitatory stimulus; then an increase in CRs to LT should occur. Thus, there still remains no evidence that disinhibition of a truly inhibitory stimulus can occur, whether the tone was strongly inhibitory or was in the process of becoming inhibitory (i.e., was still being responded to).

EXPERIMENT 5

Although no evidence of disinhibition of an inhibitory stimulus was obtained in Experiment 4, it is possible this was due to T being "too" inhibitory. That is, after 12 days of CI training, very little responding occurred when the tone was presented alone. Those CRs that did occur were made almost entirely by a few Ss. Hence, it is possible that no disinhibition of T was evidenced simply because the inhibitory process was so solidified for most Ss that it could not be disrupted.

The original design of this experiment, then, was to give half the amount of CI training (6 days) that had been given in Experiment 4 and then test for disinhibition. After 6 days, however, similar to the findings of Marchant, Mis and Moore (1972), only one S was discriminating at all, so CI training was continued until some evidence of discrimination was obtained.

Procedure. Eight rabbits were sutured and habituated to the apparatus, and then given 3 days of acquisition to L and T. Three Ss who were still not responding to T on Day 3 were given additional training until they consistently responded to it. CI training then occurred for the next 8-15 sessions, depending upon the rabbits' performance. The testing procedure was the same as in Experiment 4. Two Ss did not show any sign of discrimination after 15 days and were dropped from the study. Additionally, two Ss were dropped because they screamed when given B.[13] Hence, data from only four Ss are presented.

Results. Since data are presented from only four Ss, and since they received differing amounts of CI training, their performance is presented individually. Subject #13 received 8 days of CI, the last day of which he gave 90% CRs to L and 60% CRs to LT. Over 3 days of testing, no CRs were given to T, whether preceded by B or presented alone. Similarly, after 11 days of CI training, S #15 gave 96% CRs to L and 78% CRs to LT and gave no CRs to T over 3 days of testing.

Subject #12 failed to show discrimination in terms of percent CRs to L and LT, but did show a magnitude discrimination. That is, on Day 11 of CI training, the mean magnitude of the CR to L was 14.6 mm, while to LT it was 13.4 mm. Despite the fact that CR magnitude tended to increase on BLT trials relative to other LT trials, S #12 gave six CRs (out of a possible 15) to T and only five to BT over 3 days of testing. The magnitude of all these CRs was virtually identical. This result is the opposite of a disinhibitory effect.

Finally, S #16 made 96% CRs to L and 90% CRs to LT on Day 11 of CI training. Through all 3 days of testing, this S showed a strong tendency to increase response magnitude on both BLT and BT trials relative to non-shock trials. Mean magnitude differences between

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[13] The screaming elicited by the B was: 1) a surprise, as it never happened before, and 2) puzzling, since rabbits that scream also struggle to escape. These rabbits did not. Nervous rabbits sometimes scream, and I believe these bunnies were "uptight," rather than in pain.

BT and T trials over 3 days of testing were 1.6 mm, 5.8 mm, and 7.2 mm. Two additional days of testing were given, including five Bs prior to L (BL) trials each day to test for the possible occurrence of external inhibition. Over all 5 days of testing, the probability of a CR to T alone was .48, while the probability of a CR to a BT trial was .72. Finally, CR magnitude to BL trials relative to the prior L trial increased 6 times, stayed the same 3 times, and decreased once. Thus, there was a tendency for S #16 to show a CR magnitude increase no matter what stimulus the B presentations preceded.

Discussion. The results of three Ss in this experiment indicated that as soon as differentiation between L and LT appeared in the form of CR percentage difference, response rate to T was zero. Furthermore, although S #12 showed only a 1 mm difference in mean CR magnitude between CS+ and CS- at the end of CI training difference, he responded to T only 37% of the time during testing. It would seem, then, that by the time a magnitude difference is detected between L and LT, response strength to T is rapidly approaching zero.

Finally, S #16 showed strong evidence of disinhibition—an increase in the CR probability and magnitude whenever B is presented prior to T or LT. However, since the same effect also occurred when B was presented prior to L, it would seem disinhibition, at least as Pavlov meant the term, was not involved. Had true disinhibition occurred, one would expect the opposite—external inhibition—to occur when the B was presented prior to the excitatory L stimulus. Instead, the opposite happened; there was an increase rather than a decrease in response magnitude. Therefore, it would seem more parsimonious to assume that the increases in CR magnitude on trials following B presentations exhibited by S #16 were the result of general arousal, rather than disinhibition.

EXPERIMENT 6

Although B presentations failed to disrupt "deep" inhibition in Experiments 4 and 5, it still remained possible that inhibition could be disrupted early in training. The first group in Experiment 6, then, represented an attempt to find a point in CI training when response strength to T was below the excitatory level attained at the end of excitatory acquisition to the tone, but was still above zero.

Based on the results obtained from Group 1, a second group of animals were given CI training for 4 days and then tested with B. For both groups, B was presented only prior to T trials during testing for disinhibition.

Procedure. The eight Ss that constituted Group 1 were all pretrained to L and T for 3 days and then received at least 5 days of CI training. After this, testing for disinhibition began when a mean magnitude difference was detected between CS+ and CS- trials. The testing sequence for all Ss in this experiment was the same as that used in Experiments 4 and 5, except that B was no longer presented prior to LT trials. Five Ss from Group 1 received such testing. The other three Ss were only given T alone "probe" trials in an attempt to determine when in CI training response strength to T was at an intermediate level between the initial high excitatory value generated by preconditioning and zero.

Group 2 (n=8) in this experiment received the standard 3 days of conditioning to L and T, followed by 4 days of CI training. At the end of the fourth day of CI, the eight Ss were divided into two equal groups, matched on the basis of their performance on Days 3 and 4 of CI training. The experimental group then received 4 days of testing with five BT trials and five T alone trials each day. The control group received the same trial sequence, but without the B.

Results. All Ss were responding at a high level to T by the end of initial conditioning. The mean percentage of CRs to T was 95.5 in Group 1 and 92.5 in Group 2 on the third day of acquisition. Table 7 shows the percentage CR difference and mean magnitude difference on the last day of CI training, as well as the probability of a CR to T alone and to BT trials for Group 1. It may be seen that for three of the Ss, response rate to T alone was zero, despite the fact that the mean difference in CR magnitude to CS+ and CS- was quite small. The other two Ss also show very small mean magnitude differences on their last day of CI, but do respond to the tone when it is presented alone. The magnitude of their responses ranged from 1 to 3 mm. It is interesting to note that while CRs were often made to T alone, responses rarely occurred when T was preceded by B.

The performance of the three Ss given only T alone probe trials was similar to that which had been obtained previously. That is, all Ss showed a reduced amplitude to tone, despite showing very small difference in magnitude to CS+ and CS-. This indicates that response strength to the tone is virtually zero by the time even a small CR magnitude difference between CS+ and CS- is detected.

Of the second group of animals, the mean difference in percentage CRs to CS+ and CS- over the last 2 days of CI training was 1.5% for

Table 7: Mean percentage CR and CR magnitude differences for Group 1 on the last day of conditioned inhibition training. P(CR) = probability of a conditioned response, T = tone trial, BT = tone trial preceded by back shock.

S #	# of days of CI	% CR diff.	mean magnitude diff.	P(CR)/T	P(CR)/BT
17	6	6	.71 mm	.375	.07
18	6	2	.24 mm	0	0
19	5	0	2.17 mm	0	0
21	6	0	.78 mm	.625	.07
23	5	4	1.31 mm	0	0

Table 8: The probability of a CR to tone (T) or tone preceded by back shock (BT) over four days of testing during conditioned inhibition training by Group 2.

	Day							
	1		2		3		4	
	T	BT	T	BT	T	BT	T	BT
Group E	.30	.30	.75	.50	.60	.40	.55	.35
Group C	.73	.60	.30	.05	.50	.25	.45	.15

the control group and -3.25% for the experimental group. Thus, there was a tendency for poorer discrimination on the part of the experimental group, and this trend is reflected by the greater number of CRs made to T by this group during testing, as may be seen in Table 8. (Data for individual Ss may be seen in Appendix C.) It may also be noted in Table 8 that in both the experimental and control groups there is a stronger tendency to respond to T alone trials than to BT trials, and that this tendency is slightly smaller in the experimental group than the control group. Thus, despite the fact that the B was presented quite early in CI training, it still failed to produce a disinhibitory effect when preceding tone.

Discussion. The results from the Ss in Group 1 showed that when even a small magnitude difference occurs between CS+ and CS- trials in CI training, response strength to T is approaching, or is at, zero. Based on this finding, then, the second group was run for only 4 days prior to testing. Additionally, the performance of the two Ss in Group 1 that did respond to tone during testing represents the opposite of disinhibition. That is, response strength decreased, rather than increased, following presentation of the novel B stimulus.

Of the second group of Ss, both experimental and control Ss showed the same tendency to respond to T alone trials more often than when T was preceded by B, and the differences between response rates to these two trial types were similar for both groups over days. Thus, no evidence of disinhibition of the inhibitory T stimulus was obtained in this experiment. In fact, the results obtained showed a response tendency in the opposite direction; the novel stimulus presentation tended to reduce, rather than increase, the probability of a CR.

Additionally, the results of Experiments 5 and 6 provide some interesting support for the Rescorla-Wagner model. The model states that the associative strength of the LT compound should be essentially a summation of the associative strength of the two stimuli. Since associative strength only roughly translates to response strength (Rescorla and Wagner, 1972, p. 77), it would be expected that response strength to T should be around zero before a magnitude difference would be detected between responses to L and LT. That is, in fact, what was generally found.

GENERAL DISCUSSION

Disinhibition during extinction of the rabbit's conditioned nictitating membrane response was obtained in Experiment 1 using a novel back shock as the disinhibiting stimulus. In Experiment 2 it was found, however, than an extinguished CS produced an excitatory summation effect implying that an extinguished stimulus is not inhibitory. Thus, Experiments 3-6 represented an attempt to obtain disinhibition in a CI paradigm. Although there was some suggestion that CI performance could be disrupted by novel stimulus presentations, no evidence of disinhibition of the inhibitory tonal stimulus was obtained.

The results of Experiment 1 are similar to those of previous investigators (e.g., Razran, 1939) who have obtained disinhibition during extinction of a CS. Based on Experiment 2 above, as well as the work of other researchers (e.g., Reberg, 1972), it was concluded, however, that simple extinction of a CR does not result in the CS becoming a conditioned inhibitor. That is, an extinguished CS does not produce an inhibitory summation effect and hence, does not satisfy the current criteria for conditioned inhibition as delineated by Rescorla (1969).

The finding that disinhibition can occur in extinction and in other paradigms which have not been demonstrated to produce a conditioned inhibitor (e.g., Brimer, 1972), suggests that so-called disinhibition may not involve "inhibition of inhibition." Instead, it is suggested that response decrements in extinction are, in part, mediated by attentional factors; that is, the animal begins to cease attending to the CS and, presumably, attends to other stimuli. The sudden presentation of a novel stimulus then briefly causes a shift of attention, resulting in a short-lived reinstatement or magnitude increment of the CR. Such a loosely formulated account seems compatible with all currently existing literature.

Also, it should be noted that this attentional account of the effects of a novel stimulus presentation is somewhat similar to the accounts of disinhibition given by both Pavlov (1927) and Hilgard and Marquis (1940). That is, these authors used the term "distraction" when discussing novel stimulus presentations, but assumed that associative, rather than attentional, processes were being disrupted. Finally, Sokolov (1963) has suggested that a novel stimulus elicits an OR which interferes with the dominant inhibitory process. This model, however, also assumes that extinction produces conditioned inhibition, an assumption which the data suggest is incorrect.

The major empirical finding in this series of experiments, however, was the failure to obtain disinhibition of a conditioned inhibitor utilizing the same novel stimulus and parameters that produced disinhibition in extinction.[14] Since there are no demonstrations of

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[14] It should be noted that it is logically possible that the 5 sec interval by which B preceded the CS may not have been appropriate during CI training. That is, simple extinction is a much more simple task than is CI training and it is possible that the same parameters which are maximally effective in extinction may not be optimal during a more complex task. For example, Hartman and Grant (1962) have shown that the optimal CS-UCS interval is greater for differential conditioning than it is for simple conditioning. By analogy, it is logically possible that the novel stimulus-CS interval should have been longer in CI training than it was during extinction.

disinhibition of a conditioned inhibitor currently available in the literature, it is suggested that disinhibiting a conditioned inhibitor may not be possible.

Should such be the case, the failure to obtain disinhibition of a conditioned inhibitor is of theoretical, as well as empirical, interest. That is, the Rescorla-Wagner model (Rescorla and Wagner, 1972) specifically could not account for the disinhibition of a conditioned inhibitor. That is, the response strength (V for associative strength in their model) for a single stimulus can be positive (excitatory), negative (inhibitory), or zero (neutral), but can not simultaneously have excitatory and inhibitory components. Alternatively, the model of inhibition proposed by Pavlov (1927) stipulated that excitation and inhibition are present simultaneously; therefore a novel stimulus presentation can disrupt the inhibitory process, laying bare the excitatory process and producing a reinstatement of responding. Similarly, Hull (1943) states that response strength (${}_{S}E_{R}$--effective reaction potential) is a summation, among other variables, of both excitation (${}_{S}H_{R}$--habit strength) and inhibition (${}_{S}I_{R}$). Disinhibition, then, represents a disruption of ${}_{S}I_{R}$. (For the actual derivation, see Appendix D.)

Hence, while both the Pavlovian and Hullian models of inhibition can account for disinhibition of a conditioned inhibitor, the Rescorla-Wagner model cannot. The suggestion then, that disinhibition of a conditioned inhibitor may not be possible lends strong heuristic value to the Rescorla-Wagner model. Finally, additional support for the

Rescorla-Wagner model was obtained when it was found that the response strength to tone was zero before a magnitude difference between CS+ and CS- occurred during CI training, as would be predicted by the Rescorla-Wagner model, because $V_{LT} = V_L + V_T$.

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Singh, D. & Wickens, D.D. Disinhibition in instrumental conditioning. Journal of Comparative and Physiological Psychology, 1968, 66, 557-559.

Skinner, B.F. A failure to obtain "disinhibition." Journal of Genetic Psychology, 1936, 14, 127-135.

Sokolov, Ye.N. Perception and the Conditioned Reflex. New York: The Macmillan Co., 1963.

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APPENDIX A: Literature Review of Disinhibition

1. Pavlov's work

Pavlov did not limit himself solely to the previously described disinhibition of an extinctive inhibitor. In this section I will describe briefly other paradigms in which he investigated disinhibition as well as some of the parametric observations he made.

In addition to its occurrence during extinction Pavlov reports disinhibition during training similar to the type used by Marchant and Moore (1974). For example, in an experiment conducted by Nikolaev (Pavlov, 1927, pp. 82-83) a rotating object was the CS+ and the compound of the rotating object and a tone was the CS-. The compound[1] acquired inhibitory properties; but, whenever the compound was presented simultaneously with a novel stimulus—a metronome, thermal or tactile stimulus—the amount of salivation was far greater than when the compound was presented by itself. This increase in response amplitude constitutes, of course, disinhibition.

However, Pavlov also reports that if the disinhibiting stimulus is a strong one, disinhibition will not be evidenced unless the disinhibitor is presented a substantial period of time prior to the inhibitory stimulus, thus giving the strong after-effects of the disinhibitor a chance to dissipate. For example, Nikolaev, using the same conditioned stimuli and a different dog (Pavlov, p. 88),

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[1] The tone was not tested by itself, but was presumably inhibitory.

Weisman, R.G. & Litner, J.S. The course of Pavlovian excitation and inhibition of fear in rats. Journal of Comparative and Physiological Psychology, 1969, 72, 667-672.

Wenger, M.A. External inhibition and disinhibition produced by duplicate stimuli. American Journal of Psychology, 1936, 48, 446-456.

Winnick, W.A. & Hunt, J.McV. The effect of an extra stimulus upon strength of response during acquisition and extinction. Journal of Experimental Psychology, 1951, 41, 205-215.

Wolach, A.H. & Ferraro, D.P. A failure to obtain disinhibition in fixed-interval operant conditioning. Psychonomic Science, 1969, 15, 47-48.

Yamagachi, H.I. & Ladioray, G.I. Disinhibition as a function of extinction trials and stimulus intensity. Journal of Comparative and Physiological Psychology, 1962, 55, 572-577.

found that 10 cc of a 5% solution of sodium carbonate introduced into the dog's mouth served as a disinhibitor of the inhibitory compound, despite the fact that it preceded the compound presentation by 5 min.

The preceding of an inhibitor by the disinhibitor, as opposed to presenting them simultaneously, was not limited to this paradigm. Pavlov describes work on disinhibition in extinction (Pavlov, p. 65) in which a stimulus which had been absent for a minute nonetheless produced disinhibition. In the same experiment Zavadsky found that acid introduced into the dog's mouth produced disinhibition if the inhibitor were presented 40 sec after the salivary flow elicited by the disinhibitor had ceased, but did not produce disinhibition if the time lag was 7 min and 10 sec. While Pavlov does not report systematic parametric data on the time lag between the offset of the disinhibitor and the onset of the inhibitor, from these and other data it is clear that Pavlov felt that, to be effective, the disinhibitor must overlap the inhibitor or precede it by not too great a time, perhaps not more than one-half the inter-trial interval.

Another inhibition-producing paradigm Pavlov found susceptible to disinhibition was inhibition of delay. For example, with an unusually long duration CS+ (for Pavlov's preparation 1-2 min instead of 1-5 sec) the first half of the CS not only did not elicit a CR, but furthermore, was said to be inhibitory, while the second half was excitatory. In fact, Pavlov (Pavlov, p. 103) regarded inhibition of delay as an analogous paradigm to PCI, with the CS and "early in the time interval" acting as the compound CS-. Therefore, it was not surprising to Pavlov that a "delay" inhibitor could, like a PCI, be disinhibited. For example, Zavadsky (Pavlov, p. 93) conditioned a 3 min tactile CS with an acid UCS. Following sufficient training, the dog salivated heavily during the second half of the CS, but little--if at all—during the first 90 sec. A metronome was then paired with the tactile stimulus and the dog suddenly began salivating during the first 90 sec of the compound presentation. This type of result was replicated a number of times with various novel disinhibitors and was usually accompanied by external inhibition during the second half of the compound presentation. That is, instead of the normal salivary pattern of no secretion during the first half of the CS and a progressive increase during the last three 30-sec intervals, it was found that approximately the same amount of salivation was secreted during all six 30-sec intervals. Thus, a novel stimulus presented simultaneously with a CS could produce either sudden responding or a lack of responding depending on what stage of training the animal was in, or, even more simply, how long the CS had been on.

This paradoxical reciprocity between external inhibition and disinhibition, however, was not complete. Pavlov reports (Pavlov, p. 96) that, although a large number of (perhaps, any) stimuli could serve as disinhibitors or external inhibitors, not all stimuli could serve as both. Basically, Pavlov generally found that a weak stimulus could function as a disinhibitor, but not an external inhibitor, whereas for a strongly salient stimulus the situation was reversed. Stimuli of medium strength could function as both. The salience of the stimuli

Pavlov used, unfortunately, is hard to equate with stimuli used by modern investigators. For example, stimuli of medium strength included the rotating object, metronome and tactile stimuli. This point is perhaps germane to the fact that modern investigators have not always found the above relationship in their parametric investigations of the effects of stimulus intensity on disinhibition.

Finally, although no parametric data are presented, Pavlov (Pavlov, p. 65) reports that "the restoration of the extinguished reflexes lasts only for a few minutes." Thus, the effects of a novel stimulus should be relatively short-lived, unless the stimulus is presented throughout the entire experimental session, in which case it should have an effect throughout the session. Also, Pavlov reported that—at least in an experienced dog—the novel stimulus soon lost its effectiveness as a disinhibitor.

Summarizing this section, Pavlov reported demonstrations of disinhibition with three forms of internal inhibition: extinction, PCI, and inhibition of delay. Additionally, his data imply that a stimulus of medium strength presented just prior to or overlapping the CS presentation should be optimal for producing disinhibition. Finally, he reports that disinhibition is a relatively short-term process and that it may well have a transitory nature.

2. Work since Pavlov

a. Extinction

Since by far the bulk of experiments investigating disinhibition performed since Pavlov have utilized extinction as the paradigm used to generate inhibition, these studies will be reviewed first. Furthermore, it is important to note at this time that recent evidence from a number of investigators (Cousins, 1972; Donahoe, 1974; Henderson, 1973;[2] Reberg, 1972; Weisman and Litner, 1969) indicates that extinction does not produce a truly inhibitory stimulus, but rather a return to "zero" response strength—neither excitatory, nor inhibitory. It is possible, then, that "disinhibition" produced in extinction is not the same as disinhibition of a true conditioned inhibitor.

Chronologically, the first disinhibition study performed in this country was done by Switzer (1933). Utilizing the GSR, she conditioned 15 Ss using a light CS and a shock UCS. Following conditioning and extinction to zero responding, she presented a 300 msec "loud raucous buzzer" 38.5 sec prior to the next light presentation.[3] Disinhibition occurred, as the response to the light was reinstated. Continued testing with more extinction trials produced further disinhibition, but the magnitude of the effect decreased over a few trials. A control group (n = 15) received first UCS trials, followed by CS trials and disinhibition testing. Not only did they give a GSR to the light, they also showed generally as large a response to the buzzer-preceded light trial as they had to the first light trial. Thus, the GSR elicited by the light was probably not a sensitized response, nor a conditioned one,

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[2] Henderson utilized a CS which had suppressive effects of its own in the CER. Hence, he found that extinction merely produced a return to the initially suppressed baseline, rather than absolute neutrality.

[3] Incidentally, the novel buzzer generally elicited a larger GSR than had the conditioned light stimulus on the first extinction trial.

but rather an orienting response (Sokolov, 1963). The buzzer presentation seems to have functioned merely to reinstate the orienting response to the light, and probably served the same function in the experimental group. Therefore, although suggestive, this study does not provide very solid evidence for the existence of disinhibition in extinction.[4]

Hunter (1935) attempted to produce disinhibition with rats. The onset of two hundred w bulbs signalled a 2 sec interval followed by an electrical grid shock. A response of a movement of one body length from the grid placed in the middle of an alleyway enabled the rat to avoid the shock. The CS was presented once a minute until the animals had acquired the response, at which time extinction began and was continued until the response was not given on 10 successive trials. At this point a buzzer was sounded, followed 1 sec later by the resumption of light trials with the 1 min ITI. Of the four rats involved, one gave three responses on consecutive trials following the buzzer. Another did not respond on the next 3 trials, but then gave six consecutive responses; the third did not respond for 2 trials then gave two CRs, while the last did not respond at all. Hunter also reports that with three rats in a counterbalanced conditition similar results were obtained, and Hunter felt that disinhibition had been demonstrated.

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[4] Studies similar to this and, therefore, open to the same criticism, were performed by Wenger (1936) and Hovland (1937).

However, the performance of the four Ss whose data are provided does not compel such a conclusion. One of the Ss showed no effect and two showed the effect with none of the immediacy one would expect from Pavlov's data. Furthermore, Hunter's conclusion must be tempered by the fact that two of the rats for whom light was the CS, and one with the buzzer CS, initially conditioned in 10 min. Since Hunter used a 1 min ITI and conditioned to a criterion of 10 CRs, presumably these Ss never made an "error." This implies a very strong unconditioned effect of both CSs, and casts serious doubt on the conclusion that disinhibition actually took place.

A more convincing demonstration of disinhibition was presented by Razran (1939). Twenty-four human Ss were presented with brief flashes of a 15 w red light bulb during periods of eating. Such a procedure produces conditioned salivation; following such conditioning, each S was given 24 extinction trials with a 1 min ITI. Preceding any two of the even numbered trials, a buzzer was sounded. This procedure gave Razran two data points on each even numbered trial against which the other 20 "control" Ss could be compared. During the first 10 trials of the extinction session, the buzzer reduced the magnitude of the CR, but thereafter increased the size of the response. In fact, Razran reports a correlation of .91 (± .02) between the number of extinction trials and the magnitude of the incremental change. This correlation implies that disinhibition is greater after more extinction has taken place, at least up to 24 trials. This study, then, seems to demonstrate not only disinhibition, but also external inhibition of the CR in the early stages of extinction. This result is reminiscent of Pavlov's finding that a novel stimulus presented with a long delay CS "flattens" the response magnitude curve.

A demonstration of disinhibition in an operant preparation, as opposed to Razran's classical conditioning preparation, was presented by Horns and Heron (1940). Twenty-four albino rats were given 10 one-hour sessions of bar pressing for food on a fixed interval 4 (FI 4') min schedule. This was followed by 5 extinction sessions and 5 disinhibition testing sessions. On the first day of testing, 30 sec buzzer presentations were given 20 and 40 min into the session. Both presentations were followed by responding, but the overall mean number of responses for the session was lower than it had been the previous day. The stimuli used on the second testing day consisted of first 90 sec of grid shock and later 90 more sec of grid shock followed by 60 shocks of less than a second's duration a second apart. Each produced a large increase in bar press rate and the overall mean number of presses for the session was more than twice the mean number of presses in the final extinction session. Similar results were obtained from shock presentations on testing days 3 and 4, but the tone presentations on days 3, 4, and 5, as well as a light stimulus on day 5, did not produce increments of the magnitude the shock had.[5] Thus, disinhibition was clearly

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[5] It should also be noted that the mean level of responding for all the testing sessions was always under one response per minute. This point will become important in the later consideration of Brimer's work.

demonstrated in an operant situation, with what was probably the strongest (most salient) stimulus being the best disinhibitor.

Another demonstration of disinhibition in extinction in an instrumental situation was presented by Gagné (1941). Following habituation in the 3 foot alleyway, albino rats were given 15 acquisition trials and then extinction trials until they did not run the alley in a period of 3 min. A 75 sec ITI was used throughout training. A control group (G_1; $n = 12$) received no stimuli. The other four groups are represented by the following 2 X 2 table:

		Acquisition & Extinction trial on which novel stimulus was presented	
		1 & 5	4 & 5
Disinhibitor	Buzzer	G_2, n = 8	G_3, n = 10
	Scratch	G_4, n = 12	G_5, n = 10

Thus, in G_2 and G_3, *S*s received a 4 sec buzzer presentation 2 sec prior to the opening of the start box door on acquisition trial 1 or 4, as well as a buzzer presentation prior to extinction trial 5. The procedure for the other two groups was the same, except the novel stimulus was a light scratch on the back of the start box which persisted until the rat faced the back of the box. Two seconds later, the start box door opened, and Gagne reports that most of the rats responded almost immediately by leaving the start box.

The results showed that neither stimulus had an effect on trial 1 of acquisition, but both the buzzer (t-test, $p<.01$) and the scratch ($p<.05$) significantly retarded the latency to go 4 in. on trial 4.

In extinction, however, the scratch groups had significantly ($p<.01$) shorter latencies on trial 5 than did the control group. The buzzer group, on the other hand, showed significantly longer latencies ($p<.01$) than the control group on trial 6. Both effects dissipated within a couple of trials. The retardation on extinction trial 5 in the buzzer groups may have been due to the fact that the rats froze when the buzzer came on. This species-specific-defense-response (Blanchard and Blanchard, 1971; Bolles, 1970) would be expected to reduce latency to run, but why it did not do so on trial 1 of acquisition is not clear.[6] Also, the latency to run was greatly reduced on trial 6. This seems analogous to Pavlov's report (Pavlov, p. 96) that the aftereffects of a strong stimulus must dissipate before disinhibition can be seen. Gagné then demonstrated both external inhibition and disinhibition using the same stimuli. Furthermore, there is some support for Pavlov's contention that the two processes are not completely reciprocal. That is, the scratch produced less external inhibition than did the buzzer, but better disinhibition.

Another demonstration of disinhibition in extinction was conducted by Yamaguchi and Ladioray (1962). In their first experiment using albino rats, they attempted to manipulate the amount of extinction by comparing groups (n = 12) which had received either 10 or 20 extinction

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[6] It might be argued that on trial 1, the buzz followed by the opening of the door allowed the rat to make a more dominant SSDR—escape (Blanchard & Blanchard, 1971). This analysis, however, should predict a shorter latency in Groups 2 and 4 than in the other groups. Such was not the case. Furthermore, the opposite effect was observed on other buzzer preceded trials.

trials, prior to the presentation of the disinhibitor with the performance of a non-stimulated group. The control group Ss received stimulation on one of the last 3 acquisition trials. It was found that when the 500 Hz, 100 db SPL novel tone stimulus was sounded, control Ss ran significantly more slowly, while experimental Ss ran significantly more rapidly. The disinhibitory effect was of greatest magnitude on the first post-test trial and dissipated rapidly, being no longer significant relative to the control group by the fourth post-test trial. Thus, disinhibition was demonstrated, but there was no difference between the experimental groups in magnitude of effect. This fails to replicate the strong correlation Razran (1939) found, and the authors suggest this may be due to differences in "depth" of extinction. Thus, after 24 trials, Razran's Ss were giving an average response 75% lower than the first extinction trial, whereas in the Yamaguchi and Ladioray study, the mean response had been reduced to 83% of the original by trial 10. It is possible, then, that the magnitude of disinhibition varies systematically with amount of extinction only until late in extinction.

Having successfully demonstrated external inhibition and disinhibition, the authors performed a second experiment by manipulating the db level of the tone in acquisition and extinction. Tones of 0, 45, 58, or 85 db were presented on trial 116 of acquisition or trial 36 of extinction. The tones remained on during the entire latency period—the time required to traverse approximately 4/5 of a circular runway. Once again, external inhibition occurred, but only at the 45 and 85 db levels, the stronger effect being at 85 db. Disinhibition also occurred, but only with the 45 db tone. In fact, the effect with the 85 db tone was suppressive, although not significantly. The failure to find disinhibition in the 85 db group does not seem compatible with the disinhibition demonstrated with a 100 db tone in the first experiment. Procedural differences between the two experiments--a shorter straight runway was used in experiment 1, as well as 60 as opposed to 80 acquisition trials--might account for the difference in results, but it still seems puzzling that similar stimuli could produce robust disinhibition in one experiment and a non-significant trend in the opposite direction in the next experiment.[7]

The most recent experiment involving disinhibition in extinction was reported by Franklin, Hearst and Mueller (1974). In a series of studies, pigeons were placed on discrete-trial FI schedules and then given extinction. Franklin et al. found that a switch from a 25-sec ITI to a 5-sec ITI following extinction produced a recovery of responding; but birds which had been trained and extinguished using a 5-sec ITI and were then shifted to a 25-sec ITI did not show any recovery. Although disinhibition was clearly demonstrated, the lack of symmetry remains puzzling.

In concluding this section, there seems to be reasonable evidence that disinhibition in extinction, as well as external inhibition in acquisition, can be demonstrated. The phenomenon does not, however,

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[7] Additionally, Pennypacker (1964) presented evidence that disinhibition can occur during extinction of the human eyeblink. This evidence was ambiguous, as Pennypacker himself points out, due to lack of a control group.

seem to be as robust as a reading of Pavlov's writings might lead one to believe. Furthermore, what few parametric studies of the phenomenon have been performed do not elucidate any consistent relationship between any independent variables and the magnitude, or existence of, a disinhibiting effect.

b. Disinhibition in Pavlovian Conditioned Inhibition

What seems to be the only study of disinhibition in PCI was reported by Hunter (1938). In a series of studies using finger withdrawal avoidance, Hunter conditioned Ss to respond to one stimulus, but not to respond when that stimulus was compounded with another; this is analogous to the procedure utilized by Marchant and Moore (1974). A buzzer presentation to which the unconditioned startle, and subsequent finger withdrawal, response had been habituated was used as the disinhibitor. For example, in one experiment Ss were trained to differentiate between the reinforced presentation of a single light and the nonreinforced presentation of two lights. Following establishment of the discrimination, the startle response to the buzzer was habituated by presenting it during the 59-sec ITI and preceding both trial types. The buzzer was then presented simultaneously with the CS- compound and six of the seven Ss responded to the CS-. Similar experiments were performed using either visual presentation of the word "no" preceding a single light, or the phrase "Don't lift your finger" delivered via earphones preceding the phrase "Lift your finger" (CS+), as the CS-. Similar results were obtained in each experiment with 4/6 and 5/8 Ss showing disinhibition on the buzzer trial.

The disinhibitory effect demonstrated, however, is confounded by a failure to control certain independent variables. First, the Ss were generally not naive, since many of them were used in more than one experiment. Of the 21 Ss used in the three experiments described above, only three were experimentally naive. A more serious problem, however, was the use of the buzzer as the disinhibiting stimulus. The buzzer produced the withdrawal response as an unconditioned effect. Despite the habituation of this startle response over trials, the habituation was done during the ITI and one might reasonably expect that the presentation of the buzzer in compound with CS- for the first time might produce dishabituation. Finally, no unstimulated control group was included to ensure that the "disinhibited" responses were not merely random variations in performance during the ongoing differentiation.

Thus, the only PCI study in the literature which purports to demonstrate disinhibition resulted in a seeming increase in the probability of a CR. This disinhibitory effect, however, seems likely to have actually been caused by dishabituation of an habituated response.

c. Disinhibition of Inhibition of Delay

Pavlov, as described earlier, reported successful disinhibition of a "delay" inhibitor in both a delay and a trace paradigm. Although the Pavlovian delay paradigm is believed to produce an active inhibitor (Rescorla, 1969), only one study since Pavlov has been reported attempting to disinhibit inhibition of delay.[8] Kimmel and Greene (1964)

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[8] Rodnick (1937) reports trying to disinhibit a long-delay CR in the GSR, but does not describe the results.

conditioned the GSR in human Ss for 0, 1, 2, 10, 25, or 50 trials. A novel 100 msec, 3,000 Hz tonal stimulus (described by the authors as a "squeak") was presented to all Ss in each group at various points during the 7.5 sec ISI. An increase relative to the preceding trial in GSR following the tone presentation occurred, primarily in the two groups with the most training. While the authors regarded this as evidence of disinhibition, it does not seem to be terribly compelling evidence. Specifically, one would expect the novel stimulus to produce an increment in CR amplitude during the first part of the CS and, perhaps, a decrement during the second part (as Pavlov found), if the CS were inhibitory--which was never demonstrated. However, the greatest CR amplitude increment occurred during the last half of the CS. Hence, it seems unlikely that disinhibition of an inhibitory stimulus occurred.

A number of analogous studies in the operant literature are available. These studies are analogous to the extent that the behavior produced after extensive training with a fixed interval (FI) schedule of reinforcement is a "scallop" during the interval. That is, the animal responds very little during the first half of the interval, with the bulk of its responses occurring during the second half of the interval. Such a response pattern closely resembles the increasing response magnitude seen in a long-delay or trace paradigm in a Pavlovian preparation. However, although the pattern of responding in both the operant and classical preparations is similar, while reviewing the studies involving disinhibition in an FI schedule to be described below it should be remembered that, as of yet, no study has demonstrated the first part of an FI scallop is truly inhibitory. In fact, the opposite seems to be true. Miller and Ackley (1970) have found that when light and tone stimuli, each controlling responding in an FI schedule of reinforcement, were presented as a compound, the compound controlled a response rate higher than either of its components. This finding held true for the first half of the interval, as well as the second, suggesting that a stimulus correlated with an FI interval is excitatory, not inhibitory, and is therefore probably not an appropriate analogy to a Pavlovian trace CS.

The first description of the effect of a novel stimulus on an FI scallop was given by Ferster and Skinner (1957, p. 319). A pigeon whose performance had been stable for a long time on an FI 39'' schedule was switched to an FI 20'' schedule and the response key was partially covered—a novel stimulus. The result was that the scallop, in essence, flattened with the first half elevated and the second half somewhat depressed. Theoretically, then, such a result suggests the occurrence of both disinhibition and external inhibition, but the effects are confounded by the simultaneous switch to the FI 20''.

In an attempt to present a clearer demonstration, Flanagan and Webb (1964) trained five albino rats to bar press for water on an FI 1' schedule. After 15 one-hour sessions, a white noise stimulus 5 db above the background noise (85 db) was turned on 10 or 40 sec into each of 4 intervals during the 16th session. The noise terminated at the end of the one-minute interval. Each presentation that onset 10 sec into the interval produced a marked elevation of mean response rate, but neither type of presentation affected the latter half of the curve. Training on the FI 1' was resumed for four weeks and a similar test session was run, this time using a vibrator attached to the outside of the Skinner Box as the disinhibitor. This time the presentation starting 10 sec into the interval flattened the scallop and the 40 sec presentation reduced response rate in the last third of the intervals. In both experiments the performance during intervals containing no stimulation exhibited the normal scallops. Although no statistical tests of significance were reported, these results suggest an operant analogy to Pavlov's report of disinhibition of inhibition of delay. Furthermore, the discrepancy in results between the noise and vibratory stimuli might be accounted for by assuming the 5 db rise in noise was a "low strength" stimulus, so it produced only disinhibition, while the vibratory stimulus was of "medium" strength and produced both disinhibition and external inhibition.

In a somewhat similar demonstration, Singh and Wickens (1968) trained 24 albino rats to bar press for food on a cued FI 3' schedule—the cue was the termination of light at the end of the FI interval. After an average of 16 days of training with 10 trials per day, a buzzer was introduced which raised the noise level in the box from 57 db to 68 db. For one group of Ss (n = 8), the buzzer was on throughout an interval; for another group, it was on only during the second half. In all groups the buzzer was presented on each of the 10 intervals that constituted the session. In all groups response rate during the first half of the FI intervals was approximately the same response rate as in the second half of the intervals (i.e., the scallop flattened). Presumably, the presentations of the buzzer during only the half intervals had an effect on the other half due to the after-effects of the buzzer (Pavlov, p. 96). Testing was continued for 3 more days, but was marked only by a return to scalloping. This is perhaps analogous to Pavlov's contention that the effects of a novel stimulus decay in a relatively short period of time (Pavlov, p. 98).

Similar results were obtained by Hinrichs (1968) using three pigeons. The birds were trained to key peck for food on an FI 1' schedule with the first 5 seconds of each one minute being a blackout. Training was continued until performance stabilized and then 4 sessions were run in which six of the 60 intervals were marked by a change in key color. Such a change produced a highly significant ($p<.01$) flattening of the scallop relative to intervals on which the original colored key was presented. Following 3 more sessions of training with the original color, the key color was changed 1/3 of the way through the next session. Such a change flattened the scallop, but a recovery occurred within 15-20 trials. Training with the new key color continued for 10 sessions. Then, a test similar to the first test was conducted, only this time the "novel" stimulus was the original key color. Results of this last test were similar to the first test, but the return to the scallop was more rapid--presumably due to the prior experience with the disinhibitor.

The most recent attempt to obtain disinhibition during an FI interval was made by Stucka (1971). Eight albino rats were trained to bar press for water on an FI 1' schedule for 1 hour/day until performance stabilized--between 15-20 sessions. Ss were then randomly assigned to one of four levels of novel stimulus intensity. This disinhibitor was a 2,000 Hz tone 10 (75 db), 20, 30, or 40 db above the background noise. The tone onset 10 min into the session and terminated at the end of the session. The results showed basically that the two weaker tones had no significant effect in either half of the interval; the next tone significantly increased response rate relative to the previous day's session during both halves of the intervals; while the loudest tone had the opposite effect, decreasing response rate--significantly only in the second half--in both halves of the intervals. Both disinhibition and external inhibition were demonstrated, but each at only one of the four levels of the independent variable. While the pattern of results is not in line with the previously described research, the generality of the results seems limited by the small number (two) of Ss in each group.

In concluding this section, then, it is clear that effects analogous to both disinhibition and external inhibition in an FI schedule can be demonstrated, but are not always obtained. The causes of failure are not clear, nor is the relationship between novel stimulus intensity and amount of disinhibition clear. These points will be elaborated on in greater depth in the next section.

3. Reported Failures to Obtain Disinhibition

The first, and by far most influential, failure to obtain disinhibition was described by Skinner (1936). Skinner utilized rats with a past history of acquisition extinction cycles and presented them with a novel stimulus some time during extinction. The novel stimuli used consisted of: throwing the rats in the air, pricking their tails with a needle, clicks from the food magazine, or a 3 candle power light turned on in the Skinner Box. Generally, he found no change in response rate, or a slight decrease. When the rats did show an increase in response rate, such an increase merely brought the cumulative response record up to the extinction "envelope"--the curve interpolated from the initial response rate in extinction. Thus, any evidence of disinhibition in the experiment was regarded as bringing response rate "back to where it should be." No control groups were employed to ensure what the response rate was "supposed" to be. Skinner, then, was unable to find evidence of disinhibition in extinction, but, in light of the stimuli used, the criterion for disinhibition employed, and the lack of experimentally naive Ss, the generality of this result remains ambiguous. At best it supports the contention that disinhibition is not easily obtainable.

Another often cited failure to obtain disinhibition was reported by Rexroad (1937). Rexroad, however, used a rather unusual procedure in attempting to obtain disinhibition. Using primarily human females in a reaction time paradigm, Rexroad standardly had a buzzer precede a light by 3 sec. The light was a signal to the S to turn a crank;

the crank turning response was used as a measure of reaction time. Following training, Ss presented with a "nonreinforced" buzzer trial tended to decrease response latency on following buzzer-light trials. During this depression of latency, presentations of a bell and "tickler" (vibro-tactile stimulus) did not increase reaction time, even when presented prior to or simultaneous with the presentation of a buzzer. This failure to obtain "disinhibition," while influential in the past literature, is perhaps best disregarded.

A study described by Winnick and Hunt (1951) utilized a procedure similar to that used by Gagné (1941). During 15 acquisition and 8 extinction trials in an alleyway, a buzzer was sounded at certain times; each of four groups received one presentation during acquisition on trials 4, 8, 12, or 16 and during extinction on trials 2, 4, 5, or 6. Each group showed significant external inhibition in acquisition with the exception of the presentation on trial 14. This group, however, showed shorter latencies throughout extinction than did the unstimulated control group and, hence, never exhibited disinhibition. The group given a buzzer presentation on extinction trial 2 responded significantly more rapidly (t-test, $p<.05$) than did the control group. A similar effect was observed for the groups stimulated on trials 4 and 5, but the effect was not significant. Hence, although this study was reported as a failure to obtain disinhibition, the results do not seem to greatly contradict other demonstrations of disinhibition during extinction. The results do, however, once again imply that parametric manipulations of the phenomenon are not always easily demonstrable.

Reinforcing this last point, a careful parametric study was performed by Gormezano (1958). Using the human conditioned eyelid paradigm, this E had Ss acquire, then extinguish, a CR. A 4 sec white noise burst was then interpolated 0, 4, 8, or 16 sec prior to the next CS presentation. Additionally, the noise was either 0, 77.5, 87.5, or 97.5 db. Unfortunately, not only was there no effect of any of the parametric manipulations, but furthermore, there was no evidence of disinhibition. Perhaps the fact that Gormezano included only those Ss who had extinguished their CR within 13 trials (almost all had) had some bearing on this outcome. Although this finding does not raise serious doubt about the existence of the phenomenon, it does once again suggest that parametric manipulations are not easily made and that the preparation utilized may be of importance in obtaining a successful demonstration of disinhibition.

Another perplexing failure to obtain disinhibition was reported by Wolach and Ferraro (1969). These investigators attempted to obtain disinhibition and external inhibition in an FI interval. Rats of the same age and strain as those used by Flanagan and Webb (1964) were trained to bar press for a water reinforcer. After 5 sessions on CRF and 20 sessions on an FI 1' schedule, the eight Ss were presented with a novel stimulus during 5 of the 100 intervals in the next 4 sessions. The disinhibitor was an auditory stimulus of 4 clicks per sec at either 65, 66, 67, 68, or 69 db. Hence, each S received one presentation of the clicker at each db level on each of 4 successive days. Relative to the three pre- and three post-stimulation intervals, no change in response rate was found during the first 1/5 of the stimulated intervals. During the third 1/5 of the stimulated intervals, there tended to be an increase in response rate with the lower db clicks and a decrease at higher intensities. Unfortunately, the rates for the second 1/5 were not reported, making it impossible to ensure that disinhibition was not found. Finally, the last 1/5 of the intervals showed a powerful external inhibition of response rate. After 2 more days of FI training, somewhat similar manipulations were made utilizing a 1,000 Hz tone at either 62 or 68 db for 2 sessions. Results similar to the others were obtained--only external inhibition was clearly demonstrated.

These results, although not completely unambiguous, fail to replicate those of Flanagan and Webb (1964). This lack of consistency was also obtained in a second experiment in which a 65 or 69 db clicker was presented during the second or last fifth of 5 FI intervals over 2 days to FI-trained rats. Despite the fact that between- instead of within-group testing was used in this experiment, the results were similar to those obtained in the first experiment; robust external inhibition occurred during the latter segments of the interval, but no concrete indication of disinhibition was obtained.

Another puzzling failure to obtain disinhibition in extinction was reported by Boakes (1974) and Boakes and Halliday (1975). These investigators essentially replicated the procedure reported by Brimer (1972) and failed to obtain disinhibition. The reasons for this failure to obtain disinhibition are, like that of Wolach and Ferraro, unclear.

The studies reviewed in this section on failures to obtain disinhibition, then, do not cast serious doubt on the existence of the phenomenon. Instead, these studies point to the fact that disinhibition is not always an easily replicable phenomenon within or across preparations. Furthermore, no one group of parameters seems to consistently produce the phenomenon, nor does any simple parametric manipulation seem to produce consistent results across experiments.

Overall, the phenomenon of disinhibition is elusive. It can be demonstrated, in both classical and instrumental preparations, but not consistently. It seems at times to produce orderly parametric data, yet across experiments, the orderliness proves illusory. In short, the phenomenon represents an empirical and theoretical puzzle.

4. Theoretical and Empirical Considerations

Before attempting an empirically sensible theoretical discussion of the phenomenon of disinhibition, a resumé of the work of the late C. J. Brimer (cf., Brimer, 1972) is called for, as he was the one experimenter since Pavlov most concerned with disinhibition. The review will be brief, however, for Brimer defined the phenomenon of inhibition very broadly. For him, any manipulation that reduced response rate constituted inhibition. Thus, a decrease in responding due to punishment by electrical shock,[9] a DRL schedule of reinforcement,[10] a CER,[11] satiation, etc., all were considered inhibitory manipulations.

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[9] See also Brimer & Kamin (1963).

[10] See also Contrucci, Joyce, Hothersall & Wickens (1971).

[11] See also Adkins (1970).

Following this, if a novel stimulus presentation produced an increase in response rate, disinhibition was said to have been demonstrated. Despite the fact that Brimer's "inhibitory" manipulations were not those generally conceived of as being truly inhibitory (Hearst, 1972; Rescorla, 1969), Brimer's work produced one very consistent pattern of results: Given that a manipulation has reduced (rather than produced) response rate to a certain critical level, the response rate can be "disinhibited" by virtually any salient stimulus.

Although this critical rate--1/2 response per minute--is less than that generated in other studies demonstrating disinhibition,[12] a more important theoretical consideration remains: Is "disinhibition" limited only to inhibitory situations, or is it a far more general phenomenon? In fact, in light of all the evidence reviewed, can disinhibition of a conditioned inhibitor actually occur?

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[12] For example, Flanagan & Webb (1964), Yamaguchi & Ladioray, 1962, etc.

APPENDIX B: Percentage CRs to CS+ and CS-, as well as mean CR magnitude on the last day, over 8 days of conditioned inhibition training.

			Day								CR mean magnitude in mm
	S#	CS	1	2	3	4	5	6	7	8	
Group B-0	1	+	0	2	2	14	98	98	100	100	4.48
		-	0	2	2	2	76	84	100	98	4.20
	3	+	0	12	100	86	90	90	88	98	23.88
		-	0	6	86	12	42	26	64	86	11.30
	4	+	50	90	100	98	100	100	100	100	10.45
		-	50	90	100	100	100	100	100	100	11.08
	8	+	36	98	98	98	92	100	100	100	8.25
		-	20	76	86	84	88	94	94	98	4.75
Group 0-B	2	+	0	0	4	0	6	78	76	74	4.18
		-	0	0	2	2	0	62	66	66	2.08
	5	+	0	4	82	94	100	98	100	96	12.68
		-	0	6	76	94	96	98	98	90	7.20
	6	+	36	20	60	98	100	98	98	100	25.49
		-	22	16	80	18	98	100	100	100	28.90
	7	+	4	8	2	70	98	100	100	98	10.72
		-	10	20	4	66	82	88	100	100	8.42

APPENDIX C: Probability of a CR and mean CR amplitude over 4 days of testing to T and BT.

		Day							
		1		2		3		4	
	S#	T	BT	T	BT	T	BT	T	BT
Group E	25	.20	.20	.40	.20	0	0	0	0
	26	0	.20	1.0	.80	.60	.60	.20	.20
	27	.40	.20	1.0	1.0	1.0	.80	1.0	1.0
	28	.60	.60	.60	0	.80	.20	1.0	.20
	$\bar{x}$ =	.30	.30	.75	.50	.60	.40	.55	.35
								$\bar{x}$ diff. over 4 days = .16	
Group C	29	.75	.60	.60	0	.60	0	.40	.20
	30	.75	.60	.20	0	.40	.40	.80	0
	31	1.0	.80	.40	.20	1.0	.60	.60	.40
	32	.40	.40	0	0	0	0	0	0
	$\bar{x}$ =	.73	.60	.30	.05	.50	.25	.45	.15
								$\bar{x}$ diff. over 4 days = .23	
				Mean Amplitude					
		T	BT	T	BT	T	BT	T	BT
Group E	25	1.0	1.0	1.0	1.0	0	0	0	0
	26	0	1.0	1.60	1.25	1.0	1.3	2.0	1.0
	27	13.5	1.0	9.6	17.6	15.0	15.5	10.8	7.5
	28	14.7	8.0	8.0	0	6.0	1.0	11.6	11.0
Group C	29	2.33	4.33	1.0	0	4.0	.0	3.0	1.5
	30	6.33	2.33	1.0	0	2.5	1.5	15.25	0
	31	5.0	4.50	1.0	1.0	3.3	2.4	3.0	3.0
	32	3.5	3.5	0	0	0	0	0	0

APPENDIX D: Hull's Derivation of Disinhibition
(Taken primarily from Hull, 1943, pp. 288-289)

In Hull's 1943 system, whether an S responds depends on $_S\bar{E}_R$—effective reaction potential. $_S\bar{E}_R = {_SE_R} - I_R$, where $_SE_R$ is simple reaction potential and I_R is aggregate inhibitory potential, a summation of I_R—reactive inhibition and $_SI_R$—conditioned inhibition. Given, then, the equation $_S\bar{E}_R = {_SE_R} - (I_R + {_SI_R})$, it is assumed that a novel stimulus affects only the learned components of the equation ($_SE_R$ and $_SI_R$) through afferent neural interaction (essentially, generalization decrement in this case). Still, the effect should equally decrement both processes. To circumvent this, Hull accepts Pavlov's statement that conditioned inhibition is more labile than excitation. The effect of the novel stimulus, then, decrements $_SI_R$ more than $_SE_R$ and disinhibition occurs.

Finally, since the novel stimulus affects only $_SI_R$ and not I_R, Hull is able to predict that CR increment or restoration during extinction will never be complete.