DISINHIBITION OF AN OPERANT RESPONSE

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A total of 320 rats were employed in experiments analyzing the phenomenon of disinhibition of an operant bar-pressing response. The parameters explored were type of "inhibitory operation" preceding the test for disinhibition, and modality, duration, directionality, intensity, and prior exposure to the disinhibiting stimulus. The disinhibition phenomenon was highly general, occurring under almost all test conditions. The phenomenon, however, could be produced during extinction only when probability of response was at a "critical" low, but greater than zero, level. When animals with the same "critical" response probability were tested during acquisition, before bar-pressing had reached a higher level of probability, disinhibition never occurred. Theories attempting to account for both the inhibiting and disinhibiting effects of extraneous stimuli were examined.

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CHAPTER ONE

INTRODUCTION

The problem with which this dissertation is concerned stems directly from some earlier experimental work (Brimer and Kamin, 1963). The relevant portion of the earlier work can be summarized very briefly. First, rats were trained to press a bar for a food reward. The training procedure resulted in a stable rate of bar-pressing behavior. Then, "free", un-signalled electric shock was administered to the animals, programmed independently of their bar-pressing behavior. The result of this experience was to radically reduce the baseline response rate. Finally, a white noise stimulus of three minutes duration was presented to the rat; in the presence of this extraneous stimulus there was a marked increase in the rate of bar pressing. This occurred with virtually all subjects tested.

This temporary re-appearance of an inhibited response in the presence of an extraneous stimulus seemed reminiscent of the phenomenon of "disinhibition", first described by Pavlov (1927). Though Pavlov studied classically conditioned responses, and our own procedure was that of operant conditioning, the descriptive similarity of the effect we noted, to that of Pavlovian disinhibition was striking. However, inhibition of responding by the use of electric shock was not one of the inhibitory procedures used by Pavlov to demonstrate disinhibition. The inhibitory procedures employed by Pavlov all involved selective non-reinforcement of a conditioned stimulus. Further, Pavlov had demonstrated that within classical conditioning disinhibition could be produced with extraneous stimuli in many sensory modalities. Our effect might well be specific to the use of noise and/or electric shock. Thus, one aim of the present research was to establish the generality of the effect which we had observed, thereby further justifying its identification with Pavlovian disinhibition.

While Pavlov frequently demonstrated the occurrence of disinhibition, he did not do any rigorous experimental work on the parameters which control the effect. The concept of disinhibition was instead speedily incorporated into the circular framework of Pavlovian "neurophysiological" theory, and was used to explain other phenomena before it itself was sufficiently understood. This lack of parametric study is not specific to Russian research on disinhibition. While several North American investigators have published reports demonstrating the effect - or analogues to it, - none has reported a detailed parametric study.

There is thus surprisingly little information on the variables which control disinhibition. This is doubtless in large measure due to the commitment of research workers in this area to the Pavlovian theoretical approach to the problem. The present research was conceived as a theoretically non-committed experimental attack on the problem of the effects of extraneous stimulation on operant behavior. We assumed that theoretical interpretations suggested by our own data would be relevant, as well, to the Pavlovian phenomenon.

We report some experiments dealing with characteristics of the extraneous stimulus which produces disinhibition,and some experiments concerned with the question of whether - as in the Pavlovian demonstration -

the _same_ extraneous stimulus which disinhibits a suppressed response _inhibits_ an "intact", full-strength response. These experiments, designed to fill gaps in existing knowledge, will be described in detail in subsequent chapters. We shall first, however, have to trace the history of previous research on the disinhibition topic.

CHAPTER TWO

HISTORY OF RESEARCH ON DISINHIBITION

Research by Pavlov on Disinhibition

The phenomenon of disinhibition is inextricably connected with the classical work on conditioned reflexes carried out by Ivan P. Pavlov (1927). Pavlov first observed the empirical fact, coined the name of "disinhibition", and in his published works referred to the phenomenon repeatedly. Disinhibition was one of the empirical cornerstones on which Pavlov erected his neurophysiological theory of behavior. Thus, it would be inconceivable to begin a discussion of the history of research on the problem of disinhibition at any point other than Pavlov. We shall start with an extensive description of Pavlov's work; and because the empirical phenomenon called "disinhibition" was from the outset enmeshed in the web of Pavlovian neurophysiological theory, it will be necessary to review some aspects of the theory at length. This peculiar theory, with its almost total disregard for the synaptic nature of the nervous system, has had little impact on Western psychology or neurophysiology. For better or for worse, however, it has left an indelible imprint on all subsequent disinhibition research. This will become clear when, following our review of Pavlovian work, we turn to North American research on disinhibition.

The phenomenon of disinhibition is exemplified by Pavlov (1927, pp.65-67) in the following experiment. A hungry dog is first presented with meat powder at a distance, and is then allowed to eat. Repeated experiences of this sequence of events result in the mere presentation of the meat powder reliably eliciting a salivary response.[1] In Pavlovian conditioning terms the sight of the meat powder at a distance is the conditioned stimulus (CS) and the actual presence of the food in the mouth is the unconditioned stimulus (US). Initially the CS is neutral[2] in regard to any "food reaction", while the US *reflexively* elicits a complex of responses such as chewing, swallowing, salivating, etc., which collectively are designated the unconditioned response or UR. When, as a consequence of repeated CS - US pairings, salivation (a reaction similar to the UR) comes to be elicited by the CS, a conditioned response (CR) is said to have been acquired. Now, after the acquisition of the conditioned response, when the CS is repeatedly presented without the US (i.e., the CS is not reinforced), the CR progressively diminishes until it no longer occurs. This process, referred to as experimental extinction, depended, according to Pavlov, on the accumulation of "internal inhibition". The CR has not literally been destroyed, but is being

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[1] That this is an acquired reaction was demonstrated by the fact that dogs raised on a liquid diet do not salivate to solid food presented at a distance (Pavlov, 1928, pp. 266-267).

[2] Employing meat powder presented at a distance as the CS does not do full justice to the neutrality of most of the CS stimuli (bells, metronones, lights, etc.) that Pavlov employed. The only reason that this particular example was chosen is that it is one of the few experimental demonstrations of disinhibition that is reported by Pavlov is some detail.

actively inhibited.[1] This sets the stage for demonstrating disinhibition. For, if the CS is now presented accompanied by an "extra stimulus", the salivary CR again manifests itself. The extra stimulus is the disinhibitor - the reappearance of the CR is the empirical phenomenon of disinhibition attributed by Pavlov to hypothetical cortical processes soon to be described. The actual experimental results given by Pavlov (1927, p. 65) appear in the accompanying Table.

Time	Stimulus applied during one minute	Amount of Saliva in drops during one minute
1.53 p.m.	Meat powder presented at a distance.	18
1.58 "		6
2.3 "		0
2.8 "	Same + tactile stimulation of skin.	4
2.13 "	Same + knocks under the table.	3
2.18 "	Meat powder at a distance.	0
2.20 "	Prof. Pavlov enters the room containing the dog, talks, and stays for two minutes.	
2.23 "	Meat powder at a distance.	7
2.28 "	Same.	0

In this example, three disinhibitors were employed: tactile stimulation of the skin, knocks under the table, and the presence of Professor Pavlov in the experimental room. (These somewhat "rough-and-ready" relatively

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[1] Pavlov supports the idea of an active process of inhibition by the following three facts. First, a CS which has been extinguished is capable of suppressing any CR (with which it has not previously been associated) if it is presented together with the CS for the non-extinguished CR. Second, there is "spontaneous recovery" of an extinguished CR, simply with the passage of time. Third, and likely most important, is the very demonstration of disinhibition, where an extinguished CR can be temporarily restored through the action of an extraneous stimulus.

uncontrolled stimuli are not uncharacteristic of early Pavlovian work.) It should be noted that all three stimuli had been repeatedly shown in previous experiments not to produce any secretory effect, in and of themselves. They seem instead to have the property of temporarily restoring an extinguished conditioned response.

From this example we can note three things. First, different "extra stimuli" can act as disinhibitors, at least partially restoring an extinguished conditioned response. Second, the restorative effect is temporary. There was no disinhibitory effect of table knocking four minutes after it occurred. But third, the extra stimulus need not still be present at the time of testing; disinhibition occurred _after_ Pavlov had left the experimental room.

Perhaps the most intriguing fact about disinhibitory "extra stimuli", not illustrated in the present example, is this. When external stimuli such as tapping or skin stimulation are presented in conjunction with the CS for an _unextinguished_, "intact" CR, they have an _inhibiting_ effect. That is, they decrease the magnitude of the CR. This apparent paradox - that the same stimulus which impedes an intact CR, facilitates an extinguished CR - was resolved by Pavlov (1928) as follows:

> ... if you, having to do with an inhibitory process in the nervous system, join to this inhibited stimulus some new extra agent, the inhibited stimulus now manifests its own effect. This fact may be understood thus: the new extra agent inhibits the inhibition and as a result there is a freeing of the previously inhibited action i.e., a positive effect. ... This is the phenomenon of inhibition of inhibition. (Pavlov, 1928, p. 109).

Thus, the extra stimulus in some basic sense always "inhibits". When it "inhibits inhibition", we observe the phenomenon of disinhibition - the temporary restoration of an inhibited conditioned response due to the presentation of an extraneous stimulus. According to Pavlov, disinhibition is not a rare, artificially-contrived laboratory phenomenon "but a striking reality of which we are freshly convinced every day" (Pavlov, 1928, p. 150).

There seem to be two obvious questions to ask in regard to disinhibition; (1) what are the properties of stimuli which can act as disinhibitors, and (2) what types of inhibited responses can be disinhibited? We shall explore what Pavlov had to say in regard to each of these two questions before turning to his neurophysiological model.

<u>The Disinhibiting Stimulus</u> Pavlov was not entirely consistent in his descriptions of what stimuli functioned as disinhibitors. First he repeatedly points out that effective disinhibitors are "stimuli belonging to the group of mild external inhibitors" (Pavlov, 1927, p. 82). An "external inhibitor" was any stimulus which diminished the CR of a non-extinguished conditioned reflex. The designation "external" was used to denote the fact that the effect of the stimulus was assumed to be due to its inherent properties, rather than to any acquired or conditioned properties.[1]

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[1] In this regard Pavlov states that "the most striking difference between external and internal inhibition is that, whereas .... external inhibition is produced on the very first application of an extra stimulus, internal inhibition on the other hand always develops progressively, quite often very slowly, and in many cases with difficulty". (Pavlov, 1927, p. 48). In this classification internal inhibition comes about through non-reinforcement of stimuli which at one time elicited the CR. External inhibition, on the other hand, is due to the elicitation of an "orienting reflex" that blocks the CR. Although this is the most typical use of the terms by Pavlov he did propose, at other times, somewhat different classifications.

Originally the presence or absence of an external stimulus was the basis for calling inhibition external or internal. (see e.g., Pavlov, 1928, p. 125). At that time "conditioned inhibition" was considered a case of external inhibition and only in the later system did it become an example of internal inhibition.

Sleep was originally considered a third type of inhibition (in addition to external and internal) but later it was designated a diffuse

At another time Pavlov stated that "Every unusual stimulus from the external world may be .... a disinhibiting agent" (Pavlov, 1928, p. 149). Elsewhere Pavlov mentions that disinhibition may be produced by "any additional agent of average strength which provokes the orienting reaction (looking, listening,etc.) of the animal" (Pavlov, 1928, pp. 230-231).[1]

With regard to the "mild" qualification for effective disinhibitors, Pavlov says "dis-inhibition is manifest only under certain conditions, viz., if the dis-inhibiting agent is of average strength (not very strong and not too weak). .... It is essential that this agent be of definite strength, neither too powerful, lest it inhibit the stimulus, nor too weak, lest it can not inhibit the internal inhibition. Only under these conditions is there complete dis-inhibition" (Pavlov, 1928, p. 211). Sidestepping for a moment the vagueness inherent in terms like "mild" or "average",one may ask just what it is that must possess the proper intensity. Sometimes Pavlov uses "mildness" in reference to the external inhibition properties of the stimulus, sometimes in reference to the orienting reflex, elicited by the stimulus, and yet other times simply designates the disinhibitor as a "mild" stimulus. Perhaps an experimental example will help to clarify Pavlov's use of the term. The type of inhibition investigated was what Pavlov termed "inhibition of delay". This phenomenon takes the following form. The CS rather than being presented just a few seconds prior to the US is presented continuously for three minutes before being reinforced by the US. After repeated stimulus

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internal inhibition.

In a final pronouncement Pavlov suggested that there was a strong probability that "external and internal inhibition are fundamentally the same" (Pavlov, 1927, p. 388).

[1] Although the orienting reflex was supposed to be identifiable in terms of the behavioral reactions of the animal (e.g., moving towards the stimulus, pricking-up of ears to an auditory stimulus, etc.) the concept was such as to be readily anthropomorphized. Thus one feels that Pavlov is usually employing the term to stand for an intuited "rapt attention" on the part of the animal.

sequences of this nature the CR comes to appear only during the latter half (1 - 1½-minutes) of the CS interval. The CR during the initial 1½-minutes of CS presentation is "inhibited". In the experiment which Pavlov (1927, pp. 94-95) reports, a series of extra stimuli were introduced at different time intervals after onset of the CS, and their effects on the CR were noted. On the basis of the results obtained the stimuli were grouped as follows:

> I Thermal stimuli at 5°C. and at 44°C.; a weak odour of camphor.
> II Thermal stimuli at 0.5°C. and at 50°C.
> III Noiselessly rotating objects; the sound of a metronome; tactile stimulation of the skin (the conditioned stimulus to the delayed reflex being in this animal a similar tactile stimulation of a different place on the skin); a whistle of moderate strength; the odour of amyl acetate.
> IV Intense odour of camphor; loud whistle; sound of an electric buzzer. (Pavlov, 1927, pp. 94-95).

For the empirical findings and their interpretation we can do no better than to quote Pavlov:

> Extra stimuli belonging to the first group did not in this dog affect either phase of the delayed reflexes. Extra stimuli belonging to the second group exerted an effect only upon the initial phase of the reflex, causing a salivary secretion. Extra stimuli belonging to the third group disturbed both phases of delay: during the first phase a salivary secretion was produced, and during the second phase the secretion which should normally have been present was much diminished. Extra stimuli belonging to the fourth group exercised little or no influence upon the initial phase of the delayed reflex, but completely suppressed the second phase (Pavlov, 1927, p. 95).

Thus, as Pavlov states elsewhere: "there is a graduated series of inhibition intensities - an ineffective, a disinhibiting, and an inhibiting". (Pavlov, 1928, p. 138). In the example that was given "The distribution of extra stimuli among the four groups represents a classification according to .... physiological strength" (Pavlov, 1927, p. 97). This, according to the author, may "... in some cases .... be seen by a casual glance at the list itself [but] .... the effects of these extra stimuli is also revealed by the motor reaction of the animal (investigatory reflex). With stimuli belonging to the first group there is frequently no motor reaction at all. As we pass on to stimuli belonging to the remaining groups the reactions become more and more vigorous and prolonged" (Pavlov, 1927, p. 97).

Thus, in answer to our question regarding the proper intensity for a disinhibitor, we must conclude that Pavlov used mildness in reference to the "physiological effectiveness" of the stimulus. Such "physiological mildness" may be estimated by either the physical intensity of the stimulus or the behavioral reaction of the animal, or more commonly by some combination of these two indices. Pavlov clearly suggests that the function relating the intensity of the extra stimulus to the magnitude of the disinhibition effect it produces is of an inverted U shape but the exact intensity necessary for maximal disinhibition remains obscure.

While considering disinhibitory stimuli, we must also take account of the time interval between the presentation of a disinhibiting stimulus and the testing for its effect. Pavlov clearly states that the effect of a disinhibitor may persist after the withdrawal or termination of the disinhibiting stimulus.[1] This was in fact one of the points which came to

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[1] In fact all stimuli, whether excitatory, inhibitory, or disinhibitory, were assumed to give rise to nervous activity which persisted after the termination of the environmental stimulation. Such a notion was necessary to account for phenomena such as trace conditioning, where the CS terminates prior to the US but a connection is made between the two events.

light in the first example which we gave to illustrate the disinhibition phenomenon. In that case the visit of Pavlov to the experimental chamber produced a partial recovery of the extinguished CR when the animal was tested one minute after Pavlov had left. The result of a second test five minutes later, however, revealed the CR to be again completely inhibited. The question then is, how long can the effect of a disinhibitor persist? Pavlov gives no precise answer, although in reference to the after-effects of stimulation involved in disinhibition he points out that "the after-effect can last from some seconds to some days" (Pavlov, 1928, p. 231).[1] We thus know only that a disinhibitor may show its effect some time after its application.

There is a final point to be made in regard to the properties of disinhibiting stimuli. With repeated or prolonged presentations, the stimulus rapidly loses its capacity to disinhibit. In an example cited by Pavlov (1927, p. 84) the odour of camphor lost its disinhibitory effect after one application. Although Pavlov does not specifically attempt to account for the rapid loss of disinhibitory power, it is not difficult to suggest two factors which might be operative. First, Pavlov (1928, p. 310) mentions that prolonged or repeated presentations of an external stimulus lead to the disappearance of the orienting reflex. If, as intimated earlier, a mild orienting reaction is a sufficient condition for disinhibition, then with the disappearance of this reaction we might expect some loss of disinhibitory capacity. Second, in the case of repeated presentations of a

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[1] Stimuli which elicited strong UR's were said by Pavlov to have lengthy after-effects running into hours or even days. The after-effect for most external inhibitors, however, was apparently in the order of minutes.

disinhibiting stimulus, what Pavlov termed "conditioned inhibition" would occur. (The conditioned inhibition phenomenon is discussed in a later section of this chapter.)

To sum up our answer to the question of what stimuli, according to Pavlov, can act as effective disinhibitors, we conclude with the following picture. All stimuli which produce a mild inhibiting effect on a non-extinguished conditioned response can act as disinhibitors, and these stimuli typically elicit an orienting reaction. Further a normally adequate stimulus may become ineffective after an animal has had prolonged or repeated exposure to it; and a disinhibiting stimulus may manifest its effect some time after its application.

The Disinhibited Response We turn now to the question of what properties a response must have in order for it to be disinhibited. The foremost requirement, according to Pavlov, is that the response must be suppressed through the operation of a hypothetical cortical process called "internal inhibition". There were four major empirical procedures which, in the theory, produced internal inhibition, and thus made it possible for disinhibition to occur. These were: extinction, differentiation, conditioned inhibition, and inhibition of delay.[1,2]

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[1] The phenomenon of disinhibition was employed by Pavlov as a means of demonstrating the presence of internal inhibition. Inhibition and disinhibition are not, therefore, independent concepts and it should come as no surprise that all cases of internal inhibition may be disinhibited - it could hardly be otherwise!

[2] Throughout Pavlov, the same term (e.g., "conditioned inhibition") is sometimes used to refer to an empirical training procedure and sometimes to a hypothetical cortical process.

The first procedure, experimental extinction of conditioned reflexes, refers to the "rapid and more or less smoothly progressive weakening of the reflex to a conditioned stimulus which is repeated a number of times without reinforcement" (Pavlov, 1927, p. 49). Experimental extinction is really the *sine qua non* of internal inhibition, for as will become clear all internal inhibition develops through non-reinforcement. It may seem strange to suggest that a CS which ceases to elicit the CR because it has been repeatedly presented without the US becomes an active inhibitor but, as mentioned earlier, Pavlov cited impressive experimental evidence to support such a notion.[1]

The second procedure, differentiation or differential inhibition, develops internal inhibition in the following manner. If a particular stimulus such as a tone of a certain frequency serves as the CS, then similar tones (to which the animal has not been previously exposed) will also tend to elicit the CR, attenuated in magnitude. If these similar tones continue to be presented without reinforcement, while the original tone continues to be reinforced, then a response differentiation develops. The original tone gives rise to the conditioned response, while the similar tones do not. Thus, the non-reinforced tones become inhibitory stimuli.

A similar type of inhibition is developed by the third procedure, which results in conditioned inhibition. Here reinforcement is withheld whenever the CS is presented in conjunction with an "extra stimulus", but the CS in isolation continues to be paired with the US. Under these conditions, CS - plus - extra-stimulus becomes an inhibitory stimulus complex,

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[1] The supporting evidence is given in the footnote on page 6. Although the concept of an active inhibitor is not alien to physiologists, it has not had much support from North American psychologists (cf. Diamond, Balvin, and Diamond, 1963).

and the "extra stimulus" a conditioned inhibitor. That the extra stimulus is a conditioned inhibitor is demonstrated by the fact that when presented in conjunction with a different CS it exerts an inhibitory effect (i.e., depresses the CR).

The fourth type of inhibition, inhibition of delay, was referred to previously when discussing Pavlov's experimental investigation of disinhibitor intensity. The phenomenon refers to the shift in the CR towards the latter half of a lengthy (e.g., 3-5 minutes) CS interval. This shift occurs under the procedure of delayed conditioning training. The fact that, after continued training, the CR does not occur early in the CS period means that internal inhibition is operative during this part of the CS period.

The common property of all four procedures which produce internal inhibition is obviously selective non-reinforcement of the CS. Pavlov referring to this fact states: "Internal inhibition develops when the conditioned stimulus is not attended by the unconditioned" (Pavlov, 1928, p. 308). Since the stimulus which becomes inhibitory was at one time capable of eliciting the CR, it is obvious that whether or not a stimulus produces internal inhibition depends upon the training which the subject has had with that stimulus.

Pavlov discusses the effect of three training parameters which affect the development of internal inhibition. First, the more non-reinforced trials the animal experiences, the more profound the inhibition. Thus, in the most extreme case, Pavlov demonstrated that unreinforced presentations of the CS, given after the CR had disappeared, still continued to add to the inhibition. Pavlov referred to this as "extinction below zero", in obvious reference to the fact that extinction training was continued even after the experimental protocols recorded zero salivation. The greater inhibition developed with sub-zero extinction was revealed by the longer time necessary for spontaneous recovery to occur, by the longer inhibitory after-effect of the extinguished CS, and by the longer time required to re-train the animal to respond to the CS. Second, the more established the CS is (i.e., the more the CS has been reinforced), the more difficult the development of the inhibition (i.e., the more non-reinforced trials necessary to reduce the CR to zero). And third, massed trials lead to more rapid acquisition of inhibition than spaced trials.

In summary then, we see that for Pavlov internal inhibition was the result of selective non-reinforcement, and that the greater the number of non-reinforced trials, either in the absolute, or per unit time, the more profound the inhibition that develops. Further, the weaker the CR, the more easily it is inhibited.

We thus have some notion of the variables which determine the degree of inhibition, but, Pavlov, unfortunately, gives no precise statement concerning the relationship between the degree of inhibition of a response and the ease with which it can be disinhibited. We can only suggest what Pavlov's early formulation of the problem would seem to imply. Pavlov at one time conceived of disinhibition as "inhibition of inhibition".[1] This might suggest that given a fixed external stimulus as the disinhibitor, the greater the inhibition of the CR, the more difficult it would be to disinhibit it.

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[1] At a later stage, Pavlov (1928) rejected the "inhibition of inhibition" explanation. He writes, for example, "if ... dis-inhibition were explained as a possible inhibition of inhibition, this would make more involved the understanding of an already complicated nervous reaction" (Pavlov, 1928, p. 317).

The answer to the question of what properties a response must have, in Pavlov's view, to be disinhibited, can be briefly summarized. First, any stimulus or any stimulus complex that once elicited a conditioned response will eventually cease to do so when repeatedly presented without reinforcement.[1] Second, the suppressed state of the conditioned response that comes about through non-reinforcement is due to the development of internal inhibition. And finally, all cases of internal inhibition can be disinhibited by the presentation of an appropriate external stimulus.

Pavlov's "Neurophysiological" Theory In recounting what Pavlov had to say about disinhibition we have attempted to exclude his views on the "cortical dynamics" involved in the phenomenon, although this is almost impossible to do.[2] The reason for this attempt has been two-fold. First, many of Pavlov's detailed comments on disinhibition were of an empirical nature, and second, it is not always clear how aspects of the theory which evolved at a later date apply to the disinhibition phenomenon. The picture

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[1] In the case of delayed conditioning one could argue that the "stimulus" is always reinforced. Pavlov would counter this suggestion by pointing out that temporal intervals can act as CS's. Thus in the case of delayed conditioning there would be two stimulus complexes, loosely designated as the early action of the stimulus (which is never reinforced) and the later action of the stimulus (which is always reinforced). Exactly where the dividing line falls is, of course, a moot question.

[2] It seems that when Pavlov referred to disinhibition as "inhibition of inhibition" he regarded this only as an empirical description of the phenomenon which did not "pretend ... to explain the underlying mechanism of disinhibition" (Pavlov, 1927, p. 67).

cannot be complete, however, without some reference to Pavlov's neurophysiological theory.[1] Thus, we shall give a brief outline of the Pavlovian theory and how it applies to the disinhibition phenomenon, although the predictive value of the model is, to say the least, somewhat questionable.

We may take as the starting point for Pavlov's theory the postulate that two active processes - excitation and inhibition - exist in the cortex.[2] The formation of a conditioned reflex then comes about as follows. A "neutral" stimulus impinging on the organism gives rise to excitation in the cortical point representing the stimulus; all sensory receptor elements are assumed to be represented by particular points in a "cortical mosaic". In the case of an unconditioned stimulus there is a direct "built-in" connection between the cortical representation and the motor centres controlling the unconditioned response. If one now presents the "neutral" stimulus (CS) in close temporal contiguity with the unconditioned stimulus (US), a bond is formed between the two cortical centres.[3] The CS becomes capable of activating the US centre and

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[1] American behavioral psychologists have since the time of John B. Watson (1916) heartily endorsed what Pavlov would consider simply the descriptive aspects of his work. What Pavlov considered his far more important contribution, viz., the theory of cortical activity, has been conspicuously ignored.

[2] The two molar constructs - excitation and inhibition - were not postulated by Pavlov *a priori*, but were rather inferred from the results of extensive experimentation. The neurological or biochemical nature of these basic processes is not discussed by Pavlov.

[3] Exactly why the bond should be formed Pavlov never explained further than to suggest that: "every strongly excited centre in some manner attracts to itself every other weaker excitation reaching the system simultaneously" (Pavlov, 1941, p. 47).

thus producing the conditioned response.

When the CS is presented there flow out from the corresponding cortical cells waves of excitation. The excitation first *irradiates* out from, and then *concentrates* back to, the point of origin. When, however, the CS is consistently presented without the US, rather than excitation, inhibition occurs at the cortical site. This "*internal inhibition*" elicited by the now inhibitory or negative CS also irradiates and concentrates. In the case both of excitation and inhibition, however, prior to irradiation, what Pavlov referred to as *induction* took place. In the inductive phase a process opposite in sign to that at the CS *point d'impact* arises in the surrounding cortex. Thus an inhibitory stimulus would give rise to concentrated inhibition at its cortical centre and diffuse excitation in the surrounding cortical cells. This would be an instance of positive induction. Exactly the reverse would occur in the case of negative induction, where an excitatory stimulus evoked inhibition in the cortical area surrounding the the excitatory centre. Induction was not an inevitable consequence of stimulation, however, but depended rather on the strength of the stimulus. Weak or very strong stimuli did not initially concentrate at their cortical site, but rather irradiated immediately.[1] Thus only stimuli between these two extremes gave rise to induction.

To this point the laws of the dynamics of cortical processes have already involved three bipolar variables: excitation and inhibition, irradiation and concentration, and positive and negative induction. To this Pavlov now added two functional properties of cortical cells, viz., cell *excitability* and cell *capability*. With the exception noted below, the

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[1] Pavlov, referring to this stated: "with a slight tension of either the excitatory or inhibitory process, under the action of the corresponding excitation, irradiation carries the processes from the original point; with a moderate tension it is concentrated in the original point; and with marked tension, there is again irradiation" (Pavlov, 1941, p. 87).

greater the excitability of the cell, the stronger the reaction to the stimulus. This was described by Pavlov as "the law of the relationship between the magnitude of the effect and the strength of stimulation." The "law of strength", as it became known, postulated that increasing the intensity of the CS increased the magnitude of the CR. A complication arises, however. Each cell has an upper limit of reactivity, a "top capability". When this limit is exceeded, inhibition rather than excitation occurs. Thus the law of strength now predicts an inverted U, rather than a monotonic, relationship between CS intensity and conditioning.

With regard to both cell excitability and capability there may be either temporary or chronic changes. The excitability of cortical cells of the feeding centre would, for example, increase with hunger and decrease with satiation. A cell's capability would decrease temporarily with fatigue, and chronically with old age. Undoubtedly the most important distinction that Pavlov made in regard to cell capability was between animals with innately "weak" or "strong" nervous systems. Animals with weak nervous systems had cortical cells that were in general of low capability, while a strong nervous system meant that the cells were of high capability. Thus the law of strength is not only curvilinear but it applies differentially to different "types" of animals.

Where now does disinhibition fit into this neurophysiological theory? First it should be pointed out that Pavlov eventually came to regard all external inhibitors as exerting their inhibitory effect through the process of negative induction. Thus a moderate strength extraneous stimulus which evoked the investigatory reflex would give rise to concentrated excitation at its cortical point d'impact, and surrounding inhibition that would suppress any simultaneously acting conditioned reflex. The excitatory traces left after the cessation of the extraneous stimulus, now having the intensity characteristics of a weak stimulus, would irradiate. This irradiation of excitation would cancel out the inhibition of an extinguished CS, thereby temporarily eliciting the conditioned reflex - in other words, producing disinhibition. Wendt, in an article highly critical of Pavlovian theory, has poetically described experimental extinction as the "spreading of the waters of inhibition to extinguish the fires of neural excitation" (Wendt, 1936, p. 259). In like fashion we could describe disinhibition as the spreading of the burning oil of excitation on the waters of inhibition.

But as Konorski (1948) has pointed out such a formulation of the disinhibition phenomenon presents problems. The extra stimuli which are "mild external inhibitors", and which Pavlov states are also the most effective disinhibitors, in the first case concentrate excitation and thereby produce inhibition by negative induction. In the second case they irradiate, and thereby produce disinhibition. Thus Pavlov ends up in the contradictory position of suggesting that the same external stimulus sometimes produces irradiation and sometimes concentration of excitation.

In order to resolve this contradiction Pavlov later proposed that the extraneous stimulus always irradiates. Thus external inhibition was said to be caused by the summating of excitation from the extraneous and conditioned stimuli. This summated excitation conveniently exceded the cell's capability and thus gave rise to top ("protective") inhibition. But this also must be incorrect, for such an interpretation would suggest that external inhibition occurred more readily in strong than in weak conditioned reflexes, whereas just the opposite is the case.

A further inconsistency in regard to the Pavlovian theory has been pointed out by Komorski (1948, pp. 44-45). Disinhibition is assumed to be caused by irradiating excitation, as is the familiar phenomenon of stimulus generalization. In one case, stimuli of entirely different sensory modalities from the inhibited CS produce disinhibition; in the case of generalization, dissimilar stimuli even of the same modality may fail to elicit the CR. Thus one must ask why in the generalization case the irradiation of excitation of the conditioned stimulus drops off so sharply as one moves away from the cortical point of origin, while apparently the reverse occurs in the case of disinhibition? Within the Pavlov framework there can be no reply. Stimulus generalization and disinhibition are simply not the same phenomenon, and to attribute a common underlying mechanism to them seems to draw an analogy where none exists.

Whatever seductive features the Pavlovian neurophysiological model might contain, it tended to become overweighted with unobservables. In the end all that Pavlov could observe was the stimulus presented and the organism's reaction.[1] But in the unobserved cortical area hypothetical excitation or inhibition might be irradiating or concentrating, dependent on the induced or conditioned properties of the cells and their constitutional or transient level of capability and excitability. If animals reacted differently to the same experimental program this could be attributed to their different types of nervous systems. Cortical dynamics became multi-determined, and not always internally consistent. Post-diction was nearly perfect, but prediction became difficult if not impossible.

It would be possible to point out further inconsistencies in the Pavlovian neurophysiological model, but for our purposes it is unnecessary.

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[1] With the advent of modern electro-physiological recording techniques, cortical activity may now be studied directly. Many Russian physiological psychologists are engaged in this type of work, but they still tend to conceptualize the problem in Pavlovian terms (see, e.g., Kline, 1961; Delafresnaye, 1954, 1961).

The important fact is that "knowledge" of the cortical dynamics involved in disinhibition is of little help in predicting what stimuli will act as effective disinhibitors under what circumstances. Pavlov, indeed, worked in a diametrically opposite direction. He was little concerned with such behavioral phenomena as disinhibition, *except* in so far as they demonstrated hypothetical underlying cortical events.

The Pavlovian model still dominates contemporary Russian research. Thus, though Russian investigators continue to work with disinhibition, little if any additional information concerning the phenomenon has been reported by them. The parameters of disinhibition do not concern contemporary Russian workers. They take disinhibition to be an obvious and established fact, and use a procedure which produces it only in order to make inferences about the basic nature of cortical dynamics. Thus, e.g., a recent Russian study (Bosyi, 1958) reports the use of "different extraneous stimuli" as disinhibitors, in order to study whether cortical inhibition is present at various stages of conditioned inhibition training. The nature or duration of the extraneous stimuli is simply not reported.

The current Russian studies, committed as they are to the Pavlovian inferential nervous processes, fail to describe experimental procedures, and are lacking in such elementary experimental controls as counterbalancing the order of presentation of stimuli. "Specimen protocols" are presented, and most studies make no use whatever of statistical analysis. There is thus little to be gained by attempting to review the Russian research available in translation. We turn instead to North American research on the problem of disinhibition.

American Research on Disinhibition

The Pavlovian conditioned reflex came like a gift from heaven to the American behaviorist school of psychology that was marshalling recruits in the second decade of this century. Here was a way of explaining associative learning without reference to consciousness. John B. Watson, the behaviorists' chief propagandist, enthusiastically clasped the conditioned reflex to his bosom (1916), eventually making it his central theoretical concept (1925). The general enthusiasm for Pavlov's conditioned reflex, however, was matched by a disaffection for Pavlov's speculative "cortical physiology", and in particular for his idea of inhibition. The American scene was geared to the Thorndikian (1898) excitatory stimulus-response bond type of conceptualization. As one group of authors (Diamond,et. al., 1963) has put it, American psychology was extraverted. There was no place for a concept such as inhibition. Thus the concept of inhibition was speedily dismissed, though Pavlov's experimental procedures were eagerly adopted.

Wendt (1936), a representative spokesman for his time, proposed that the events Pavlov subsumed under the heading of inhibition might more accurately and parsimoniously be viewed simply as instances of incompatible responses. Taking Sherrington's (1906) "Principle of Reciprocal Innervation" as his guide, Wendt concluded that "the results of both Pavlov's experiments and our experiments are instances of competition between reaction systems" (Wendt, 1936, p. 275).

B. F. Skinner, writing at an early date in his career, (1936) also took the concept of inhibition to task. He pointed out that inhibition subsumes a number of diverse operations (such as lowering drive level, eliciting incompatible responses, fatigue, etc.) under a single term, the only common property of which is a negative effect on a specified response. But Skinner suggested that "the use of the single property of the negativity of the change does not lead to the establishment of a significant class of data .... [and] it must not be assumed that other properties possessed by one case are common to the class" (Skinner, 1936, p. 128). Further, according to Skinner there logically should exist the converse to the inhibition concept, covering all instances of an increment in response; but this type of formulation is rarely suggested.

While attacking the concept of inhibition, Skinner acknowledged that the purported phenomenon of disinhibition represented a powerful support for the concept. This is in contrast to Wendt, who largely ignored disinhibition. To Wendt, for whom inhibition did not exist, it seems to have been obvious that there could be no such thing as disinhibition. Skinner, however, did attempt to experimentally disprove disinhibition, or at least to reinterpret it in terms of stimulus facilitation of a response. We shall describe this experiment later. The striking fact is that both Skinner and Wendt failed to deal with the most important fact to which Pavlov drew attention. The <u>same</u> stimulus will both inhibit and disinhibit (produce a decrement or increment in the CR), dependent on the stage of training. Thus, both inhibition <u>and</u> disinhibition simply cannot be explained in terms of competing responses elicited by the extraneous stimulus, which either facilitate or impede a particular conditioned response.

While the concept of disinhibition played no important part in American behavior theory, a number of investigators - particularly during the first flush of enthusiasm for Pavlovian experimental procedures - did perform studies attempting to demonstrate the reality of the empirical phenomenon. The American experiments fall naturally into two categories - those which employ a classical Pavlovian (respondent) conditioning procedure, and those in which an instrumental (operant) response is involved. The first type of conditioning is derived from Pavlov (1927), the second, from Thorndike (1898, 1911). We shall ignore the argument as to whether two fundamentally different types of conditioning exist. We may simply make the division in terms of the operational paradigm involved in the two procedures. The classical conditioning procedure involves pairing CS and US so that the signal (CS) comes to elicit a response previously associated with the reinforcer (US). The instrumental technique involves following some arbritrary response with a reinforcer. The response then comes to be emitted more frequently. For our purposes this division, crude as it may be, is adequate. We shall first review the classical and then the instrumental studies of disinhibition.

Classical Conditioning and Disinhibition Winsor (1929), in a review article concerning "inhibition and learning", makes one of the earliest references in the American literature to disinhibition. The formulation of the problem and the experimental technique closely follow Pavlov. The experimental procedure consisted of measuring the salivary secretion of an adult male subject who "had been eating his luncheon under the conditions of the experiment for a protracted period". When food was repeatedly placed on the table before the subject, but not eaten, the magnitude of the salivary response progressively decreased until eventually it reached a level comparable to that observed in the absence of food (control interval). The author stresses the fact that the response decrement observed with such an "extinction" procedure should be viewed in terms of learned or acquired inhibition. To support his interpretation Winsor reported data to show that, during extinction, if a metronome is sounded, then an increase in the salivary response occurs. The salivary response did not occur during the action of the metronome, but only after its termination.

A more thorough investigation was carried out by Switzer (1933), who employed human subjects and the conditioned galvanic skin response (GSR). Switzer first conditioned the GSR by pairing a faint light (CS) with "tetanizing" faradic shock (US) to the subject's finger. When a conditioned response had been fully developed to the light, extinction training began. The extinction training was continued until the CS failed to elicit the conditioned GSR on two or three consecutive trials. At this stage, a "raucous" buzzer was sounded for .3 seconds, and 38.5 seconds later the subject was tested with the light CS. There was a marked GSR (about 2/3 of the magnitude of the original conditioned response) elicited by the extinguished CS. The author completely ignores, however, the fact that the GSR reaction to the "extraneous" buzzer was in <u>every</u> case larger than the later "disinhibition" reaction. Further, if one computes (from the raw data that the author presents) the correlation between the magnitude of the responses elicited by the buzzer and by the CS a significant relationship is found (rho= .85, $\underline{p} < .01$). In Pavlov's experiments, the disinhibiting stimuli were <u>neutral</u> with respect to salivation, the response to be disinhibited.[1] There was in any event some reinstatement of a <u>conditioned</u> response in Switzer's experiment, as a control group given previous shock and light presentations, but not in temporal contiguity, showed essentially no response to the light presented after the buzzer. Thus Switzer concluded that his

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[1] Presumably, the long time interval between presentation of the extraneous stimulus and the test for disinhibition was necessitated in part by the need to wait for the response to the extra stimulus to subside, before the test.

"data appear to corroborate Pavlov's conclusion that disinhibition is a genuine phenomenon" (Switzer, 1933, p. 97).

Wenger (1936) reported an experiment also involving GSR conditioning in human subjects. Here the same stimulus (a tactile vibrator) was said to produce both external inhibition <u>and</u> disinhibition, depending on the stage of development of the CR.

The initial training consisted of paired presentations of a red light (CS) and an unavoidable shock (US) to the subject's foot. The CS-US interval was .94 seconds. After 25 reinforced acquisition trials, testing for external inhibition was carried out. The procedure simply consisted of activating the tactile vibrator, which was attached to the subject's hand throughout all phases of the experiment, 20 seconds prior to presentation of the CS. The reaction to the CS on this "externally inhibited" trial was then compared to the immediately preceding and following "undisturbed" tests with the CS. The CR was of smaller magnitude on the test trial preceded by the vibrator.

Following this phase of the experiment, massed unreinforced presentations of the CS were given until "the response approached its primary amplitude". Then, once again, the vibrator was presented, and its effect on the now extinguished CR noted. The external stimulus now resulted in an increase in the magnitude of the GSR over that displayed on the previous trial.

The Wenger study included an attempt to assess the effect of intensity of the disinhibiting stimulus. There were two groups treated alike in all respects except that in one case the action of the vibrator was more intense. The author summarized his results by stating that "external inhibition and disinhibition were produced by duplicate stimuli" and that there was "some indication that the greater of the two intensities of tactile vibration used produced a greater mean amount of external inhibition and disinhibition" (Wenger, 1936, p. 456).[1] The Wenger data indicate that all sign of external inhibition had disappeared in both experimental groups by the second test, although this was not true for the second disinhibition test. The vibrator, activated 20 seconds before the CS, presumably elicited a GSR itself.

Hovland (1937), in one of a series of experiments studying the generalization of conditioned galvanic skin responses, reported an instance of "disinhibition". The procedure involved half the subjects being trained with a low intensity CS (40 db. tone), and the other half, with a high intensity CS (80 db.). The US was electric shock. After 16 paired CS-US acquisition trials each subject was given four unreinforced test trials with each of the two tones. The magnitude of the CR declined during the test trials, with the decrease for the generalized response being far more marked than for the conditioned response. Hovland suggested that the conspicuous decrement in the generalized response was due to its rapid extinction. Thus he argued that an extraneous stimulus introduced at this time should, as a Pavlovian disinhibitor, result in a reinstatement of the generalized response. To demonstrate this, following the fourth test trial the lights in the experimental room were suddenly extinguished, and shortly thereafter a further test

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[1] It is possible to calculate from the raw data given by Wenger the statistical significance of the different comparisons. On the first test for external inhibition both levels of vibrator intensity can be shown independently to suppress the CR, but this is not true for either condition on the second test. The difference between the two groups is not significant. The less intense vibrator does not produce a significant increment in CR on either the first or second disinhibition test. The more intense vibrator produces significant disinhibition on both trials.

with both tones was carried out. According to the author the results were "completely in accord with the theoretical prediction. At the end of the testing before disinhibition the ratio of the conditioned to the generalized responses were 1.35, but after disinhibition it declined to 1.01, indicating marked increase in the amount of generalization" (Hovland, 1937, p. 56). This so-called "disinhibition" demonstration, however, may simply be attributable to an after-effect of the unconditioned GSR reaction to light change.

The last classical conditioning experiment on disinhibition that appears in the American literature is by Razran (1939), who studied the salivary conditioned response in human subjects under the guise of investigating "the effects of eye-fatigue upon digestion". The CS was a 2-minute flashing light, the US was the presentation of food in the form of tea-sandwiches, pretzels, lollipops, etc. The procedure was as follows. Initially all subjects were given 40 conditioning trials consisting of the flashing light presented during each 2-minute eating period. The salivary response was measured by weighing dental cotton rolls which had been placed under the subject's tongue. Since as the author points out: "cotton-in-the-mouth is by no means a totally inactive stimulus" (Razran, 1939, p. 648) control samples of salivation were alternated with experimental samples. Thus conditioned salivation was always indexed as a net difference between experimental and control measurements.

Following the 40 acquisition trials extinction was begun. The conditioned light was flashed during 24 one-minute periods without reinforcement. A buzzer was sounded during even-numbered trials to test for the effects of extraneous stimulation on the extinguishing conditioned response. An individual subject, however, was tested at only 2 points in the extinction program. Thus for each of the 12 test trials there were four subjects, one or two of which would have had a previous exposure to the extraneous stimulus.

Razran summarizes his results as follows: "The buzzer was shown to exert a double effect: while it suppressed the existing conditioned salivation, it restored the loss of conditioned salivation resulting from extinction" (Razran, 1939, p. 651). This conclusion derived from a procedure whereby Razran measured the distraction trial CR as a percentage of the preceding "control" trial CR, and the control trial in turn as a percentage of the final acquisition trial CR. The correlation between the degree of extinction (indexed by the comparison of the control and final acquisition trials) and the incremental effect of distraction was .91. That is, disinhibition was a direct function of the degree of extinction ("inhibition"). The data certainly suggest such a relationship, but we have to point out that the correlation coefficient which Razran employed is seriously bloated in view of the fact that a common factor (the control CR) entered into both variables being correlated. The subjects were not experimentally naive and the basic procedure pooled between- and within-subject variability.

The Razran measurement procedure raises an even more fundamental problem. Razran chose to calculate the effect of the disinhibiting stimulus as a percentage of the magnitude of the conditioned salivation on the preceding trial. With such a measure the decrement in the CR at the start of testing (i.e., the external inhibition) is relatively small (54%), while the increment after 23 extinction trials (i.e., the disinhibition) looms very large (320%). But for the same two stages of training the absolute changes in the CR are minus 95 and plus 158. Since the basic response tendency is inevitably of smaller magnitude as extinction progresses, even a small absolute increment will be large, in percentage terms. However, although the relative magnitudes of the measured changes vary considerably with measure employed, the conclusions as to the basic inhibition-disinhibition effects of the stimulus are not, of course, altered. This measurement problem will not seriously affect parametric studies of disinhibition in which the baseline level of response is fairly constant over time, but it will obviously be of some concern in studies where (as in Razran's) this is deliberately not the case.

The classical conditioning studies of disinhibition that we have reviewed present a number of problems. The American studies have all employed human subjects, and all but one utilized the GSR as the response. The grave problem that this involves is that almost any "extraneous" stimulus will, in and of itself, produce a GSR. In Switzer's experiment, the extra stimulus (a "raucous buzzer") actually produced a response greater in all subjects than was the "disinhibited" CR. Thus, when the extinguished CS is presented at some time interval after the extraneous stimulus, it is not at all clear whether a response elicited by the CS is to be attributed to a persisting response sensitization, or to "disinhibition". Though Wenger did demonstrate that his extra stimulus decreased GSR amplitude during acquisition, the GSR in these early studies was measured as a simple deviation from a changing basal resistance level.[1] It may well be that, during acquisition, the baseline resistance is already at an extreme value, so that a significant change produced by the extra stimulus would make it difficult to show a GSR in the immediately following period. The early experiments also tended to

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[1] For a variety of statistical and physiological reasons such an index has since been demonstrated to be inappropriate (see, e.g., Haggard, 1945; Lacey, 1956; Dykman, Reese, Galbrecht, and Thomasson, 1959).

lack necessary control groups. Particularly, before attributing a change in response magnitude over time to the use of an extraneous stimulus, it is necessary to show that subjects given the same sequence of experiences, but *without* the extraneous stimulus, do *not* show such a change. This control is absent in the early studies. Thus it is conceivable that many of the observations of disinhibition reported in these studies may simply be instances of response sensitization, spontaneous recovery, or still other factors.

We can conclude that although the evidence suggests that the disinhibition phenomenon is genuine, the classical conditioning procedure as used in America, and GSR conditioning in particular, did not lend themselves readily to detailed quantitative investigation of the phenomenon. But then, the experiments were not so much concerned with this as with merely demonstrating that the strange empirical phenomenon described by Pavlov did occur.

Operant Conditioning and Disinhibition The earliest American study of disinhibition employing an operant conditioning technique was reported by Hunter (1935). The subjects, laboratory rats, were trained to make a movement avoidance response. Two stimuli were employed, a "brilliant" light produced by two 100 watt bulbs, and a buzzer. Two experiments counterbalanced the use of the two stimuli as CS and disinhibitor respectively. The training consisted of presenting the CS, followed two seconds later by the US, which was an electric shock delivered to the rat via a grid floor. A minimum movement of one body length in the CS-US interval avoided the shock on that trial. After training to a criterion of 10 consecutive avoidance responses, the CS was presented without the US until the subject failed to respond for 10 consecutive trials. At this point of extinction training the disinhibitor was briefly presented, and one minute later the first of three test trials with the CS was carried out.

When the light served as the CS and the buzzer as the disinhibitor, the four rats in the experimental group gave a total of five responses in twelve test trials. When the stimuli were reversed in function, the three experimental subjects made six responses in their nine test trials. There were no control subjects that would allow estimates of the spontaneous recovery of the avoidance response that might have occurred in the absence of extraneous stimulation, but this was presumably a minor factor. Hunter viewed the importance of his experiments in terms of an attempt "to verify Pavlov's finding with the salivary reflex, by experiments on such a laboratory animal as the white rat using overt bodily activity of the locomotor type" (Hunter, 1935, p. 77). Presuming that the probability of a response without prior presentation of the extraneous stimulus was close to zero, Hunter's study can be viewed as the first demonstration of disinhibition in an operant response setting.[1] However, there is no report of the effect of the extraneous stimulus during acquisition.

In a later report of avoidance conditioning with human subjects, Hunter (1938) stressed the fact that voluntary responses were disinhibited.[2]

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[1] At the time that Hunter was writing the operant -respondent dichotomy had not yet been seriously proposed. While the avoidance response may be viewed as an operant, current interpretations still emphasize the importance of classical conditioning in the avoidance training procedure. Thus the demonstration is far from the perfect example of disinhibition of an operant response. It does represent, however, an early example of such a possibility.

[2] Mowrer (1947) and others have at times suggested that the voluntary-involuntary division roughly parallels the operant-respondent categories.

Hunter sought to prove that what other people might refer to as "fiats of the will" were in fact instances of conditioning.

The basic procedure in the experiment consisted of presenting a signal (CS) followed approximately one second later by an electric shock (US) to the subject's finger. If the subject made a finger withdrawal response in the CS-US interval, then shock was avoided on that trial. After the response had been acquired to a criterion of five consecutive avoidances, reinforcement was discontinued. Extinction training continued until the subject failed to respond on three to five successive occasions. When a buzzer or tactile vibrator was now presented 7-15 seconds prior to the CS, the extinguished finger flexion re-occurred. Disinhibition also was demonstrated when the training program involved "conditioned inhibition" rather than experimental extinction. In the case of conditioned inhibition a discrimination had to be made, for example, between two lights which were never reinforced when presented together, and one light which was always reinforced in isolation. The single stimulus eventually came to reliably elicit the finger withdrawal response, while the stimulus complex did not. When the disinhibitor (vibrator or buzzer) was now presented in conjunction with the non-reinforced stimulus complex, a conditioned response occurred. It is interesting to note, however, that both the buzzer and vibrator produced _of themselves_ a "startle" finger flexion. Thus, as with the earlier GSR experiments, these demonstrations appear more suggestive than conclusive.

The maze, and its limiting case, the straight runway, have also been used to study the disinhibition phenomenon. The first such studies were by Reynolds (1936, 1939), who employed the concept of disinhibition in an attempt to explain a sudden behavioral shift earlier observed by

Higginson (1924) and Valentine (1928). The experimental problem to which Higginson and Valentine drew attention was the following. Rats could be trained to first run past the food compartment of a maze, and then to return to it to eat. This was accomplished by having the food compartment door locked when the rat first encountered it, and then open on the return run. When this "long-route" maze running pattern was firmly established, the door to the food compartment was left open before the initial test trial. With the introduction of the open door, animals quickly shifted from the longer to the shorter goal path. This behavior may seem only obviously sensible, but it posed real problems to a theory which took quite literally the notion that reinforcement "stamps in" habits. Reynolds, following Hull's (1934) suggestion, interpreted the shift as due to disinhibition of the preferred short route which, although initially higher in the "habit family hierarchy", had presumably been inhibited during acquisition training. Reynolds sought to substantiate such an interpretation by employing an electric shock rather than the open door as an "extraneous disinhibiting stimulus".

Eight animals were trained to take the long maze path to a criterion of 25 consecutive correct runs, and then disinhibition testing was introduced. Eight disinhibition test sequences, each followed by retraining to the long route, were administered. Reynolds felt that the correctness of the disinhibition interpretation was substantiated by the fact that in 51 cases out of a possible 64 the short route was taken after the first application of the shock. The results are, however, ambiguous. Reynolds reports that after the initial application of the shock a maximum of two additional test trials were given if subjects did not take the shorter route. Then subjects "were given five more of the usual [i.e., long] runs after which they would be shocked on the next following run, and so on, until disinhibition did occur" (underlining mine) (Reynolds, 1936, p. 192). Thus disinhibition had to be demonstrated eventually.

Two follow-up experiments (Reynolds, 1939) attempted to improve the experimental procedure. The rats were either shocked outside the maze, or a black curtain over the goal-box door was employed as the disinhibitor. These changes in procedure did reduce the mean number of retraining trials necessary for eight disinhibition tests, but the modifications also apparently increased the number of test trials necessary to "demonstrate" disinhibition. Reynolds points out that some animals did not "disinhibit" on the initial test trial, but rather on a subsequent trial. The lack of appropriate control comparisons does not justify treating these post-test reinstatements of the inhibited CR as anything other than random occurrences, or even spontaneous recovery.

The Reynold's "disinhibition demonstrations" must be viewed as little more than impressive testimonial to an investigator's adherence to conditioning hypotheses. The goal of the experiments was not to investigate the disinhibition phenomenon, but to interpret a behavioral fact in terms of the concept of disinhibition.

Gagné (1941) on the other hand sought to study the disinhibition phenomenon itself, investigating the effects of extraneous stimulation during both the acquisition and extinction of an operant response. The procedure involved training rats to traverse a 3-foot elevated runway for a food reward. Two extraneous stimuli were employed - a "loud buzzer" and "a light scratching at the starting box of the apparatus". Each buzzer presentation was of four seconds duration and terminated two seconds prior to the start of the trial. The scratch was continued until the rat turned around and looked ("orienting reflex") toward the rear of the box, where the scratching experimenter stood. To the degree that it was possible, the duration of the scratch approximated that of the buzzer.

The subjects were given 15 reinforced acquisition trials followed by 8 non-reinforced extinction trials. There were four experimental groups of subjects, two of which were tested with each of the two stimuli. The stimuli were presented once in acquisition, prior to either the first or fourth trial, and in extinction, always prior to the fifth trial. A control group received acquisition and extinction training without any exposure to the extraneous stimuli. The measure of the response was the time the subject took to pass a mark four inches in front of the start-box door after it had been opened to start the trial. This presumably is largely a measure of response latency.

Gagné found that there were no differences in acquisition latencies between the controls and the two groups of subjects that received the extraneous stimuli prior to the first acquisition trial. The author interpreted this as a demonstration of the fact that neither stimulus had an inherent facilitating or inhibiting effect on the running response. The first trial latency in a runway, however, is so variable that it would be difficult to demonstrate any effect. On the other hand, both the buzzer and the scratch presented prior to the fourth acquisition trial significantly increased the response latency (i.e., "inhibited") of the experimental subjects in comparison to the controls. The groups were not different from the controls on the next trial - in other words the inhibitory effect was specific to the trial on which the extra stimulus was presented.

In extinction the presentation of the scratch prior to the fifth trial resulted in a significant <u>decrease</u> in latency (disinhibition) on that trial only. The buzzer, on the other hand, produced a significant <u>increase</u> in latency of the fifth extinction trial, and a significant <u>decrease</u> on the following sixth trial. All comparisons were made to the control subjects' behavior on the corresponding trial.

The effect of the scratch on the operant running response appears to parallel Pavlov's observations with salivary conditioning. During acquisition, when response latency was decreasing, this stimulus acted as an external inhibitor.

During extinction, when response latency was increasing, the scratch had just the opposite effect. That is, it decreased the latency or in other words acted as a disinhibitor. Any possibility of interpreting both these facts as due to the scratch being either inhibiting or facilitating, in and of itself, seems to be ruled out. At different stages of training exactly opposite effects were observed.

The interpretation of the results with the buzzer is not so straightforward. In both acquisition and extinction the buzzer inhibited running on the first test trial. Thus Gagné suggested that the buzzer may be called "an 'emotion-producing' stimulus", in the Skinnerian (1938) sense that it depresses the rate of responding. There appears to be some plausibility to this interpretation in view of the fact that the buzzer was "attached to the under side of the starting platform in such a manner that the clapper struck the platform" (Gagné, 1941, p. 106). Peculiarly, however, during extinction there was a marked <u>decrease</u> in latency on the second trial following presentation of the buzzer. To interpret this unexpected finding as evidence of the buzzer producing disinhibition would not conflict with Pavlov's repeated assertion that the disinhibitory effect may occur only after presentation of the extraneous stimulus; but such an interpretation would smack too much of capitalizing on a chance fluctuation in the data.

The important point of the Gagnè study is that both inhibition and disinhibition could be demonstrated in the operant setting. In view of the different effects of the buzzer and scratch one might suggest that the decremental-incremental effects of an extraneous stimulus are more reliable when, as was the case with the scratch, an "orienting reflex" was consistently elicited.

Winnick and Hunt (1951), interested in the possible dynamogenic effects of extraneous stimuli during acquisition and extinction, essentially replicated the Gagné study. These investigators felt that the same extra stimulus (buzzer) might produce different effects at different stages of both acquisition and extinction.

Employing the buzzer as the extraneous stimulus, the Winnick and Hunt investigation duplicated the Gagné experiment in almost all technical and procedural details. The point of difference between the two experiments was the stage of training at which the buzzer was presented. There were four experimental groups for each of which one acquisition and one extinction trial was preceded by presentation of the extraneous stimulus. The acquisition (A) and extinction (E) trials on which the buzzer was presented were, for the four groups: A-4, E-2; A-8, E-4; A-12, E-5; and A-14, E-6. The fifth group was comprised of control subjects which never experienced the buzzer on any trial.

During acquisition, all four experimental groups showed an increase in latency on the acquisition trial before which the buzzer was presented.[1] The magnitude of the response decrement (amount of inhibition) produced by the buzzer decreased with continued acquisition training. This was so regardless of whether the decrement was measured in absolute or in proportional terms, and whether it was viewed with respect to the behavior of the subject on its own preceding acquisition trial, or the behavior of control subjects on the <u>same</u> acquisition trial. Thus the buzzer inhibited responding more radically early in acquisition, the effect decreasing as the running response became stronger with successive reinforcements.

The results of extinction testing with the buzzer, like Gagné's findings, were somewhat ambiguous. For subjects tested on the second extinction trial, the buzzer significantly decreased the latency of response. However, as the authors comment, "When the buzzer was introduced before the fourth, fifth, or sixth extinction trial, the effect was small and equivocal in direction" (Winnick and Hunt, 1951, p. 214). Thus the buzzer in this experiment acted as a disinhibitor only <u>early</u> in extinction. This finding might be interpreted in many ways. For example, the temporal interval between the first presentation of the buzzer (during acquisition) and its subsequent presentations during extinction happened to be longest for that experimental group tested on the second extinction trial. Possibly a persisting emotional after-effect of the first buzzer presentation contaminates these data.

We may summarize the Winnick and Hunt experiment by first pointing out that the same stimulus did inhibit responding during acquisition and

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[1] It should be pointed out that both the acquisition and extinction curves for the control subjects in the Winnick and Hunt study were very similar to the corresponding control data of Gagné.

disinhibit it during at least one point of extinction. Thus the study is in basic agreement with Gagné's earlier demonstration.

These experiments seem to have demonstrated existence of the Pavlovian phenomenon in an operant setting. In any event, interest in the problem seems to have subsided for some ten years until the most recent American work on disinhibition by Yamaguchi and Ladioray (1962). They reported two studies investigating the effects of extraneous stimulation during the acquisition and extinction of a runway response. In the first experiment rats were trained to traverse an enclosed 18-inch L-shaped runway for a food reward. The response measure was the time from opening of the start box until the rat traversed the first 15 inches of the runway. On test trials a 500 cps tone (approximately 100 db.) was presented during the measured run. That is, the tone commenced with the opening of the start box door and terminated when the animal passed the fifteen inch runway mark. There were three experimental groups, each group tested with the tone only once. All subjects were given 60 acquisition training trials. On one of the three final trials (58, 59, or 60) the "acquisition group" was presented with the extraneous stimulus. After the 60 acquisition trials, only the two "extinction groups" were given massed extinction trials. One group was tested after the tenth, and the other after the twentieth extinction trial. With no further presentation of the tone, running speed was observed for the three trials following tone presentation.

The findings were that the tone significantly decreased running speed in acquisition, and increased running speed in extinction. The groups given differential degrees of extinction training did not differ in terms of the magnitude of the response increment.[1] Finally, the increment during extinction on the trial following the test presentation was significantly greater than the increment on the test trial itself. In other words, although animals ran faster on the trial with the tone present, they ran faster still on the subsequent trial. On the second post-test trial, scores were similar to the test trial, and on the third post-test trial similar to the pre-test trial.

In the second study Yamaguchi and Ladioray studied the effect of different intensities of the 500 cps tone on acquisition and extinction. In an attempt to equalize the acoustical characteristics of the stimulus at different apparatus locations, a circular maze was employed, with the loud-speaker suspended in the centre. There were three tone intensities employed, measuring respectively; 45, 58, and 85 db. Three experimental groups of subjects were tested after 115 acquisition trials, one group with each tone intensity. A fourth group which did not receive the tone acted as controls. Following 120 acquisition trials all groups received 40 extinction trials. Three of the groups were tested (each with its acquisition tone intensity) on the 36th extinction trial. The fourth group was the control.

The results may be briefly summarized as follows. Only the 85 db. noise produced significant external inhibition (decrease in running speed) during acquisition, and only the 45 db. noise produced significant disin-

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[1] Because no appropriate control groups were run, all the foregoing conclusions were based on the comparison of the response on the test trial and on succeeding trials to the response on the trial immediately preceding the test. This suggests that the magnitude of the changes which were reported was likely underestimated. In acquisition one would expect response time to be decreasing, and in extinction, increasing over trials. This is just the opposite of the changes observed in response to the test stimulus.

hibition (increase in running speed) during extinction.[1] There were no consistent post-test trial changes in running speed, such as had been observed in the first study, and in the experiments by Gagné and by Winnick and Hunt. The results of the second study by Yamaguchi and Ladioray are not really consistent with those of the first. The extrapolation of the intensity function suggested for disinhibiting stimuli in the second study would predict that a 100 db. tone would be an ineffective disinhibitor. But in fact, in the first study, which differed in procedural details, 100 db., was found to be an effective disinhibitor. Commenting on the two studies, Yamaguchi and Ladioray suggest: "that the disinhibition effect is a fact but not an easily reproducible one" (Yamaguchi and Ladioray, 1962, p. 576).

Looking at all three runway investigations (Gagné, Winnick and Hunt, Yamaguchi and Ladioray), what are we to conclude? First, they provide evidence that phenomena similar to Pavlovian external inhibition and disinhibition may be demonstrated with an operant response. The runway setting, however, has some inherent problems, and these possibly contributed to the lack of consistency in the findings.

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[1] All these comparisons could, in distinction to the first study, be made by comparing the response speeds for experimental and control subjects at the <u>same</u> stage of training.

First, runway speed itself tends to be a somewhat variable measure, easily influenced by uncontrolled events in the experimental environment. Thus, for example, in the second study by Yamaguchi and Ladioray the mean running speed for the control subjects was considerably lower on the 38th and considerably higher on the 39th extinction trial than it was on the 35th trial. This kind of trial-to-trial fluctuation, in the absence of any experimental treatment, makes it especially hazardous to attribute significance to a difference observed between behavior on a test trial and behavior on some subsequent trial, not specified in advance. The necessity of discrete trials in runway conditioning poses another problem, for it means that animals must be moved from the goal box to the start box on each trial. This disruption undoubtedly would influence to some extent the effect of the experimentally controlled extraneous stimulation. Thus it appears that runways are far from the ideal setting in which to study the inhibiting and disinhibiting properties of extraneous stimuli.

The bar-pressing operant procedure developed by Skinner, on the other hand, does not suffer from these disadvantages. We shall finally review the three studies that have investigated disinhibition employing this type of operant response.

Skinner in 1936 reported what he considered to be a "failure to obtain disinhibition". The situation involved rats bar pressing for food in what has commonly become known as a "Skinner box". The dependent variable in such an experiment is the rate of freely emitted bar presses. Initially the animals were trained to bar press under a periodic food reinforcement schedule, and then extinction was carried out by discontinuing all further reinforcements. During extinction, a "disinhibiting" stimulus was introduced, and its effect on the response rate noted. There were 18 rats in the experiment, with two records taken from six of the subjects, so that a total of 24 observations were available. The different "disinhibiting" stimuli employed by Skinner, with the number of occurrences of each given in brackets, were: onset of a 3-c.p. light (7), the click of the empty food delivery mechanism (2), pricking the tail of the subject with a needle (3), and removing the subject from the apparatus and tossing it "into the air in such a way that vigorous righting reflexes were evoked" (12).

There were no numerical analyses of changes in response rate, but Skinner reported that, with the exception of the light, there was no substantial evidence for any increase in response rate following presentation of the extraneous (?) stimulation. However, <u>all</u> instances of any increase in responding were interpreted by Skinner as temporary facilitations of an otherwise "out-of-step" extinction curve. The argument takes the following form. When extinction behavior is graphically depicted in a "cumulative record", as cumulated responses plotted against elapsed time, one observes, according to Skinner, a smooth negatively accelerated curve. This extinction curve describes an "envelope". In all instances where the extraneous stimuli resulted in a temporary <u>increase</u> in response rate (e.g., for four out of the seven tests with the light), Skinner pointed out that the extinction curve "was for some reason below its envelope and the effect of the facilitation [i.e., the extraneous stimulus] was simply to bring it to its <u>proper</u> position" (underlining mine) (Skinner, 1936, p. 133). This interpretation is infallible, because if there <u>is</u> a temporary increase in response rate, then for Skinner the curve preceding this increase will <u>always</u> be below the hypothetical envelope. The envelope is drawn simply by joining the point of maximum response rate late in extinction to the early portion of the cumulative curve! The precise shape of the "envelope" differs for individual rats, and thus no empirical control is possible.

Skinner suggested that the response increments which he did observe, as well as those reported by Pavlov, may simply be due to a facilitative effect of the extraneous stimulus on responding. But Pavlov stressed the fact that the <u>same</u> stimulus will have <u>opposite</u> effects at different stages of training. Thus, to support his contention, Skinner should have demonstrated that the light had a facilitative effect during <u>acquisition</u> of the bar-pressing response. This he conspicuously failed to do.

The very loose experimental control involved in the delivery of such "stimuli" as tossing the animal in the air and pricking its tail with a pin, make the stimuli seem inappropriate for a critical "disproof" of disinhibition. From the vantage point of 27 years, it is amusing to see the psychologist who, more than any other, has made it possible to maintain strict and automated environmental control over the experimental space, employ such "impure stimuli". Further the time of testing in extinction (and thus the degree of extinction attained) was unspecified. Possibly disinhibition only occurs when rate of operant responding has been reduced to a very low level.[1]

In summary, we object to Skinner's "failure to show disinhibition" both on the grounds that inappropriate stimuli were employed, and that no criterion for extinction was adopted. Even then, however, there occurred increases in response rate in some animals. Skinner's interpretation of these increases as due to facilitation of an "accidentally" depressed

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[1] In the three sample extinction curves that Skinner presents, the test for disinhibition took place after 45-60 minutes of extinction. The greatest increase in rate occurred in the 60 minute example, when baseline response rate was lower than in the other two curves.

extinction curve is untenable. There is certainly no disproof of the phenomenon simply by re-naming it. In any event, the next experiment to be described (Horns and Heron, 1940) was basically a replication of Skinner's attempt, but employing a more rigorous extinction criterion and more appropriate disinhibiting stimuli. The results in this case clearly indicated the occurrence of the disinhibition phenomenon.

Horns and Heron (1940) first trained rats to bar press for food on a four minute fixed interval reinforcement schedule. This training was continued for ten 1-hour daily sessions. There were then five days of extinction (where no reinforcement was given), and finally five days of disinhibition testing (with the extinction schedule remaining in effect.) Two disinhibition tests were administered during each 1-hour testing session. The three disinhibiting stimuli employed were: a buzzer, a light, and a presumably very weak electric shock (milliamperage unspecified). Each buzzer and light presentation was of 30 seconds duration. The first shock presentation was 30 seconds, the second was 150 seconds.

The authors compared the total number of responses on a disinhibition test day to the total number of responses on the fifth (i.e., pre-test) extinction day. They found that the electric shock produced nearly a 96% increment in responding. The other two stimuli (buzzer and light) caused only negligible changes in response rate when the _whole_ day's session was considered. This represents, however, an exceedingly stringent criterion for disinhibition. As an extinction program was maintained throughout the test sessions, the probability of responding would be expected to continually decline. Thus, a _temporary_ increment in rate brought about by the relatively short duration extraneous stimuli might well fail to bring the day's _total_ responses up to the number that occurred on the fifth extinction day. This was apparently the case, for the authors report that on the first presentation of the buzzer "the response rate [was] raised from 11.12 immediately before, to 22.08 immediately after, an increase of 10.96 [but that] ... the total mean response for the trial [i.e., for the 1-hour session] was 28.52, actually slightly lower than the score obtained in the final extinction trial" (Horns and Heron, 1940, p. 99). In regard to the light stimulus, the authors simply report that the average score for six animals (presumably the best "disinhibitors") increased from 6.64 to 45.32 with the first introduction of the light stimulus, which was presented on the fifth test day.

The authors minimize the effect of the extraneous stimuli on response rate by (1) considering the whole day's session rather than just that portion temporally contiguous with the stimuli, and (2) by comparing this response rate to a rate in existence one to five days earlier in extinction. This extremely conservative procedure may well have been influenced by Skinner's earlier pyrotechnics, when the "envelope" was used to explain away temporary changes in response rate. Temporary changes can always be evaluated for significance by contrasting the behavior of an experimental group to that of a control group not presented with the disinhibiting stimulus, but Horns and Heron employed no such group. Further, the *order* in which the various disinhibiting stimuli were tested was not counterbalanced.

This experiment thus does not allow clear-cut conclusions regarding all the stimuli employed. It appears from the individual and mean cumulative records that are given, however, that all three stimuli produced at least a transient increment in response rate. Because the effect of the shock apparently persisted long after its termination, this was the only extraneous stimulus that unequivocally increased the whole day's response output.

We must at least agree with the present authors' conclusion that "disinhibition can be produced in the lever-pushing situation if the proper stimulus is given" (Horns and Heron, 1940, p. 102). The analogy to the Pavlovian phenomenon would of course have been strengthened had the authors demonstrated an inhibiting effect of the same stimuli during acquisition of bar pressing.

The last study to be reported was by Brimer and Kamin (1963) who, in a series of three experiments investigated the effects of prior experience with "unsignalled" electric shock on later acquisition of a conditioned emotional response (CER). The study had been designed as an attempt to investigate "adaptation" of emotional reactivity to shock, but the authors reported an instance of accelerated responding in the presence of a stimulus, which they interpreted as disinhibition. The basic procedure was as follows. Rats, after achieving a stable level of bar-pressing behavior for periodic food reinforcement, were exposed during each daily experimental session to four ½ second electric shocks. In the first experiment, the shock intensities employed ranged from .25 ma. to 4.0 ma., the day-to-day intensity pattern varying for the three different experimental groups. By the end of ten "free shock" days, however, each experimental animal had had an equal number of experiences with each of the shock intensities employed. A fourth group of control animals were simply given undisturbed bar-pressing experience for ten days. Following the ten "shock adaptation" days, all subjects were given standard CER training. The CER procedure consisted of presentation of a 3-minute white noise (CS), terminating with a ½ second 1.0 ma. shock(US). Four such paired CS-US presentations were given during each daily two-hour bar pressing session. The three experimental groups, in contrast to the control subjects, displayed on the first CER day a significant tendency to increase responding in the presence of the white noise. This tendency, of course, was the exact opposite of the characteristic decrease in response rate produced by the CS in the control group. The experimental groups had, however, entered CER training with low baseline response rates, due to their "free shock" experience. The degree to which baseline response rate had been "inhibited" by free shock correlated significantly with the amount of response "speed-up" (rho= .83, $p < .001$) - the more the baseline rate had dropped, the greater was the tendency to accelerate responding.

The authors summarized their results by stating that "there is an apparent tendency for subjects with very low baselines to increase their response rates in the presence of the CS". (Brimer and Kamin, 1963, p. 510).[1]

Two follow-up experiments demonstrated that the acceleration in responding that had been observed in the first experiment was not a simple consequence of the prior experience with free shock, but depended upon a low baseline rate of operant responding. When animals were given free shock experience, but were then allowed to re-develop a normal rate of bar-pressing, the white noise did not produce any acceleration in response rate.

The authors interpreted the acceleration tendency in terms of Pavlovian disinhibition, suggesting that the main contribution of the studies might be "the development of a simple and stable preparation for the study of inhibitory and disinhibitory phenomena" (Brimer and Kamin, 1963, p. 515). They further pointed out, however, that to firmly establish

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[1] The Brimer and Kamin study reported a number of controls which made clear that the accelerated response rate was not a statistical artifact, but did depend on presentation of the white noise.

the disinhibition interpretation, it should be experimentally determined "whether the acceleration tendency can be reproduced when operant behavior is inhibited by other means (e.g., experimental extinction), and when extraneous stimuli in other sensory modalities are employed" (Brimer and Kamin, 1963, p. 515).

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The review of previous investigations concerned with disinhibition leads to the conclusion that, while the phenomenon has been demonstrated in several situations, little is in fact known about it. The phenomenon has appeared "fragile", and remarkably refractory to parametric investigation. This, however, may be largely attributable to the nature of the testing situations employed. Within many classical conditioning procedures, and pre-eminently with the GSR, an extraneous stimulus is not "neutral" with respect to the CR. With a discrete-trial operant response, such as in the runway studies, the handling of the subject between trials, and the distraction of the experimenter's presence during trials, does not augur well for a sensitive test of the effects of extraneous stimuli. The operant bar pressing procedure appears to be ideal in this respect, for the subject may be isolated in a light-proof, sound-proof chamber, and all experimental programming and recording done by automatic equipment. Further a "free" operant such as bar-pressing has the added advantage of making available a <u>continuous</u> record of response rate throughout the testing session.

Thus, adopting the bar-pressing operant as the conditioned response may make feasible sensitive parametric studies of the disinhibition phenomenon. This was certainly suggested by the great reliability across subjects of the effect reported by Brimer and Kamin (1963). The goal of this thesis was to carry out such investigations. Two major variables are involved in the disinhibition phenomenon, viz., the extraneous stimulus and the inhibition of the conditioned response. Thus the experimental work was directed toward these two areas. In total six experiments were carried out. Disinhibition was investigated with extraneous stimuli of different sensory modalities, different durations, and different intensities. The effects of different types of inhibitory operations were examined, although for most of the studies a standardized experimental extinction procedure was eventually employed. Finally, a series of experiments sought to determine whether response _suppression_, or only a _low_ level of responding, was the sufficient condition for the disinhibition phenomenon.

CHAPTER THREE

EXPERIMENT 1

The work to be reported consisted of six separate, but closely interrelated, experiments. The most coherent form of presentation will be to present each experiment in a separate chapter, with its own method, results, and brief discussion sections. The final chapter will review the findings of all six experiments in a concluding discussion.

METHOD

The type of subjects, the basic apparatus, and many procedural details were the same in all six experiments. Thus, an extended description of the method will be presented only in connection with Experiment 1.

Subjects and Apparatus

The subjects in all experiments were experimentally naive male hooded rats, supplied by Canadian Research Animal Farms, ranging in weight from approximately 250 to 300 grams. There were 64 subjects in Experiment 1, randomly assigned to eight experimental groups.

The apparatus consisted of eight standard Grason-Stadler operant conditioning units, ("Skinner boxes"), individually housed in sand-filled "ice-chest" type sound-attenuating wooden boxes. One wall of the Skinner box contained a food receptacle, a response lever, a loud speaker, and a house light. The two experimental stimuli employed were white noise, and light. The noise was produced by a Model 901A Grason-Stadler noise generator, which fed into the loudspeakers attached to the outside walls of the Skinner boxes. An eight-channel audio-splitter manufactured by Ashman Electronics Ltd., allowed the noise intensity delivered to each Skinner box to be independently adjusted. The mean baseline noise level for the eight Skinner boxes, with the exhaust fans operating, was 60 decibels as measured by a Type 1555-A General Radio Sound Survey Meter. The noise stimulus employed in Experiment 1 increased the sound level, at the rat's normal location, to 80 db. The light stimulus was produced by a 120-v. 6-watt electric light bulb attached to the outside wall of the Skinner box above the loudspeaker. The normal condition inside the Skinner box was complete darkness. When the light stimulus was employed, the mean reflected intensity inside the Skinner box at the rat's normal location was 2 foot candles as measured by a Leeds & Northrup Model 1046889 Macbeth Illuminator.

The entire experimental procedure was automatically programmed by Grason-Stadler relay and time circuits. Responses were recorded on Grason-Stadler print-out counters. The programming and recording equipment was contained in a room adjacent to the Skinner boxes.

Experimental Design

The first experiment was designed to extend the generality of our original observation of response acceleration to an extraneous stimulus, by employing stimuli other than 3 minute white noise, and inhibitory operations other than unsignalled electric shock.[1]

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[1] The operational definition of "inhibitory" was simply any procedure which produced a decrease in baseline response rate.

The design was a 2x2x2 factorial, with the three factors being:

(1) type of inhibitory operation

(2) modality of extraneous stimulus, and

(3) duration of extraneous stimulus.

The inhibitory operation was either experimental extinction of the bar pressing response, or food satiation. (The exact procedures employed in these two inhibitory conditions are described later.) The extraneous stimulus was either white noise at an 80 db. intensity level or house illumination of approximately 2 foot candles. The stimulus duration was either $1\frac{1}{2}$ seconds or 3 minutes, although the test measure always consisted of the number of responses in the 3-minute interval following stimulus onset.

Combining each level of each factor with each level of the other factors gives rise to eight possible experimental conditions, in a simple 2x2x2 factorial design. Eight subjects were trained under each of the eight conditions.[1]

Preliminary Training

The animals were first put on a 24-hour feeding rhythm which reduced them to approximately 75% of their ad lib body weight. The subjects were maintained at this weight throughout the preliminary phase of the experiment, being fed once daily, approximately one hour after each experimental session.

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[1] The experimental treatments were counter-balanced for both the time of day of running and the particular Skinner box employed. Thus, each experimental condition appeared equally often at each time of day and equally often in each Skinner box.

The subjects were initially trained to bar press for food and then given eight, 1-hour daily bar pressing sessions with a 2½ minute variable interval (VI) food reinforcement schedule. The reinforcement was standard Noyes food pellets. With the exception of the first VI day, when the house light was on throughout the hour, all training for all animals was carried out in complete darkness. This preliminary training procedure resulted in a stable rate of bar pressing, maintained throughout the experimental hour. On the last two VI days (Days P-1 and P-2) the appropriate extraneous stimulus (noise or light, of 1½ seconds or 3 minutes duration) was presented twice during each session as a pretest. This was to determine the effect of the stimulus on the "intact" (i.e., non-inhibited) operant response. On each pretest trial the number of responses occurring in the 3-minute interval preceding stimulus presentation, and in the 3-minute interval commencing with stimulus onset, was recorded. The effect of the stimulus could thus be quantified by an inflection ratio identical to the suppression ratio employed by Kamin (1961). This ratio is $\frac{B}{A+B}$, where "B" represents the number of responses during the 3-minute stimulus, and "A" represents the number of responses made in an identical interval immediately preceding stimulus onset. The ratio has limits of .00 and 1.00; where .00 represents complete response inhibition; .50, no effect of the stimulus on response rate; and 1.00, the case where no responses are made prior to the stimulus interval, but some are made during it.

Inhibition Training

Following the second pretest day, three inhibition training days were given. There were, as previously mentioned, two types of inhibitory operation employed; experimental extinction of the bar pressing response, and food satiation. In the extinction procedure animals were maintained at 75% of their ad lib weight but, during each experimental session, no food reinforcement was programmed. Thus, bar pressing was no longer reinforced. In the food satiation condition animals were allowed free access to food in the home cage for one hour prior to their introduction to the Skinner box; during each 1-hour experimental session the VI food reinforcement schedule remained in effect. Half of the 64 animals were run under each of the inhibitory operations. This division, of course, was orthogonal to the divisions into two stimulus modalities and two stimulus durations. The inhibition training was continued for three days.

Test for Disinhibition

Following the third inhibition training day, testing for disinhibition began. During the test phase of the experiment,the inhibitory operation of extinction remained in effect for the extinction animals as it had during the three prior inhibition days; that is, no food reinforcement was given for bar pressing. Satiation animals, however, were switched from a 1-hour to a 23-hour ad lib feeding schedule in the home cage. This change was necessitated by the fact that for some animals the 1-hour free feeding prior to Skinner box experience had not suppressed bar pressing to the requisite low level. Throughout the test, satiation animals continued to receive food reinforcement for bar pressing on the $2\frac{1}{2}$ minute VI schedule.

To assure that baseline response rate was equally inhibited in all animals at the time of testing, the presentation of the extraneous stimulus was made contingent on a criterion of three minutes with no response.

That is, the test stimulus was presented only when an animal had gone three minutes without a response.[1] The number of responses that occurred in the 3-minute interval following stimulus onset was then recorded. However, the probability of responding, after a momentary 3-minute interval without a response, is very likely greater than zero. This would be the case regardless of whether an extraneous stimulus was presented following the 3-minute interval without a response. To provide for this, each animal acted as its own control. This was accomplished as follows. On each test day, each animal received one "dummy" and one stimulus presentation, the order of the two being counterbalanced between subjects in each experimental group, and within each subject, from day to day.[2] The dummy presentation simply consisted of counting the number of responses that occurred in the 3-minute interval following the 3-minute no response criterion. Thus, disinhibition could be calculated by comparing an animal's response rate during the 3-minute stimulus interval with its rate during the comparable 3-minute dummy of the same test day. A disinhibition test trial thus refers to both the stimulus and dummy presentations occurring on the same test day.

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[1] This was accomplished through use of a recycling timer, which was reset by each bar press. When the timer timed out three minutes, the stimulus was automatically presented.

[2] This means that on each test day, following the initial 3-minute no response criterion, half the animals in each experimental group received a dummy presentation, and the other half, a stimulus presentation. For the second presentation on that day, the conditions would, of course, be reversed. The order of test presentation for each animal alternated from day to day so that animals who had a stimulus first on test day 1, received a dummy first on test day 2. Thus, for any two consecutive test days the order of presentation of stimulus and dummy was counterbalanced both between and within subjects.

The programming of test presentations was, as mentioned, contingent on the subject's behavior, but the contingency did not come into effect until nine minutes after the beginning of the daily session. The timer controlling presentations began to operate at that time. When the subject first achieved a 3-minute period without any bar presses, the first test presentation (stimulus or dummy) automatically began. The contingency was again suspended for a 6-minute interval beginning with onset of this presentation. Then, when subject next achieved a 3-minute period with no bar presses, the second test presentation began. There were never more than two test presentations (one stimulus, one dummy) for any subject on any test day. The experimental plan necessitated that the two presentations be given within a 1-hour session, as the boxes had to be utilized by other subjects.

The original test schedule called for one stimulus and one dummy presentation to be administered to each animal on each of four consecutive test days. This program was successfully carried out with all extinction subjects. However, the scheduled total of four stimulus and four dummy presentations was not obtainable with some of the satiation subjects, due to their relatively high baseline response rates. Complete data was available, however, for at least two test days for all satiation subjects.

RESULTS

Pretest Ratios

Figure 1 presents the median inflection ratios for each of the four stimulus conditions on each of the four pretest trials. Table 1 gives means, medians, and ranges for the first trial, and for mean ratios computed for individual subjects for all four pretest trials.

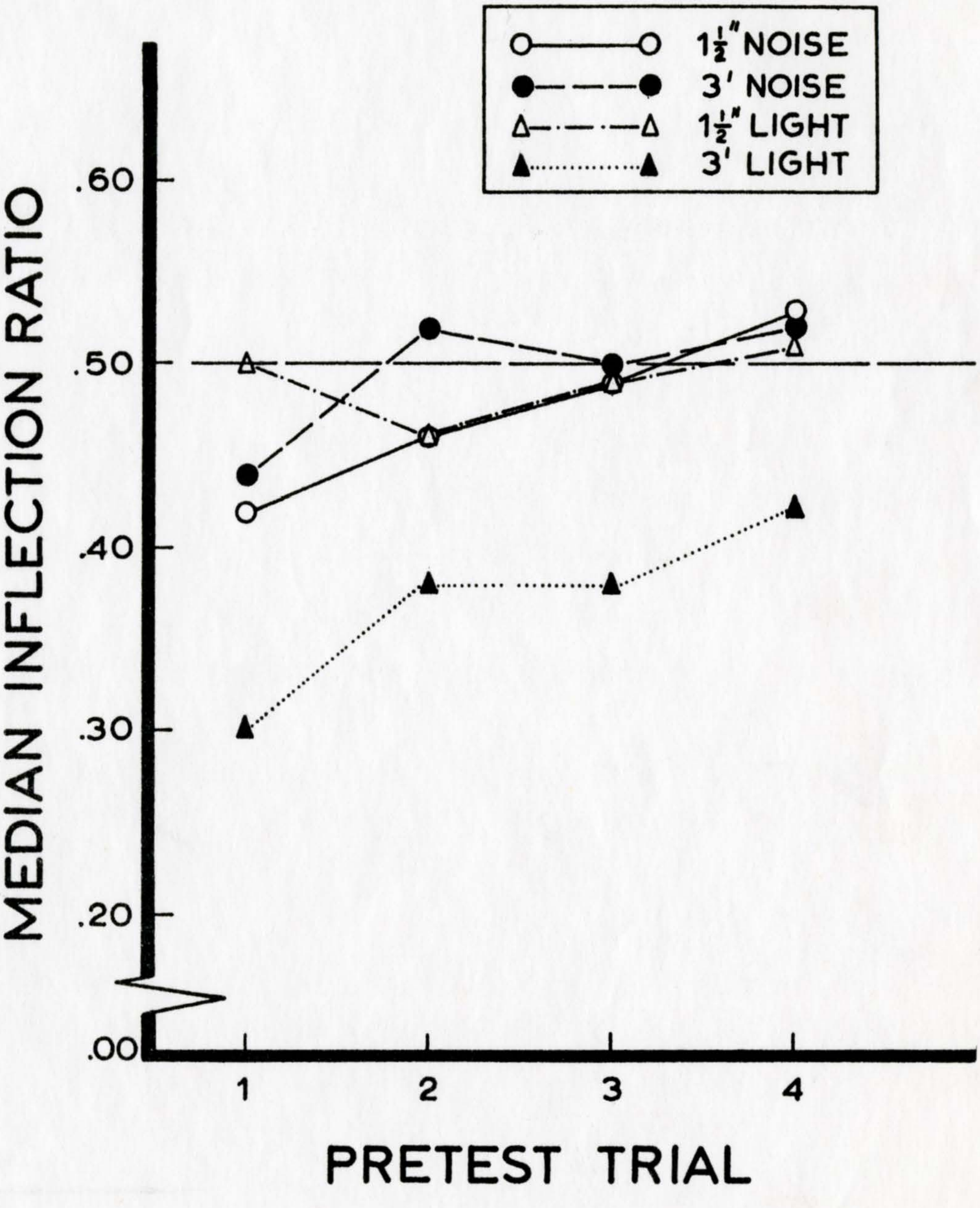

FIGURE 1. MEDIAN INFLECTION RATIOS AS A FUNCTION OF PRETEST TRIAL (EXPERIMENT 1)

TABLE 1

PRETEST INFLECTION RATIOS FOR DIFFERENT DURATION AND DIFFERENT MODALITY STIMULI (EXPERIMENT 1)

TRIAL 1

	NOISE		LIGHT	
	1½ SECOND	3 MINUTE	1½ SECOND	3 MINUTE
MEAN	.43	.43	.49	.30
MEDIAN	.42	.44	.50	.30
RANGE	.32 — .54	.25 — .59	.39 — .61	.20 — .47

TRIALS 1 - 4

	NOISE		LIGHT	
	1½ SECOND	3 MINUTE	1½ SECOND	3 MINUTE
MEAN	.47	.50	.47	.37
MEDIAN	.47	.50	.49	.38
RANGE	.42 — .53	.43 — .60	.44 — .58	.23 — .47

On the first pretest trial a significant proportion of animals in the $1\frac{1}{2}$ second noise (14/16, $\underline{p}<.01$, binomial test)[1] and 3 minute light (16/16, $\underline{p}<.01$) groups have ratios below .50, while the proportion of subjects in the 3 minute noise condition falls just short of significance (12/16, $.07<\underline{p}<.08$).[2] Thus, on initial presentation, all stimuli but the $1\frac{1}{2}$ second light tend to produce response suppression. It is clear from Figure 1, however, that the 3 minute light produces a markedly greater response decrement than the other stimuli. Testing the mean ratios for trials 1 to 4 by the Kruskal-Wallis ranked analysis of variance demonstrates that the overall differences between groups are statistically significant ($H=32$, $\underline{p}<.001$). A series of Mann-Whitney U tests reveal that the 3 minute light animals have lower ratios than the subjects in any of the three other stimulus conditions ($\underline{p}<.001$ in each case) but that none of the other groups differ significantly. It is also clear from Figure 1 that the suppressant effect of stimulus presentation dissipates with repeated trials. For each of the three groups initially showing response inhibition the inflection ratios are significantly higher on pretest trial 4 than on pretest trial 1 ($\underline{p}<.01$ in each case, Wilcoxon's test for paired replicates).

Baseline Response Rates

Figure 2 presents the daily median response rates for the subjects run under the two different inhibitory operations. These rates were based on total responses emitted by each animal during the daily 1-hour experimental session.

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[1] The null hypothesis for the binomial test is, of course, that $P=Q=\frac{1}{2}$. In other words the assumption is that half the animals will increase and the other half decrease response rate.

[2] An additional five subjects were later run with 3 minute noise for comparison with groups in the next experiment. When these subjects are added to the present 16, the proportion of animals with ratios below 50 is significant (16/21, $\underline{p}<.03$).

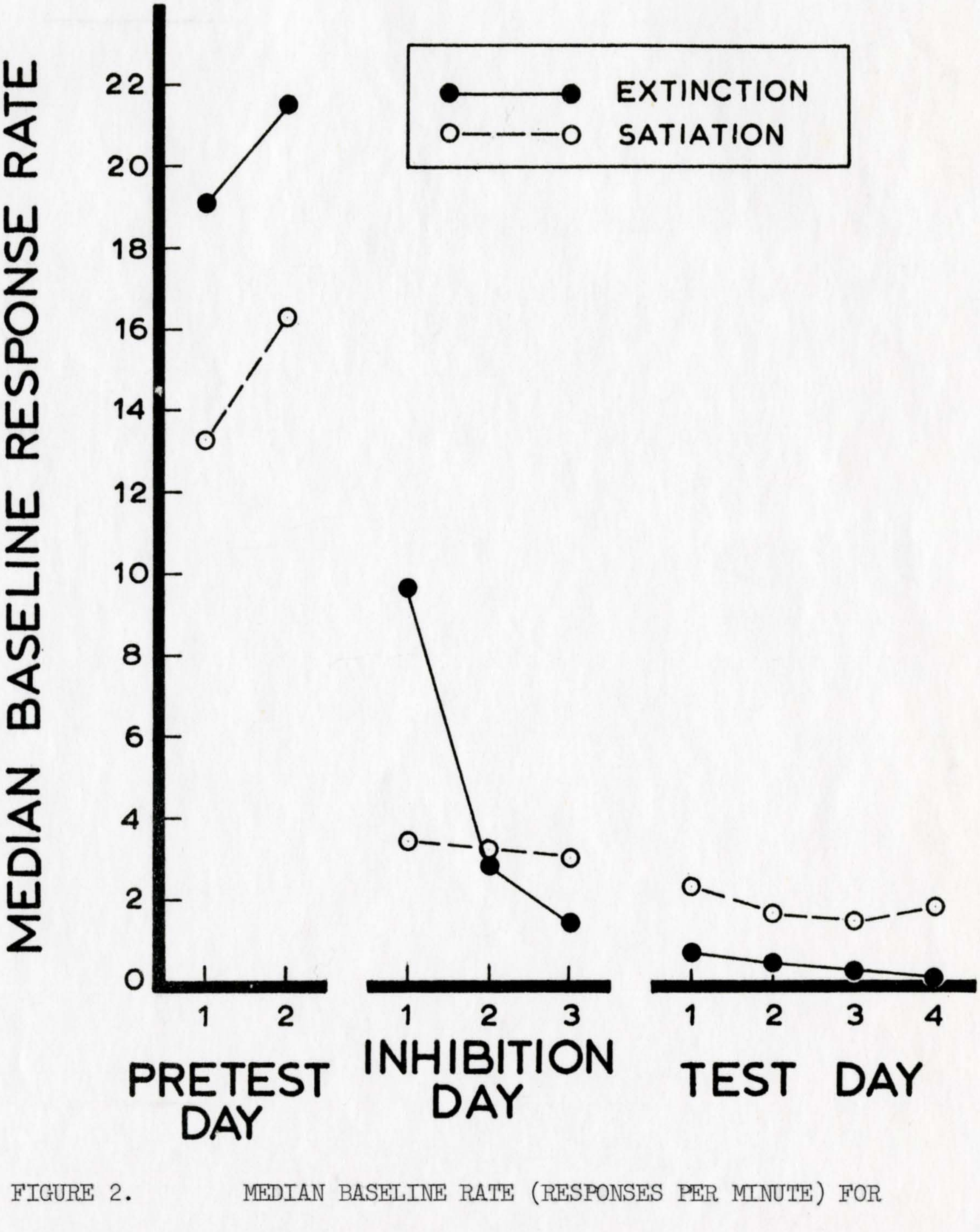

FIGURE 2. MEDIAN BASELINE RATE (RESPONSES PER MINUTE) FOR EXTINCTION AND SATIATION SUBJECTS, AS A FUNCTION OF TRAINING DAY (EXPERIMENT 1)

On pretest (Days P-1 and P-2) the two groups do not differ in response rate. With the introduction of inhibition training all animals decrease their response rates from the previous day's level ($p < .0001$, binomial test), with the satiation subjects dropping to a significantly lower rate than the extinction animals ($U=137$, $p < .001$). By the third inhibition day, however, this situation has been reversed, so that the response rate of the extinction group is now significantly lower than that of the satiation subjects ($U=150$, $p < .001$). This relationship is maintained over the four test days ($p < .01$ on each day). Thus, although the satiation procedure initially produces a dramatic drop in response rate, with continued training, extinction proves to be a more effective response inhibitor than food satiation. Both techniques, however, were successful in achieving inhibition of responding.

Test for Disinhibition

As previously mentioned complete test data was available for at least two test trials (i.e., two stimulus and two dummy presentations) for all animals. To analyze this data the responses emitted during the first two stimulus and first two dummy presentations were cumulated separately for each subject. These data are summarized in Figure 3, which presents median number of responses for each type of presentation for the eight experimental conditions. The overall rate of responding is clearly higher with the satiation than with the extinction procedure. This is true both for the stimulus ($U=254$, $p < .001$) and for the dummy ($U=315$, $p < .01$) intervals. These differences simply reflect the fact that food satiation was not so effective an inhibitory operation as was extinction. The more important finding, however, is that disinhibition

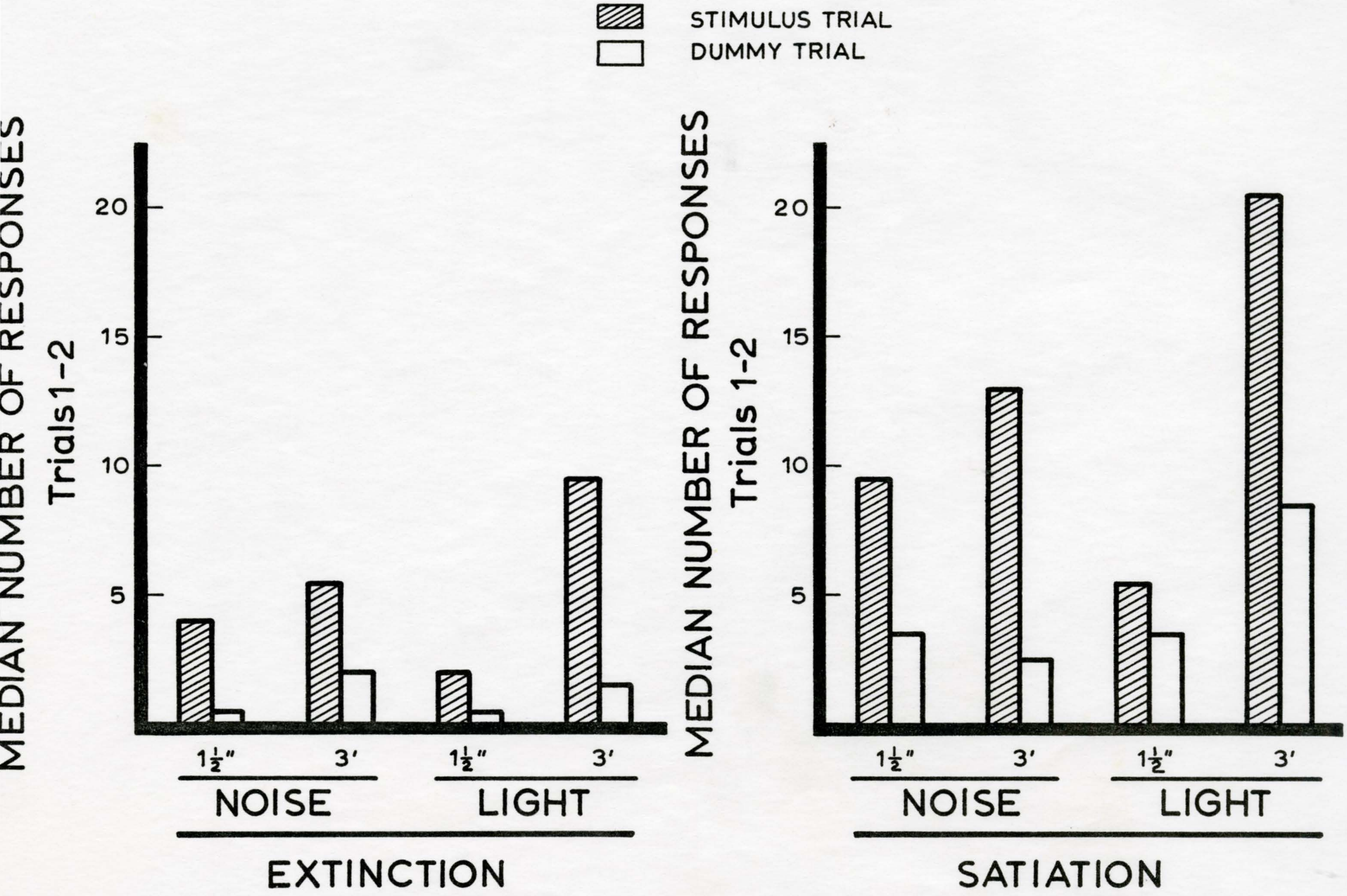

FIGURE 3. MEDIAN NUMBER OF RESPONSES (TRIALS 1 - 2) DURING STIMULUS AND DUMMY INTERVALS FOR THE EIGHT EXPERIMENTAL CONDITIONS (EXPERIMENT 1).

clearly occurred. This is shown by the consistent tendency for more responses to occur in the stimulus, than in the dummy interval.

Temporarily disregarding experimental condition, the response rate is higher during the stimulus than during the dummy interval for a significant proportion of the subjects (51.5/64, $\underline{p} < .001$).[1] Looking at the individual experimental groups, all but the satiated 1½ second light animals, have, by Wilcoxon's test, a significantly higher response rate during the stimulus than during the dummy presentation. Thus, even with only eight subjects per group, it is found that seven out of the eight experimental conditions independently produce significant disinhibition.

To examine differences between groups in the magnitude of the disinhibition effect, each animal was assigned a difference score for trials 1 and 2, cumulated. This measure was calculated simply by subtracting the number of responses emitted during the first two dummy presentations from the number of responses that occurred on the first two stimulus presentations.[2] Thus, a positive difference score indicates

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[1] In all the analyses involving difference scores, or changes in response rates, ties were split between the two categories (increase and decrease) following the procedure recommended by Edwards (1954).

[2] It would, of course, be possible to index the difference in stimulus and dummy response rates by an inflection ratio similar to that employed on pretest. The problem that this creates is that the ratio measure fails to discriminate between animals that do not respond during the dummy but have differential rates of responding during the stimulus. Thus an animal with 0 and 5 responses during the dummy and stimulus periods, respectively, would receive the same ratio score as a subject with 0 and 50 responses in the two corresponding intervals. The difference score appears to give a more sensitive picture of the magnitude of the disinhibition.

disinhibition. Table 2 presents the means, medians, and ranges for the difference scores for the eight experimental groups. Table 3 summarizes the analysis of variance of these difference scores.

Looking at the analysis of variance we find two significant main effects. First, in terms of inhibitory operation, greater disinhibition is produced by the satiation procedure than by extinction. This may well be attributable, however, to the fact that overall level of responding was higher under the satiation procedure. That is, the magnitude of the disinhibition effect may depend upon the amount of inhibition of the operant response.

The other significant main effect is that of stimulus duration. It is found that $1\frac{1}{2}$ second stimuli are less effective disinhibitors than 3 minute stimuli, when response rate is measured for the three minutes following stimulus onset. There are no significant interactions.

There is an interesting parallel between the pretest (inhibition) and test (disinhibition) results. It will be remembered that, on pretest, the 3 minute light produced the most response inhibition and the $1\frac{1}{2}$ second light the least. On test these two stimuli tend to produce, respectively, the most and the least disinhibition. If one rank orders the eight experimental groups in terms of the median inflection ratio on the first pretest trial and the median difference score on the first disinhibition test day, then the correlation between the two measures is significant (rho= -.67, $\underline{p}<.05$,n=8). The negative relationship indicates that the lower the inflection ratio was on pretest, then the higher the difference score tended to be on test. Thus, those stimuli which, before application of an inhibitory operation, are the more effective response inhibitors are, afterwards, also the more potent disinhibitors.

TABLE 2

STIMULUS-DUMMY DIFFERENCE SCORES, TRIALS 1 - 2, FOR DIFFERENT STIMULI AND DIFFERENT INHIBITORY OPERATIONS (EXPERIMENT 1)

EXTINCTION

	NOISE		LIGHT	
	1½ SECOND	3 MINUTE	1½ SECOND	3 MINUTE
MEAN	3.4	2.8	1.8	7.9
MEDIAN	3.5	1.5	1.5	7.5
RANGE	-2.0 — 8.0	-4.0 — 14.0	-6.0 — 9.0	1.0 — 16.0

SATIATION

	NOISE		LIGHT	
	1½ SECOND	3 MINUTE	1½ SECOND	3 MINUTE
MEAN	6.6	12.8	2.4	14.4
MEDIAN	3.5	10.5	1.5	18.5
RANGE	0 — 17.0	0 — 37.0	-9 — 13.0	-20.0 — 36.0

TABLE 3

SUMMARY OF ANALYSIS OF VARIANCE OF DIFFERENCE SCORES, TRIALS 1 - 2. (EXPERIMENT 1)

SOURCE	d.f.	MEAN SQUARE	F	p
INHIBITORY OPERATION (A)	1	415.2	4.85	$<.05$
STIMULUS MODALITY (B)	1	.8	-	
STIMULUS DURATION (C)	1	558.2	6.52	$<.02$
A x B	1	37.4	-	
A x C	1	159.3	1.86	
B x C	1	159.3	1.86	
A x B x C	1	.8	-	
ERROR	56	85.6		

The preceding analyses have concerned the data for the first two stimulus and dummy presentations only, since some animals in the satiation groups received only two trials. We now turn to the question of the magnitude of the disinhibition effect as a function of test trial. For this purpose, we have complete data for 4 trials for the extinction subjects, and for 2 trials for the satiation subjects.

Figure 4 presents the median number of responses made on each stimulus and dummy presentation, separately for extinction and satiation subjects. Within each of these conditions, the four stimulus groups have been pooled.[1] For the extinction procedure, it seems clear that the disinhibition effect, calculated as the difference between stimulus and dummy, decreases with continued testing. There is a significant drop in difference score occurring from trial 1 to 4 ($T= 150$, $\underline{p} < .05$). The significance of this finding is ambiguous, however. For, while consecutive trials involve increasing familiarity with the stimulus, they also appear to involve an increasingly lower overall response rate. When dummy presentations alone are considered there is also a significant difference in the number of responses on test days 1 and 4 ($T=118$, $\underline{p} < .01$). Thus, it is unclear whether decreasing disinhibition is attributable to increasing familiarity with the stimulus or to decreasing baseline response rate.

For the satiation animals, there are no significant changes in response rates from test day 1 to test day 2. For the 13 animals which

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[1] The raw data for the subjects in the different groups appears in the Appendix. Unfortunately the small size of the groups does not allow individual statistical treatment of the different stimulus conditions.

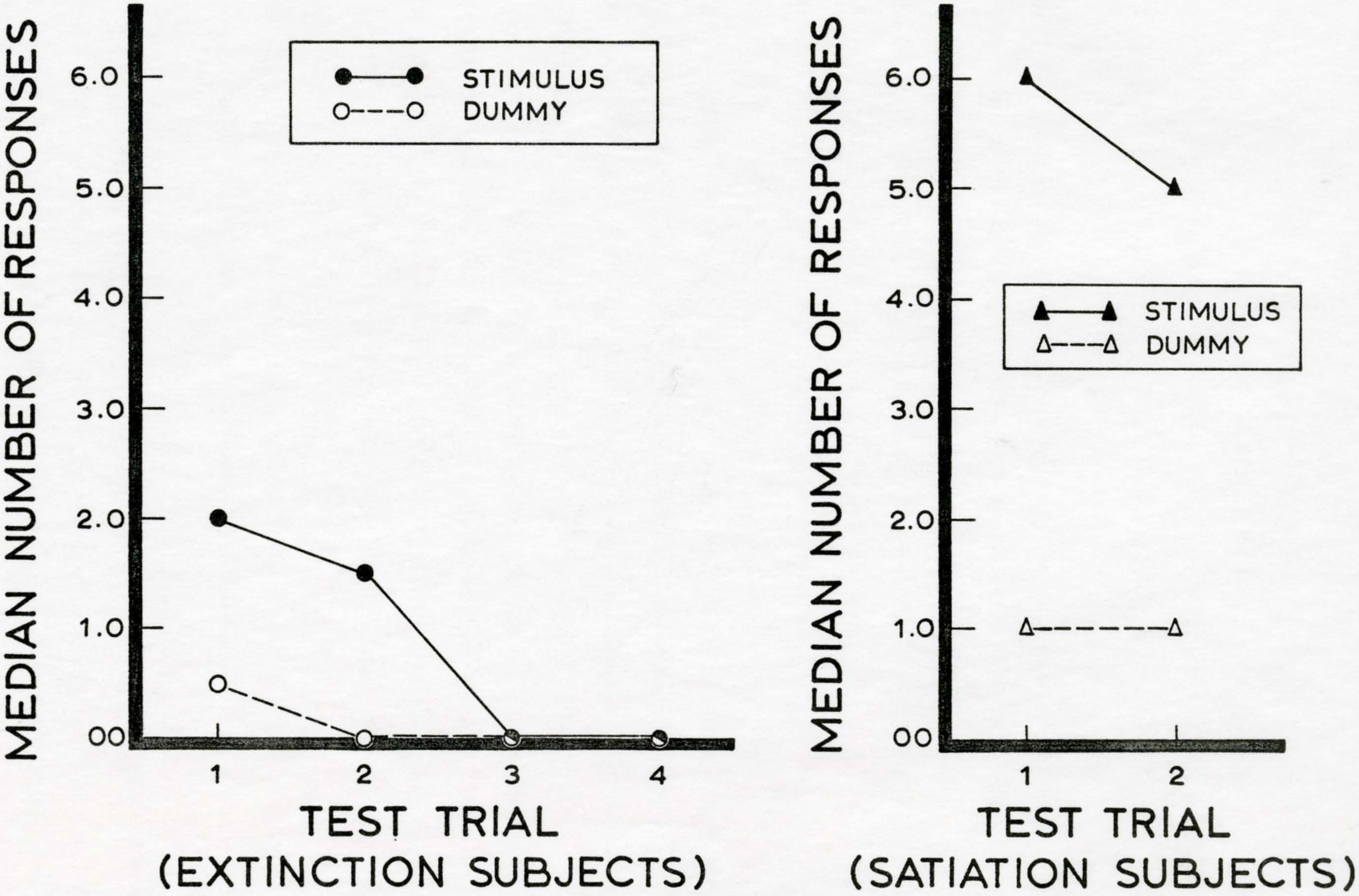

FIGURE 4. MEDIAN NUMBER OF RESPONSES DURING STIMULUS AND DUMMY INTERVALS

received four test trials there was again no significant change either in baseline rates or responses to the stimulus from days 1 to 4. These 13 animals, however, constitute a selected sample - those rats with baseline rates sufficiently low to allow 2 test presentations on each test day.

Following completion of the main portion of Experiment 1, a final group of 8 rats was subjected to a different inhibitory operation, as an attempted _tour de force_. The question raised was whether, if bar pressing were inhibited by _punishment_ (electric shock for each bar press), the disinhibitory effect would still be exhibited - even though shock punishment for bar pressing remained in effect throughout the test for disinhibition. The basic procedure was the same as that previously outlined, except that there were no inhibition training days separate from the testing days. The punishment contingency suppressed bar pressing so rapidly that each subject could receive both stimulus and dummy presentations on the first day of punishment, as well as on all subsequent days. The shock employed to inhibit bar pressing was delivered to the feet of the animal from the grid floor of the Skinner box. The shock intensity was individually adjusted for different animals to a level that effectively inhibited responding. The intensities employed varied from .25 ma to .50 ma calibrated on a Grason-Stadler Model E1064GS shock generator. Four test days were given immediately following pretest day, each animal receiving one dummy and one stimulus presentation on each day. The presentations, as in the earlier experiment, were contingent on a 3-minute no response criterion. Throughout the test phase of the experiment, the VI food reinforcement schedule remained in effect. The disinhibiting stimulus employed consisted of both the light and noise, presented concurrently. In all other respects the procedure was identical to that of Experiment 1.

Figure 5 presents the median number of stimulus and dummy responses, summed for the four test days. All eight animals have a higher response rate during the stimulus than during the dummy interval (T=0, p =.01).

DISCUSSION

The major conclusion to be drawn from Experiment 1 is, simply, that the disinhibition effect has a considerable generality. The observed differences in the amount of disinhibition produced in various experimental groups are of relatively less theoretical significance. The important point is that, considered independently, all but one of the eight experimental treatments produced clear disinhibition. Thus, the acceleration in response to an extraneous stimulus which we first observed in association with "free shock" as an inhibitor (Brimer and Kamin, 1963) clearly is not specific to the use of shock as an inhibitor, nor to the use of white noise as a disinhibitor. We have now demonstrated that the response acceleration effect occurs under four types of inhibitory operation (unsignalled shock, extinction, food satiation, and punishment), two types of stimulus (light and noise), and two stimulus durations ($1\frac{1}{2}$ second and 3 minute). The facts that the effect appears when operant responding is inhibited by experimental extinction, and that it is produced by extraneous stimuli in at least two sensory modalities, appear to justify identification of the phenomenon with Pavlovian disinhibition. The phenomenon, indeed, appears even more general than Pavlov reported; for we observed it when operant responding was diminished by satiating the animals with food, without any experimental extinction (this, in Pavlovian language, amounts to reducing the excitability of the centre of the unconditioned reflex).

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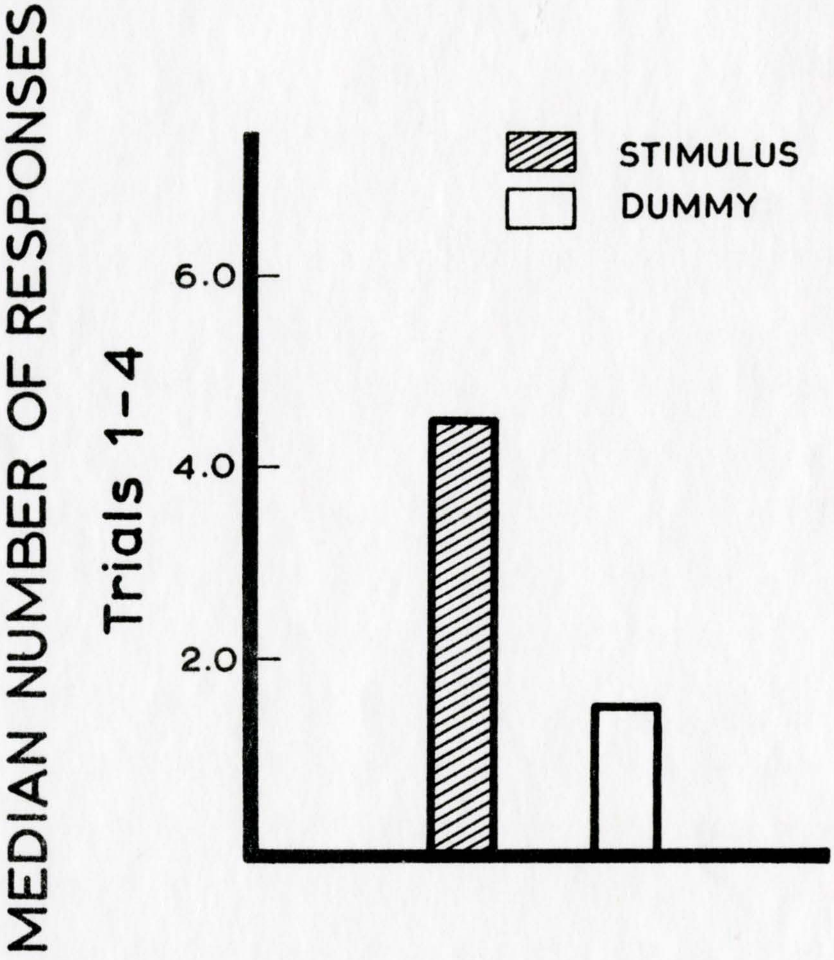

FIGURE 5. MEDIAN NUMBER OF RESPONSES (TRIALS 1 - 4) DURING STIMULUS AND DUMMY INTERVALS FOR PUNISHMENT PROCEDURE (EXPERIMENT 1)

The fact that response acceleration occurs with the presentation of an extraneous stimulus even when each bar press is punished with shock, dramatically demonstrates the strength of the tendency to disinhibit.

We found, as Pavlov and others have reported, that the stimuli which disinhibit the suppressed response, inhibit the non-suppressed response. The relationship between the inhibitory and disinhibitory capacity of stimuli appeared to be linear, rather than curvilinear as Pavlov had suggested. The better inhibitors were the better disinhibitors. Possibly, however, had we employed stimuli which more radically inhibited pretest responding, the disinhibition that occurred on testing with these stimuli might have been attenuated. The important point seems to be that the same stimulus which reduces the probability of responding when the animal is in one state, increases the probability of responding when the animal is in another state. The effects of an extraneous stimulus on bar pressing rate cannot, therefore, be attributed to any simple relation between the stimulus and responses directly elicited by it which interfere with or facilitate bar pressing. The state of the animal at the time of presentation of the stimulus is crucial.

The fact that the disinhibition effect diminishes with repeated trials (at least when the inhibitory operation is experimental extinction) raises the question of whether this diminution is to be attributed to increasing familiarity with (adaptation to) the stimulus, or to the progressive weakening of the basic tendency to perform the bar pressing response. The second experiment was designed to investigate the effect of differential degrees of familiarity with the stimulus on the disinhibition effect.

CHAPTER FOUR

EXPERIMENT 2

This study was designed to examine the effects of previous familiarity with the stimulus on its subsequent efficacy as a disinhibitor. Within Experiment 1, all groups had had four experiences (pretest trials) with the stimulus <u>before</u> it was employed as a disinhibitor. Within Experiment 2, new groups are added, providing different amounts of previous experience with the stimulus.

Those new groups in the present study which receive <u>no</u> pretest trials control for the possibility of an order-effect artifact in the inhibition-disinhibition phenomenon. It is logically possible that the reaction to an extraneous stimulus changes sign with repeated exposure to the stimulus. Inhibition might occur when the stimulus is presented for the first time, and disinhibition occur after the subject is familiar with the stimulus. Thus, it might be that the opposed outcomes of inhibition and disinhibition could be accounted for merely in terms of experience with the test stimulus.[1]

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[1] Lending some credence to this possibility is the fact that typically investigators have pretested animals with the extraneous stimulus prior to inhibition training and the later test for disinhibition. Thus familiarity with the stimulus and the animal's baseline response rate are usually confounded.

METHOD

Within Experiment 2, and in all subsequent experiments, the inhibiting operation was extinction, and the disinhibiting stimulus was of three minutes' duration. The procedural details of Experiment 2 were in most respects identical to those of Experiment 1.

Three new groups of rats (32 subjects) were trained for Experiment 2. Two of these groups were trained exactly as were the 3 minute noise and light (extinction) groups in Experiment 1, except that _no stimulus_ was presented on the pretest days. The third new group was pretested with noise for eight days (i.e., 16 pretest trials) prior to the introduction of the inhibitory operation. The training procedure for this group was otherwise identical to that of the others. It should be noted that the extra pretests meant, however, that this one group received six extra days of VI training before extinction was begun.

Finally, both to increase the size of a group in Experiment 2, and for use in a subsequent experiment, five additional rats were trained with four noise pretest trials (two pretest days). These subjects were added to the eight already so trained in Experiment 1. The total number of animals in each of the five groups considered in Experiment 2 is given in the table below:

Group	N
Noise, 0 pretest trials	12
Noise, 4 pretest trials	13
Noise, 16 pretest trials	8
Light, 0 pretest trials	12
Light, 4 pretest trials	8

Thus, with the noise condition, 3 degrees of familiarity with the stimulus can be compared. Within the light condition, two degrees of familiarity were studied.

RESULTS

Pretest

The pretest data for groups given four pretest trials has already been analyzed in Experiment 1 (cf. Figure 1, Table 1). Figure 6 presents the median inflection ratios for the subjects given 16 pretest stimulus presentations. It is clear that, as in Experiment 1, the noise significantly suppresses responding only on the first trial. Thereafter, inflection ratios tend to hover about the indifference value of .50. On the initial pretest stimulus presentation, the drop in response rate from the pre-stimulus level was significant ($T=1.0$, $\underline{p} < .02$). Seven of the eight animals had inflection ratios below .50. By the sixteenth trial the stimulus produces no consistent response change ($T=13.0$, $\underline{p} > .05$). There is a significant increase in the magnitude of the inflection ratio between trials 1 and 16. ($T=0.0$, $\underline{p} < .01$).

Test for Disinhibition

Figure 7 presents the median number of responses during the stimulus and dummy intervals for all groups. The scores in all cases are based on disinhibition test days 1 to 4.

The amount of disinhibition was, as in Experiment 1, measured for each subject by a difference score contrasting its responses during the stimulus to its responses during the dummy. The figure makes it obvious that there is no difference between the light subjects that did and did not undergo pretest ($U=43.0$, $\underline{p} > .05$). Pooling both light groups, however, a

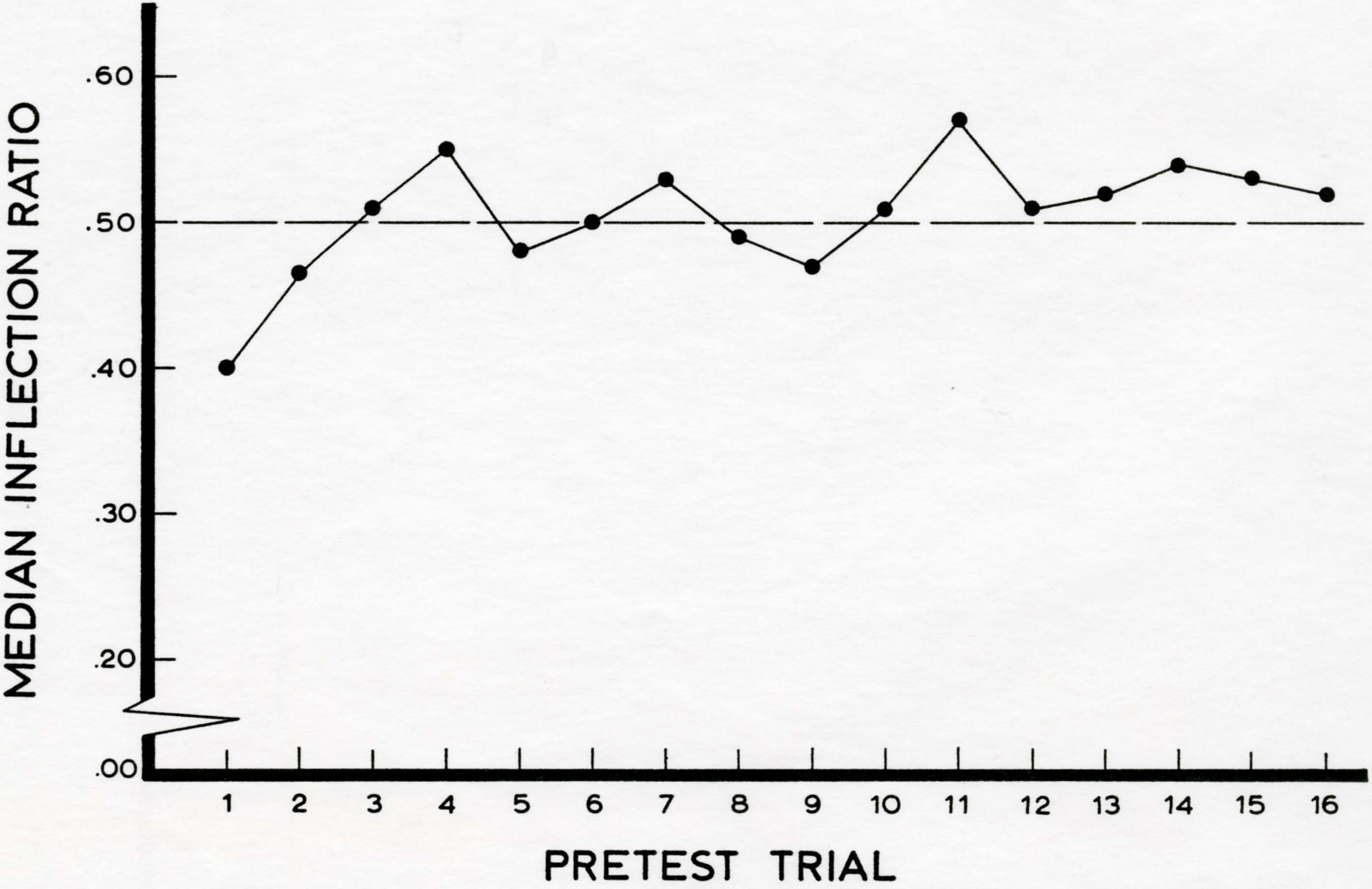

FIGURE 6. MEDIAN INFLECTION RATIOS AS A FUNCTION OF PRETEST TRIAL (EXPERIMENT 2)

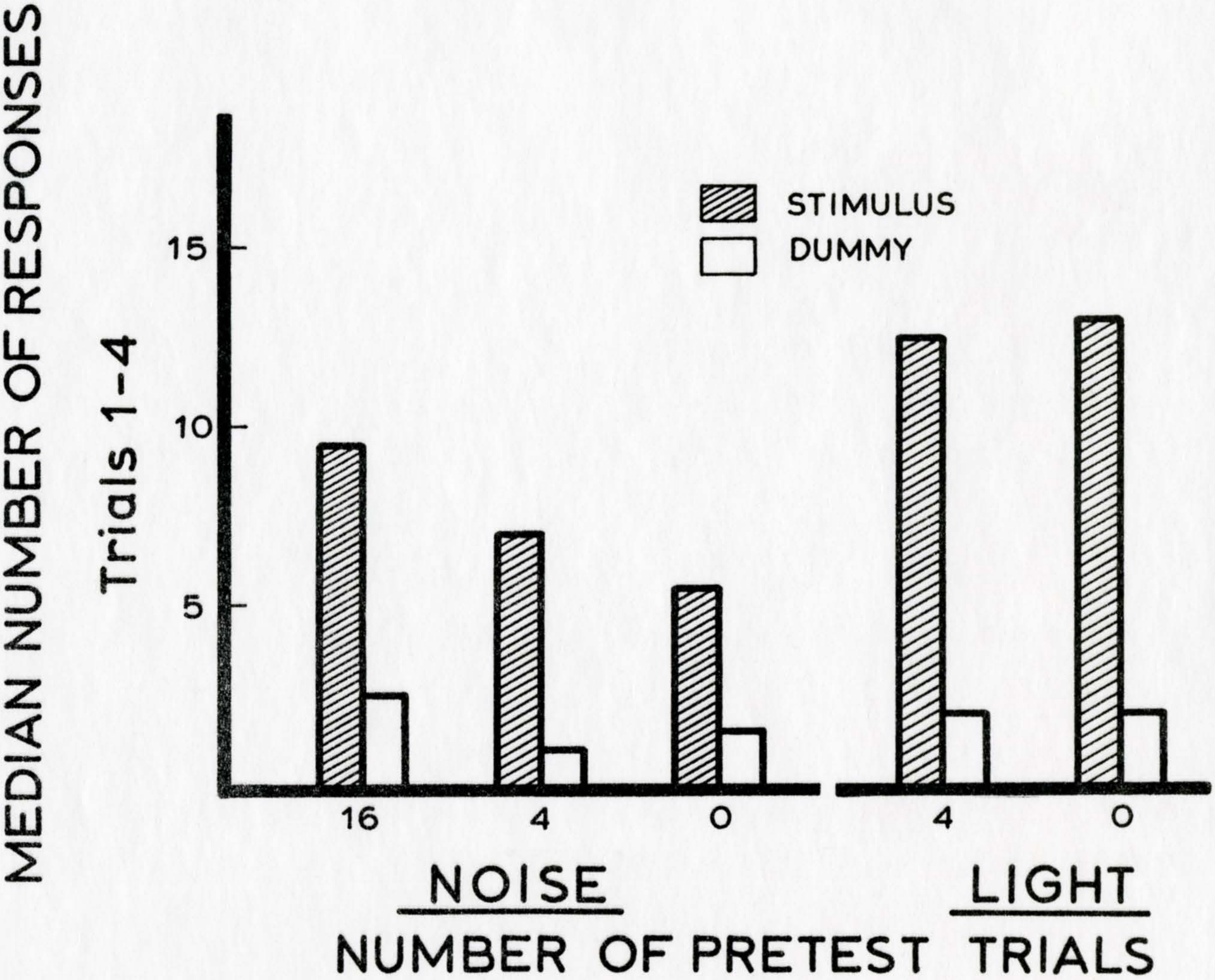

FIGURE 7. MEDIAN NUMBER OF RESPONSES (TRIALS 1 - 4) DURING STIMULUS AND DUMMY INTERVALS FOR NOISE AND LIGHT SUBJECTS AS A FUNCTION OF NUMBER OF PRIOR PRETEST TRIALS (EXPERIMENT 2)

significant proportion of the animals (19.5/20, $p < .01$) have higher response rates during the stimulus than during the dummy intervals. Thus, independent of pretest experience, 3 minute light produces significant disinhibition on testing.

Within the three noise groups there was again no significant effect of number of pretest trials on amount of disinhibition ($H=3.2$, $p > .05$). The non-significant differences actually indicate more disinhibition with repeated pretesting. Again, as with light, a significant proportion of the noise subjects (27.5/33, $p < .01$) did show the disinhibition effect.

DISCUSSION

The results of Experiment 2 seem clear. The amount of previous experience with the stimulus did not differentially affect the magnitude of disinhibition - at least not within the limits tested. Thus, it seems safe to conclude that familiarity with the stimulus is not a very important variable in the disinhibition phenomenon. Therefore, the diminishing amount of disinhibition observed (cf. Figure 4) with repeated test trials cannot be attributed to the stimulus familiarity variable alone. This in turn encourages the speculation that the lessening amount of disinhibition with repeated trials is attributable to the progressive weakening of the basic tendency to bar-press. Possibly, then, disinhibition can only be demonstrated when the basic tendency to respond is not _too_ inhibited. The conditions of our experiments to date have confounded repeated test trials with (in Pavlovian language) "extinction below zero" of the bar pressing response.[1] This is a problem to which we shall return in a later experiment.

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[1] There is, of course, at least one alternative interpretation. Possibly, each "exercise" of the disinhibition effect makes further such exercise less probable. This might occur quite independently of increasing familiarity with the stimulus, or of progressive changes in strength of the baseline response. We also should note that the "familiarity" with the stimulus in the present studies was acquired *after* the bar-pressing response was well established.

CHAPTER FIVE

EXPERIMENT 3

The preceding experiment examined the effect of familiarity with the stimulus on disinhibition. The following two experiments investigate other characteristics of the disinhibiting stimulus.

Within Experiment 3, the characteristic of the stimulus investigated was that of "onset" versus "offset" of a physical energy. The early Pavlovian literature seemed to imply that conditioning, at least, was a direct function of the total amount of physical energy impinging on the "cortical analyzers" (cf., e.g., Kupalov and Gantt, 1927). This notion is in many essential respects similar to Hull's concept of "stimulus intensity dynamism" (Hull, 1951).

Taken literally, this notion suggests that conditioning should be more effective when the conditioned stimulus consists of the onset of, for example, noise against a background of silence, than when it consists of the offset of an otherwise steady background noise. There has, in fact, been relatively little work on the effects of onset versus offset on conditioning (cf., e.g., Champion, 1962; Logan, 1954; Logan and Wagner, 1962). Recent unpublished work by Hilton and Kamin (1963), however, shows clearly that onset of a 3 minute 80 db. white noise is a far more effective CS in establishing a conditioned emotional response in the rat than is offset of an 80 db. noise. However, Kamin (1963) also reports that there is no difference between light onset and light offset in establishing a CER in the same animal.

There has been no previous study of the effects of this variable on disinhibition. Experiment 3 explores this variable, using the same baseline procedures (rats bar pressing on a 2½ minute VI schedule) used by Kamin in his CER conditioning studies.

METHOD

For Experiment 3 two new experimental groups (20 subjects) were trained. These groups received exactly the same training given the 3 minute noise and 3 minute light groups of Experiment 2, with one exception. From the second day of preliminary training, either the 80 db. noise or the light was continually on inside the Skinner box. For both pretest and disinhibition testing, the experimental stimulus consisted of a 3 minute interruption in either the noise or the light. The inhibition training procedure was, as usual, experimental extinction. For comparison the appropriate noise-on and light-on groups from Experiment 1 were incorporated in the analysis. The numbers of animals in each of the four experimental groups were: noise-on, 13; noise-off, 12; light-on, 8; light-off, 8.

RESULTS

Pretest

Figure 8 presents the median inflection ratios plotted over the four pretest trials. Table 4 presents the means, medians, and ranges for

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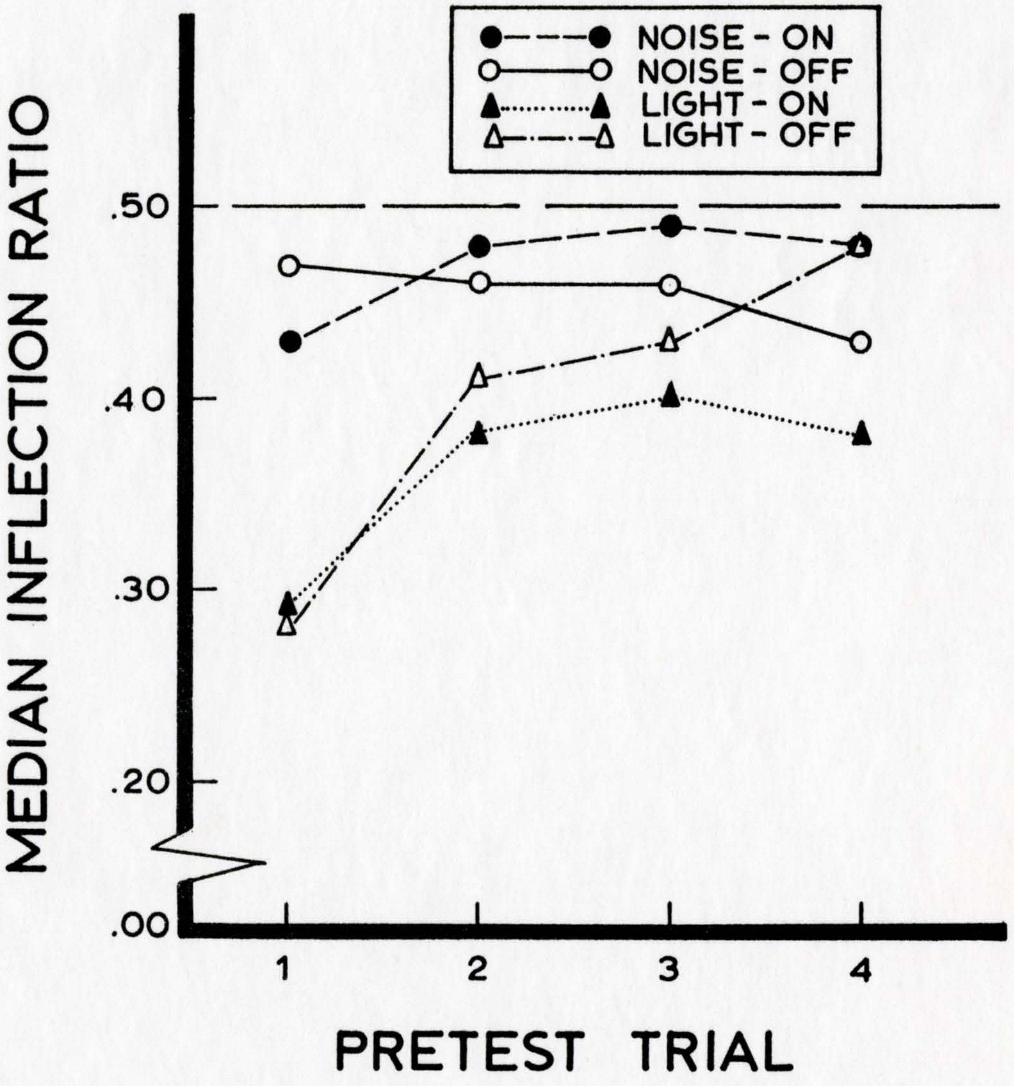

FIGURE 8. MEDIAN INFLECTION RATIOS FOR THE FOUR EXPERIMENTAL CONDITIONS, AS A FUNCTION OF PRETEST TRIAL (EXPERIMENT 3)

TABLE 4

PRETEST INFLECTION RATIOS FOR THE ONSET AND TERMINATION OF NOISE AND LIGHT (EXPERIMENT 3)

TRIAL 1

	NOISE		LIGHT	
	ON	OFF	ON	OFF
MEAN	.44	.47	.32	.27
MEDIAN	.43	.47	.29	.28
RANGE	.28 — .58	.36 — .56	.20 — .47	.20 — .34

TRIALS 1 - 4

	NOISE		LIGHT	
	ON	OFF	ON	OFF
MEAN	.48	.45	.37	.39
MEDIAN	.47	.46	.37	.40
RANGE	.39 — .56	.44 — .50	.23 — .47	.35 — .41

pretest inflection ratios for the first trial and for mean scores based on trials 1 to 4.

Considering the four groups individually, on the first pretest trial there is a significant tendency for all but the noise-off animals to suppress responding. Testing the differences in response rate between the three minute stimulus and three minute pre-stimulus periods (Wilcoxon's test) yields the following results: noise-on ($T=10$, $p =.01$), noise-off ($T=15$, $p >.05$), light-on ($T=0$, $p =.01$), light-off ($T=0$, $p =.01$). Within either the noise or light treatment the difference in ratios between the on and off groups is not significant. However, the light groups pooled, do show significantly more inhibition than the noise groups pooled.

Test for Disinhibition

Figure 9 presents the median number of responses emitted during the stimulus and dummy intervals for each of the four experimental groups on test days 1 to 4. Employing difference scores for trials 1 to 4 as our usual measure of disinhibition, we find that the light-on and light-off groups do not differ ($U=30$, $p >.30$). The light subjects overall show a significant disinhibition effect, 15 of the 16 subjects having higher stimulus than dummy response rates ($p <.001$).

The difference between the noise-on and noise-off groups is on the borderline of statistical significance ($U=44.5$, $.06 < p < .07$). Considered separately, the noise-on subjects do have significantly higher response rates during the stimulus than during the dummy periods ($T=5.5$, $p <.01$), but the noise-off animals do not ($T=18.5$, $.10 < p < .15$). Thus, turning noise on as the stimulus did produce significant disinhibition, whereas, turning noise off, did not.

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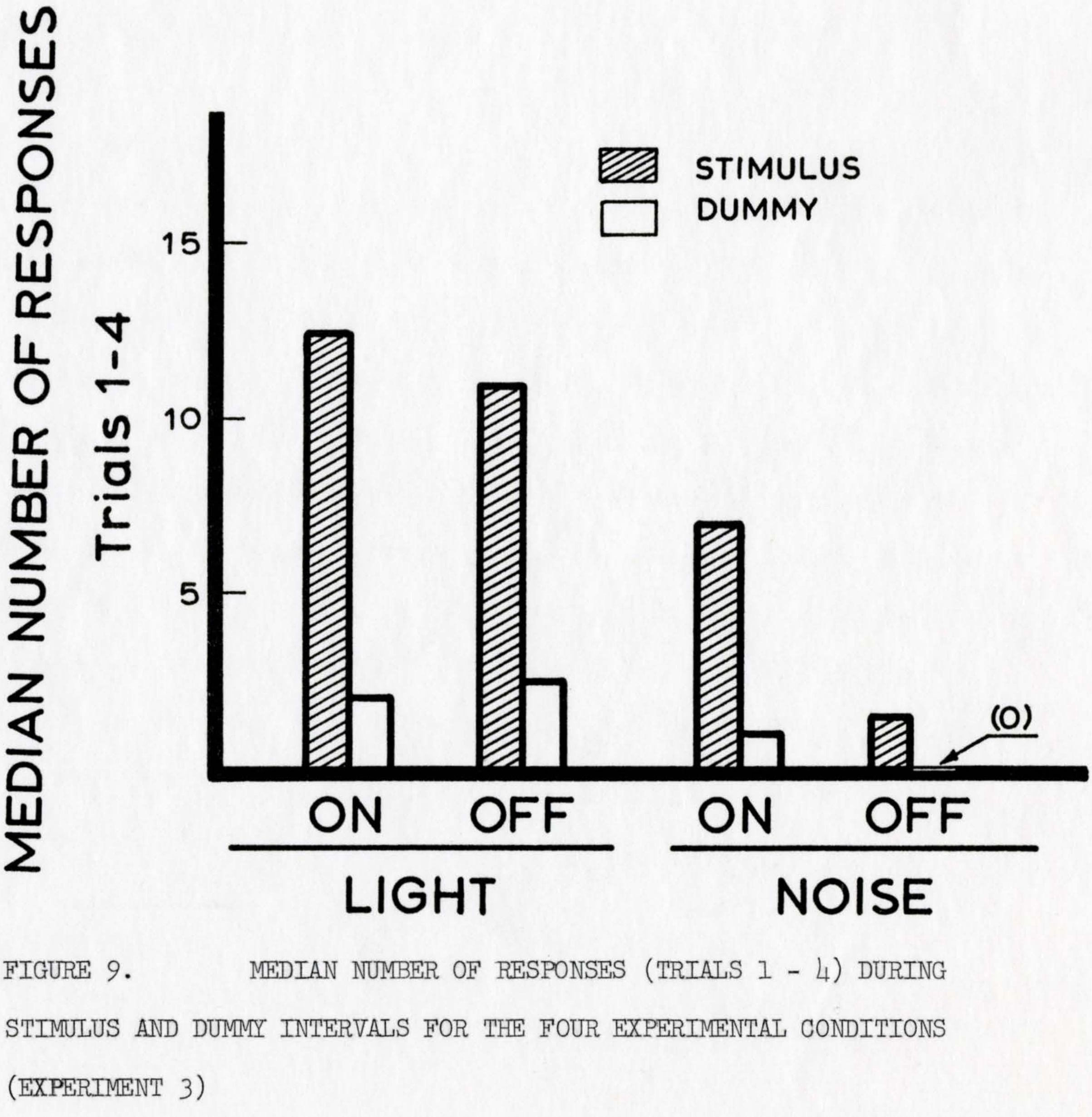

FIGURE 9. MEDIAN NUMBER OF RESPONSES (TRIALS 1 - 4) DURING STIMULUS AND DUMMY INTERVALS FOR THE FOUR EXPERIMENTAL CONDITIONS (EXPERIMENT 3)

DISCUSSION

The results of Experiment 3 warrant the following conclusions. Whether the extraneous stimulus is the turning-on or turning-off of light appears to make no difference. This is true both for the inhibition of non-suppressed responding (pretest) and for the disinhibition of inhibited responding. With noise, however, turning-off was not a sufficient stimulus to produce either significant inhibition or significant disinhibition; conversely, turning-on of noise was a sufficient stimulus to produce both effects.

These findings correspond closely with the results of Kamin (1963) and Hilton and Kamin (1963) who found in classical CER conditioning, that light-on and light-off were equally effective as conditioned stimuli, but noise-on was far superior to noise-off. Thus, to at least some degree, the dimensions of the stimulus which control amount of conditioning appear also to control amount of disinhibition. This correspondence, however, need not argue for any "central" similarity between processes involved in conditioning and disinhibition; since both phenomena depend upon stimulus reception by the subject, the common factor may lie in a relatively peripheral sensitivity to stimulation. Put simply, the rat may not sense the change involved in turning noise off as well as it senses the change involved in turning noise on. Thus, noise-off would for any purpose be a relatively ineffective stimulus for the rat. Such a distinction between light and noise might in turn be attributable to a greater frequency of neural elements giving off-responses in the visual system than in the auditory system.

CHAPTER SIX

EXPERIMENT 4

The preceding experiment indicated that at least one stimulus dimension affects both conditioning and disinhibition similarly. Within Experiment 4, another stimulus dimension - intensity - is examined. Previous work (Kamin and Schaub, 1963; Kamin and Brimer, 1963) has already shown a direct monotonic function relating the conditioned emotional response in the rat to intensity of a white noise conditioned stimulus over the range 45-82 db. The present study explores the effects of varying the intensity of a white noise extraneous stimulus on amount of disinhibition.

There appears to have been little, if any, rigorous experimental work on intensity of a disinhibiting stimulus. Pavlov at one time clearly suggested that stimuli of "moderate" intensity produced the most disinhibition; "too weak", or "too strong" stimuli produced either less disinhibition or none (Pavlov, 1928, pp. 138, 211). This view corresponded to Pavlov's views on the effects of conditioned stimulus intensity, since he believed that conditioning was relatively ineffective when the CS was "too strong".

METHOD

Three new experimental groups (thirteen subjects per group) were trained for this experiment. The 13 subjects in Experiment 3, trained with an 80 db. noise-on stimulus, were included in the design. The training procedure for new groups was identical to that used with the 80 db. noise-on group of Experiment 3. The new groups were trained with white noise stimuli of 45, 65, and 100 db., respectively. Thus, there were four independent experimental groups with which to test the effect of the stimulus intensity parameter.

RESULTS

Pretest

The median pretest inflection ratios for the four noise intensities are presented in Figure 10. Table 5 gives means, medians, and ranges for pretest ratios for the first pretest trial and for ratios based on the means of trials 1 to 4.

Although there may be a tendency for the 100 db.noise to produce the most suppression, due to the considerable variability that exists within each of the four groups, no significant differences are found between the different intensities for either trial 1 or for trials 1 to 4. Considering the noise intensity groups collectively, a significant proportion of the subjects on trial 1 have ratios below .50 (41/52, $\underline{p} < .01$). Thus, white noise consistently inhibits ongoing response rate, but the magnitude of the disruption is minimally affected even by extreme differences in noise intensity.

Test for Disinhibition

Figure 11 presents the median number of stimulus and dummy responses on test trials 1 to 4 for the four noise intensity groups. The means, medians, and ranges for the difference scores are given in Table 6.

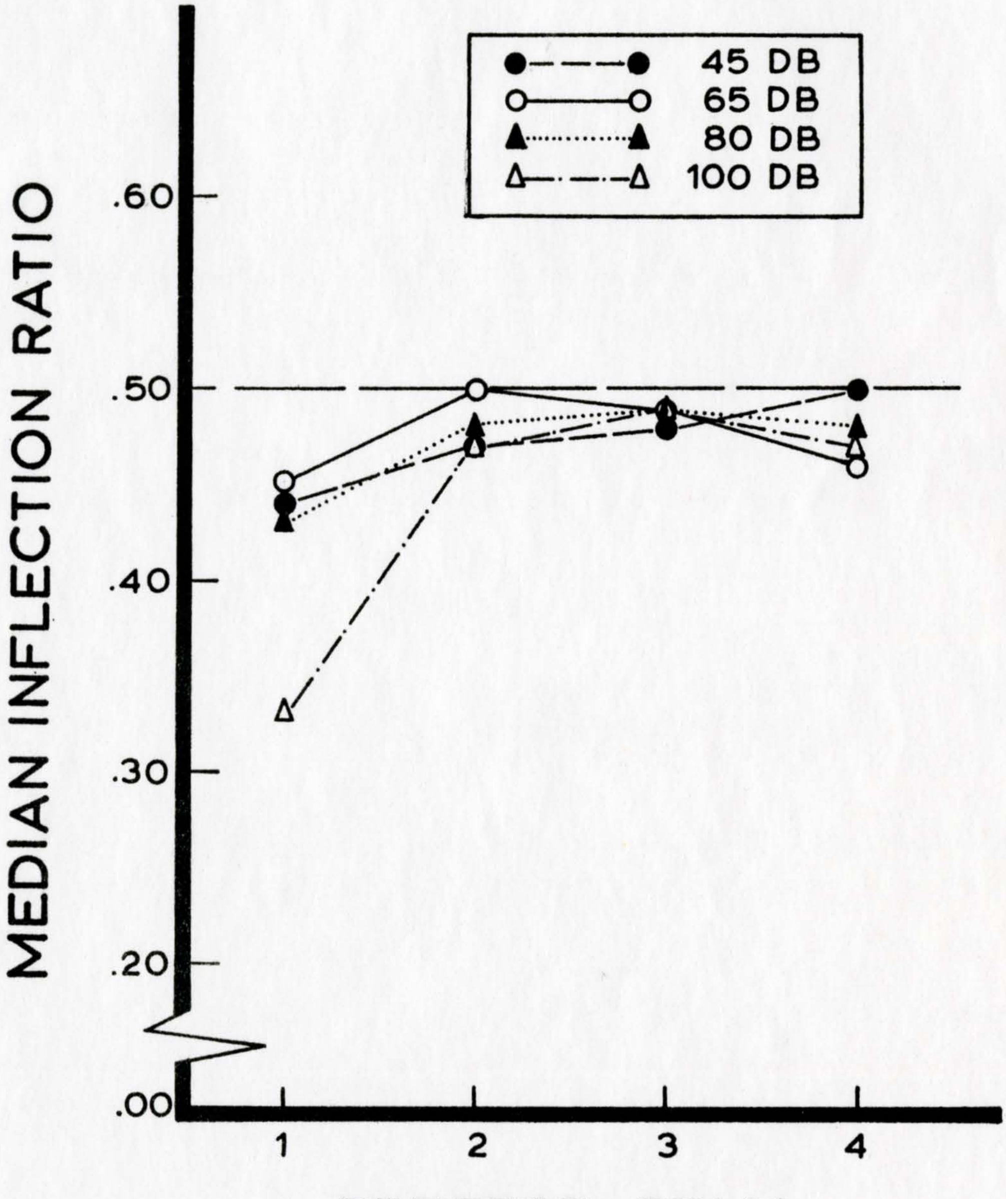

FIGURE 10. MEDIAN INFLECTION RATIOS AS A FUNCTION OF PRETEST TRIAL. PARAMETER IS NOISE INTENSITY. (EXPERIMENT 4)

TABLE 5

PRETEST INFLECTION RATIOS AS A FUNCTION OF STIMULUS INTENSITY (EXPERIMENT 4)

TRIAL 1

	NOISE INTENSITY (db.)			
	45	65	80	100
MEAN	.43	.46	.44	.32
MEDIAN	.44	.45	.43	.33
RANGE	.14 — .54	.35 — .63	.28 — .58	.09 — .58

TRIALS 1 - 4

	NOISE INTENSITY (db.)			
	45	65	80	100
MEAN	.42	.47	.48	.42
MEDIAN	.45	.47	.47	.45
RANGE	.25 — .55	.37 — .55	.39 — .56	.25 — .55

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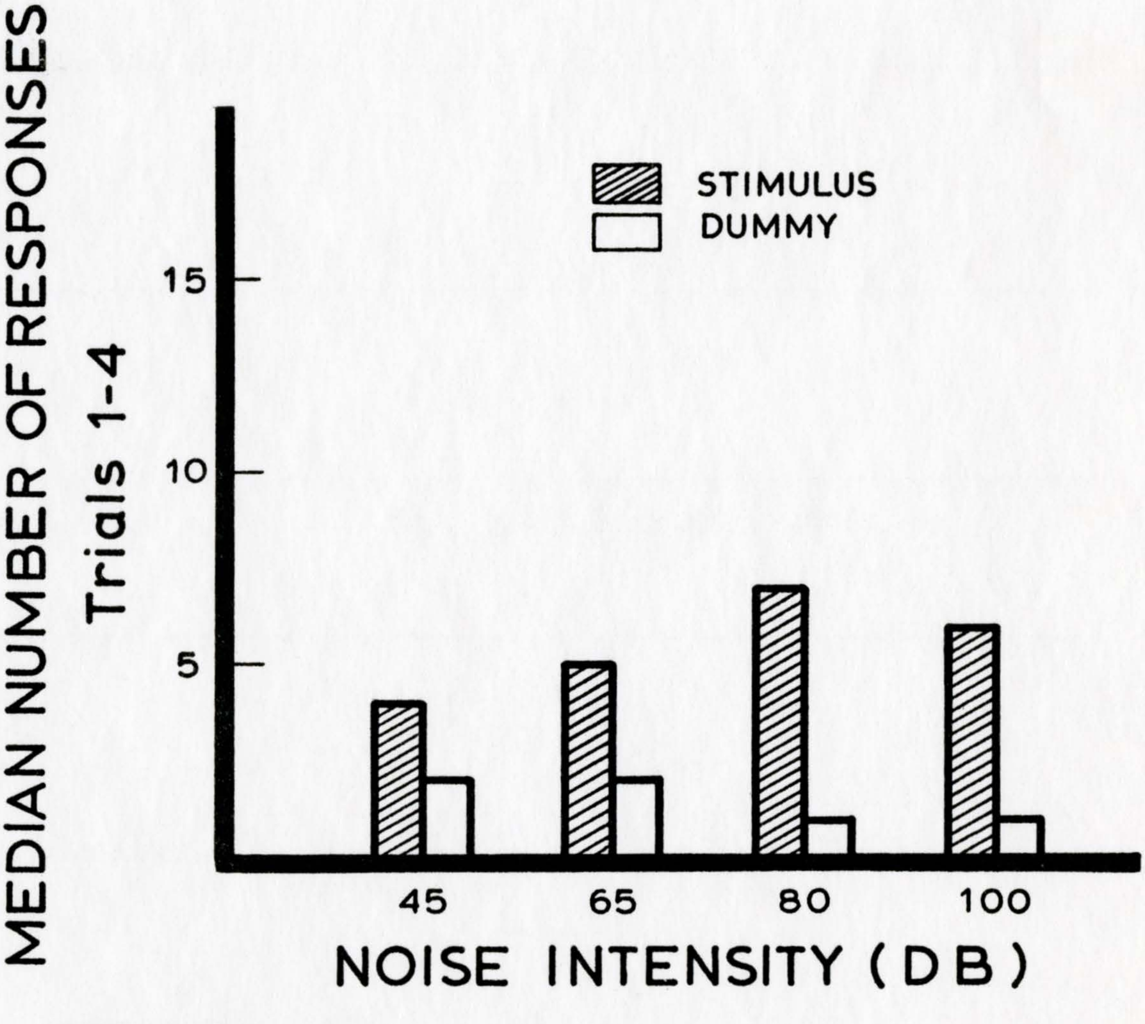

FIGURE 11. MEDIAN NUMBER OF RESPONSES (TRIALS 1 - 4) DURING STIMULUS AND DUMMY INTERVALS AS A FUNCTION OF NOISE INTENSITY (EXPERIMENT 4)

TABLE 6

STIMULUS-DUMMY DIFFERENCE SCORES AS A FUNCTION OF STIMULUS INTENSITY (EXPERIMENT 4)

TRIAL 1

	NOISE INTENSITY (db.)			
	45	65	80	100
MEAN	1.0	2.6	2.1	3.9
MEDIAN	1.0	2.0	1.0	4.0
RANGE	-6.0 — 5.0	0 — 9.0	-4.0 — 19.0	-5.0 — 19.0

TRIALS 1 - 4

	NOISE INTENSITY (db.)			
	45	65	80	100
MEAN	4.2	4.8	5.0	7.1
MEDIAN	4.0	5.0	5.0	5.0
RANGE	-6 — 19.0	-4.0 — 10.0	-4.0 — 14.0	-5.0 — 38.0

The data may suggest some slight tendency for difference scores to increase with stimulus intensity, but the differences between groups for trials 1 to 4 do not approach significance.($H=1.5$, $p > .50$). The possible relationship between noise intensity and disinhibition seemed more pronounced, however, on the _first_ test trial than over the entire four test days. This is made clear in Figure 12, where the median number of responses to the stimulus on the first test presentation is shown to increase progressively with stimulus intensity. The median animal does not respond at all during its first dummy interval. Figure 12 presents a very pretty picture. However, the difference scores between dummy and stimulus interval, are not significantly different between the four groups ($H=2.8$, $p > .30$).[1] The only way that one can demonstrate any significant difference is by separately analyzing the stimulus and dummy responses on trial 1. No differences exist between the experimental groups for the dummy responses ($H=2.5$, $.30 < p < .50$). However, if one-tailed probabilities are utilized with Ferguson's nonparametric trend test (1962), the number of responses during the stimulus increases with noise intensity ($S=219$, $p < .05$). Thus, after an extensive series of statistical gymnastics some tenuous evidence emerges to suggest that differences in amount of disinhibition _may_ be produced by differences in stimulus intensity.

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[1] Note that the median difference score is not identical to the difference between the median stimulus score and the median dummy score.

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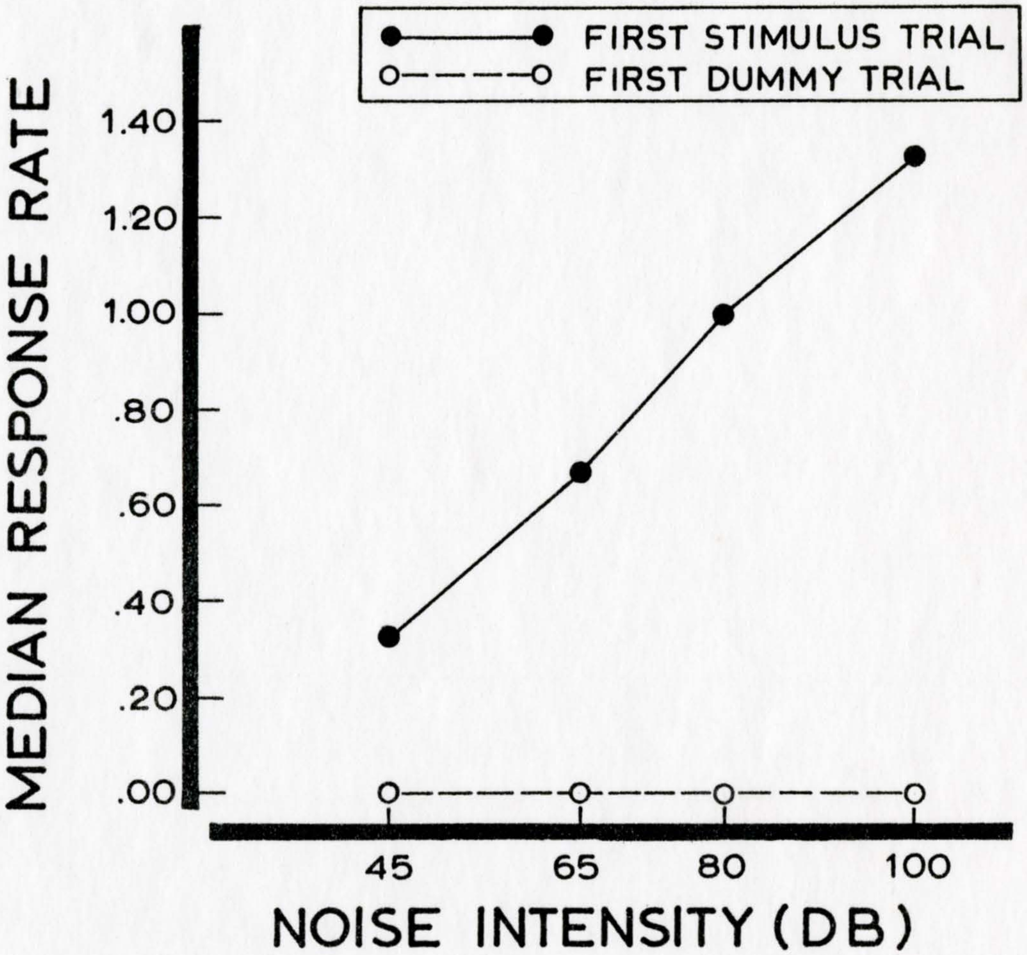

FIGURE 12. MEDIAN RATE (RESPONSES PER MINUTE) DURING STIMULUS AND DUMMY INTERVALS OF FIRST TRIAL AS A FUNCTION OF NOISE INTENSITY (EXPERIMENT 4)

Of course, over trials 1 to 4 a significant proportion of animals respond more during the stimulus than during the dummy intervals (44.5/52, $\underline{p} < .001$). If the four groups are considered individually, then a significant proportion of the subjects in each group, with the exception of the 45 db. intensity animals (10/13, $\underline{p} > .05$), display disinhibition. This distinction of the 45 db. group again may suggest some small effect of stimulus intensity, since 45 db. was the weakest intensity studied.

DISCUSSION

The obvious conclusion to be drawn from the fourth experiment is that both inhibition and disinhibition are relatively insensitive to stimulus intensity. There is some very slight suggestion that both inhibition on pretest and disinhibition on test may increase with noise intensity, but the striking fact is that over a range as vast as 45 db. to 100 db. the differences in the response rate changes are so minimal. The data also suggest that to the extent that any relationship exists between stimulus intensity and disinhibition the function is monotonic (cf. Figure 12). There is certainly no indication of a U-shaped function relating stimulus intensity to disinhibition, as suggested at one time by Pavlov.

The minimal differences which we have obtained by varying intensity are in marked contrast to CER conditioning, which is very sensitive to far smaller variations in CS intensity. For example, Kamin and Schaub (1963), employing a three minute white noise CS, obtained highly significantly different CER acquisition curves with intensity values of 49, 62.5, and 82 db. Conditioning was an increasing monotonic function of CS intensity.

This discrepancy between the effects of stimulus intensity on conditioning and on disinhibition encourages speculation that the stimulus plays very different roles in the two phenomena. Perhaps, since conditioning requires that the subject associate the onset of the CS with a subsequent event in time (the US), the role of CS intensity in conditioning is to provide a long-lasting neural "stimulus trace" to be contiguous in time with the US. This is consonant with observations by Kamin and Schaub (1963) and by Kamin and Gray (1963) that CS intensity is an overwhelmingly important variable in trace, as opposed to delayed, conditioning. This "time-bridging" function, of course, is not present in disinhibition, where only a single stimulus is involved. This kind of speculation is rather different from Pavlov's theorizing about "spreading cortical waves" of excitation related to stimulus intensity, and important in his view for both conditioning and disinhibition.

CHAPTER SEVEN

EXPERIMENT 5

The experiments previously reported make one fact eminently clear. The extraneous stimulus, which decreases the substantial baseline response rate prevalent on pretest days, increases the response rate at a later time, after baseline responding has been inhibited to a level close to zero. Therefore, the effect of the stimulus is obviously a function of the stage of training of the animal. Experiment 5 was designed to clarify the relationship between the amount of inhibition of the baseline response rate and the magnitude of the disinhibition effect. The aim of the experiment was to "map" the reaction to white noise at consecutive stages of extinction training, as the baseline response became progressively more inhibited. This involved a considerably different experimental procedure than had been previously employed.

METHOD

Preliminary Training

The preliminary training was basically the same as that in all the previous investiagations. After the initial training to bar press for food, there were six VI practice days. The last two of these days included, as before, four pretest stimulus presentations for all groups. The stimulus employed was white noise at an 80 db. intensity level. In this experiment, the house light in the Skinner box was on throughout all stages of training.

Inhibition Training and Disinhibition Testing

On the day following pretest, the inhibitory operation of extinction was introduced, and testing for disinhibition was carried out. The first response made by each animal on this day was reinforced by a food pellet, but no subsequent response was reinforced. All testing was done during the single experimental session which, on this day only, lasted two hours.[1] There were forty-eight new animals randomly assigned to five experimental groups. The experimental variable was the amount of extinction training received prior to testing. There were no inihibition training days prior to the testing day, and presentation of the test stimulus was not contingent on any response criterion. The test stimulus was presented instead at one of five fixed times; 3, 30, 60, 90, or 120 minutes after the start of the experimental session. Each of the five different experimental groups received the stimulus presentation at only one of these times.

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1. All previous sessions in this experiment had, as in previous experiments, been of one hour duration. An attempt to control for the discrepancy in time of testing vis-a-vis the previous day's session, was made by starting the first squad of animals three hours earlier than usual and then, within the limits of the experimental design, running an equal number of the experimental treatments at each two-hour interval throughout the day. Because of the fact that eight animals were run at a time, but there were only five experimental conditions,complete counterbalancing was impossible.

Responses were recorded for all groups for the 3-minute interval preceding stimulus onset and for the 3-minute interval of stimulus presentation. The measure of disinhibition in this study was an inflection ratio, which compared the response rate during the 3-minute stimulus to the immediately preceding rate.[1] However, response rate would presumably be steadily declining through a large part of the testing session, perhaps even in the course of a test trial. Therefore, inflection ratios computed for stimulus presentations had to be compared to dummy inflection ratios computed at the same stage of extinction for control animals which had <u>not</u> been presented with a stimulus. The 120 minute experimental group served as a control group for all other experimental subjects. Dummy ratios were computed for the 120 minute subjects at 3, 30, 60, and 90 minutes after beginning of the session. There were, of course, no dummy ratios available at 120 minutes. Thus, the comparison between stimulus and dummy ratios in this study, unlike in earlier experiments, is between, rather than within, subjects.

There were ten subjects in each of the 3, 60, and 120 minute groups, and nine subjects in each of the 30 and 90 minute groups.

RESULTS

Pretest

Table 7 presents means, medians, and ranges for the inflection ratios for each of the four pretest trials for the 48 subjects of the

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[1] This ratio is identical in form to that previously used to index inhibition on pretest trials; only now, values above, rather than below, .50 are expected.

TABLE 7

PRETEST INFLECTION RATIOS AS A FUNCTION OF PRETEST TRIAL (EXPERIMENT 5)

	PRETEST TRIAL			
	1	2	3	4
MEAN	.45	.49	.52	.49
MEDIAN	.46	.50	.52	.50
RANGE	.20 — .86	.31 — .64	.32 — .81	.27 — .73

experiment. The pattern observed with 80 db. noise in previous experiments is again evident. On the initial trial the stimulus produces a moderate suppression of response rate but this effect quickly "washes out" with continued testing. On the initial stimulus presentation a significant proportion of subjects have ratios below .50 (35/48, $\underline{p} < .01$) but this is no longer true by the second trial (25.5/48, $\underline{p} > .05$).

Test for Disinhibition

We had originally intended to analyze the results of this study solely in terms of inflection ratios. This, as will be seen, may give a misleading picture. We shall begin by analyzing inflection ratios, even though the apparent outcome of the experiment will be considerably modified by a subsequent analysis.

Figure 13 presents the median inflection ratio and the corresponding baseline response rate for both experimental and control groups at the different stages of extinction training which were tested. The baseline rate is calculated on the basis of the number of responses that occurred in the three minute pre-stimulus period. The baseline at each plotted point is based only on those animals for which corresponding inflection ratios are given. The stimulus inflection ratios and the corresponding baseline response rates, are for different experimental groups. The dummy inflection ratios and control baseline response rates are for a single group of subjects (the 120 minute experimental group). Whenever an animal failed to respond during <u>both</u> the 3-minute intervals (pre-stimulus and "stimulus") of <u>either</u> a stimulus or a dummy trial, it had to be excluded from this analysis since no inflection ratio could be

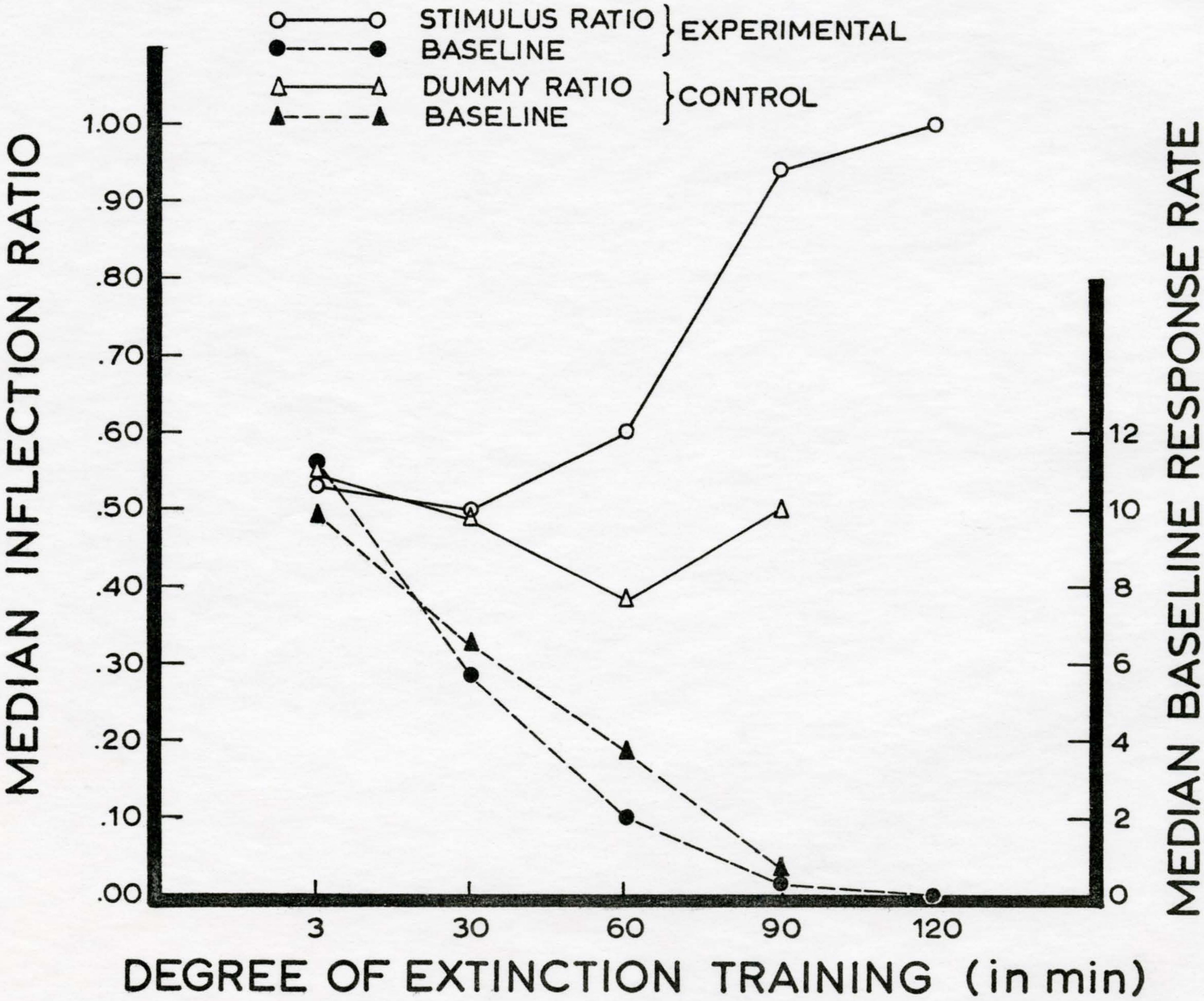

FIGURE 13. MEDIAN INFLECTION RATIOS AND MEDIAN BASELINE RATES (RESPONSES PER MINUTE) FOR EXPERIMENTAL AND CONTROL SUBJECTS, AS A FUNCTION OF DEGREE OF EXTINCTION TRAINING (EXPERIMENT 5) RATIOS ON LEFT-HAND AXIS, RATES ON RIGHT-HAND AXIS

computed. Table 8 presents the means, medians, ranges and _N_'s for the experimental and control groups at the different stages of extinction training.

It is obvious from Figure 13 that the baseline response rates for both the experimental and control subjects progressively decline as extinction training is extended, reaching a close to zero level by 90 minutes.[1] The inflection ratios for the experimental animals, on the other hand, show the opposite effect; viz., an increase with progressive extinction training. Finally, the dummy ratios for the control subjects tend to be low after 60 minutes of extinction training but otherwise do not deviate markedly from the indifference value of .50.

The point of major interest is the stage of training at which animals first show a reliable increase in responding during the presence of the noise. After 60 minutes of extinction experience (when the baseline rate is about 2 responses per minute) the stimulus ratios are significantly higher than are the dummy ratios ($U=10.5$, $p < .02$). It is at this stage of training, therefore, that disinhibition can first be demonstrated. With continued extinction training the response acceleration tendency as

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[1] Those animals that did not respond both prior to and during the test, are excluded from Figure 13. There were two control subjects excluded at 60 minutes, and three at 90 minutes of extinction training. Four experimental subjects were excluded at the 120 minute test. The inclusion of these non-responders would lower the control baseline rate slightly at the 60 and 90 minute test points. It would not affect the experimental curve, as the median response rate at 120 minutes for included subjects was already zero. It is clear that even without these possible corrections the extinction curves of baseline responding are quite comparable, whether based on comparisons of different groups at different stages of extinction, or plotted progressively for a single group.

TABLE 8

STIMULUS AND DUMMY INFLECTION RATIOS AS A FUNCTION OF DEGREE OF EXTINCTION TRAINING (EXPERIMENT 5)

STIMULUS RATIOS

	EXTINCTION (IN MINUTES)				
	3	30	60	90	120
MEAN	.54	.50	.59	.80	.92
MEDIAN	.53	.50	.60	.94	1.00
RANGE	.41 — .66	.40 — .68	.17 — .86	.47 — 1.00	.60 — 1.00
N	10	9	10	9	6

DUMMY RATIOS

	EXTINCTION (IN MINUTES)			
	3	30	60	90
MEAN	.60	.46	.37	.52
MEDIAN	.55	.49	.38	.50
RANGE	.41 — .81	.00 — .66	.00 — .52	.00 — 1.00
N	10	10	8	7

measured by the inflection ratio becomes more pronounced, while the baseline response rate continues to drop. After 90 minutes, the stimulus ratios are not quite significantly greater than after 60 minutes ($U=23.5$, $.05 \leq p < .10$), but after 120 minutes, the stimulus ratios are significantly greater than after 60 minutes ($U=5.0$, $p < .02$).

It should be noted that for the 30 minute group the baseline response rate (about 6 per minute) has dropped markedly from the level that this same group displayed at the start of the experimental session ($T=3.0$, $p < .05$). In spite of this degree of response inhibition, however, there is no suggestion of a tendency for the stimulus to produce an increase in response rate. Both the mean and median stimulus ratios are .50, and the stimulus and dummy ratios do not differ significantly ($U=42.5$, $p > .05$).

To this point we have omitted from the analysis those animals that did not respond during both the pre-stimulus and stimulus periods, because their inflection ratios were indeterminate. However, the omission of these subjects contributes to the impression given in Figure 13 that disinhibition increases monotonically over the latter half of extinction training. If we now adopt the position that all animals which fail to respond during the stimulus are to be considered as not showing disinhibition, then a different picture emerges for the last two tested points. We can now plot the proportion of animals showing disinhibition at each tested point, where disinhibition is defined as making more responses in the stimulus than in the pre-stimulus period. This has been done in Figure 14, which also depicts the proportion of control animals "disinhibiting". The control data is based, as before, on the 120 minute experimental group.

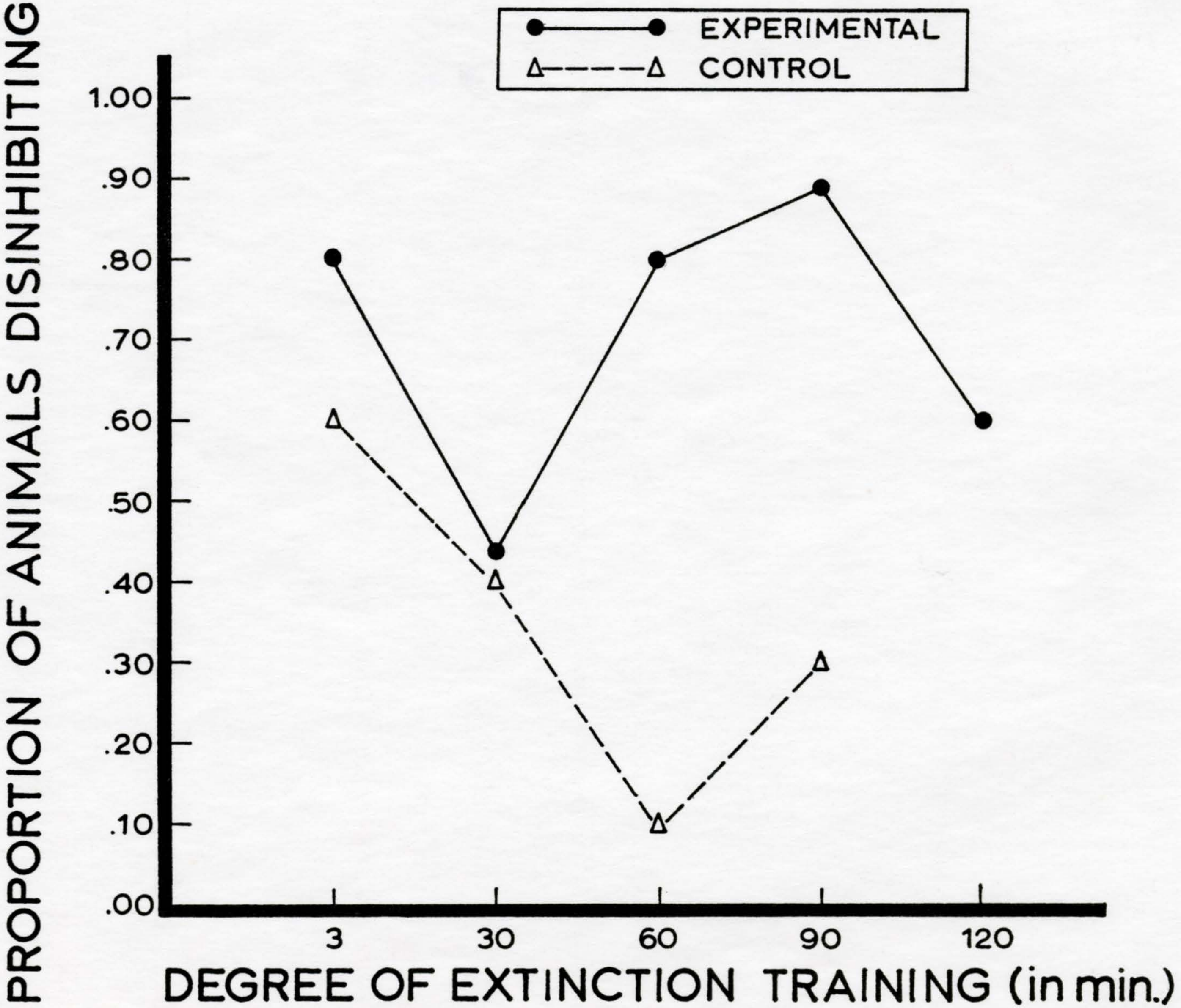

FIGURE 14. PROPORTION OF EXPERIMENTAL AND CONTROL ANIMALS "DISINHIBITING" AS A FUNCTION OF DEGREE OF EXTINCTION TRAINING (EXPERIMENT 5)

The crucial measure is now the difference in the proportions of "disinhibitors" between the experimental (stimulus) and control (dummy) groups. The proportions differ significantly at only two tested points - 60 and 90 minutes ($\underline{p} < .05$).[1] There are unfortunately no dummy scores for 120 minutes, so that no test can be made. The proportion of disinhibitors in the stimulus group at this time is, however, only 6/10, so it is unlikely that significant disinhibition could have been demonstrated.[2]

DISCUSSION

The attempt to "map" amount of disinhibition at successive stages of extinction training leaves us with some ambiguities. This much is clear. The baseline rate drops very significantly (to about 6 responses per minute after 30 minutes) without any measure giving any indication of disinhibition. With both of the measures we employed, significant disinhibition first appears after 60 minutes, when baseline rate has dropped to about 2 responses per minute. The amount of disinhibition

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[1] The tendency for both the experimental and control groups to accelerate responding at the start of the session (3 minutes) may be due to the motivating effects of non-reward as proposed by Amsel (1962). However, the effect is not statistically significant for the two groups considered either individually or collectively. Thus the relatively high proportions may simply be local irregularities. In regard to our primary interest, viz., disinhibition, the important point remains that there are no differences between the experimental and control groups at this early stage of testing.

[2] In order to have demonstrated significant disinhibition at 120 minutes it would have been necessary that none of the ten control subjects have a higher response rate during the test than during the pre-test interval. It seems highly improbable that this condition would have been met.

shown at subsequent points depends upon whether or not we include subjects with indeterminate inflection ratios. The notion that amount of disinhibition, as measured by the inflection ratio, increases throughout the extinction session could arise only by ignoring this class of subjects; and the proportion of animals falling into this class increases with stage of extinction.

Thus, the fairest summary seems to be this. If an animal does show disinhibition late in extinction, when baseline rate is extremely low, the nature of the ratio measure is such that the magnitude of disinhibition will loom very large.[1] However, the proportion of animals which show disinhibition at all seems to be at a maximum when the baseline rate is neither "too high" nor "too low".

We may, of course, be confronting a problem of "disinhibition below zero". We can conceive of competing tendencies, to bar press and <u>not</u> to bar press, summing algebraically within the animal to determine overt behavior. Then, when extinction training has driven the tendency <u>not</u> to bar press to an extreme value, an extraneous stimulus might produce a large increment in the tendency to bar press <u>without</u> resulting in an overt response. This argument essentially follows Pavlov in assuming that during extinction training an inhibitory process gradually counteracts an excitatory process, and that the dominance of the inhibitory process can continue to grow long after overt responding disappears. We might now assume that the increment in a tendency to respond produced by

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[1] That is, the value of the ratio will be large, when the baseline is low, even if number of responses to the stimulus is a constant for all values of baseline. This suggests that the inflection ratio may not be a very suitable measure for comparing groups with different baseline rates; it appears perfectly suitable, however, for parametric studies in which baseline rates do not differ among groups.

an extraneous stimulus is a constant at all levels of extinction training.

Put together, these assumptions provide for the failure of the disinhibition effect to appear empirically when extinction is too far advanced. They lead us, however, to new embarrassments. Thus, when extinction has already substantially depressed response rate (as after 30 minutes in the present study) the extraneous stimulus should increase the already dominant tendency to respond; detectable disinhibition should occur. This unhappily, does not in fact happen. Further, this theorizing has nothing to say about the suppressant effect of the same stimulus during pretest, before extinction.

We could, of course, simply assume that the extraneous stimulus gives rise to a process which counteracts whichever process (excitation or inhibition) is currently "dominant". This would force us to assert that, after 30 minutes of extinction, excitatory and inhibitory processes were close to an equal balance. This seems rather difficult, for it is not intuitively obvious that if both processes were balanced the animal would be bar pressing six times per minute. Perhaps, as Pavlov at times seems to imply, the effect of the extraneous stimulus depends upon the "relative dominance" of one process over the other. Since both processes are unobservable, and since "relative dominance" can continue to change after overt responding disappears, and since both the theoretical "relative dominance" and the empirical differences in response rates might be expressed either in terms of absolute differences or of ratios, any serious attempt to relate empirical disinhibition effects quantitatively to relative dominance of underlying processes seems out of the question.[1]

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[1] These same difficulties would apply to any theory which assumes underlying processes which are polar opposites.

The theoretical flight of fancy should not obscure the basic notion that baseline responding may sometimes be too inhibited for maximal disinhibition to occur. There is considerable empirical support for this idea. Within Experiment 5 there is an obvious suggestion that, when baseline rate is literally zero, many animals do not show disinhibition. Within Experiment 1, the greater disinhibition produced by the satiation, as compared to the extinction, treatment might be interpreted in much the same way. While it is true that the momentary rate at the time of testing was deliberately set at zero for all subjects in that study, it was also obvious that the basic baseline rate was higher during the testing sessions for satiation subjects. This was indicated by the significantly higher rates of the satiation subjects during dummy periods. The repeated observation that amount of disinhibition declines with repeated test trials may provide further evidence for the notion. Within Experiment 2, it appeared that amount of disinhibition was not affected by mere previous experience with the stimulus; and consecutive disinhibition test trials are, of course completely confounded with a declining basic baseline rate.

Thus, we might now postulate that disinhibition occurs when, and only when, the baseline response rate lies within a critical range of values - not too high, but not too low. The notion that disinhibition depends upon a critical value of baseline rate is theoretically very simple. Put this baldly, indeed, it is essentially an atheoretical statement. The notion, however, has some interesting consequences, to which the next (and final) experiment was addressed.

CHAPTER EIGHT

EXPERIMENT 6

The experiments already discussed contain a fundamental consistency. The same extraneous stimulus which, before inhibition training, produces a decrement in operant responding, later produces an increment in the same response. Thus, it seems perfectly valid to say that the stimulus has opposite effects upon non-inhibited and inhibited responses. The response, at the time of the final test, is known to be "inhibited" since we have deliberately applied some operation (experimental extinction, satiation, punishment, unsignalled shock) which we have observed to result in a diminished rate of operant responding. This type of finding, of course, produced the classical Pavlovian theory relating the effects of extraneous stimulation to excitatory and inhibitory cortical processes.

The fact that the "disinhibition effect" can be observed when inhibitory operations <u>other</u> than experimental extinction are applied poses some theoretical problems. Within Pavlovian theory, quite independently of the disinhibition phenomenon, other experimental data had led Pavlov to postulate that experimental extinction involved the accumulation of cortical inhibition.[1] We must now ask, what do our various "inhibitory operations", each of which produces disinhibition, have in common?

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[1] Two such phenomena, it will be remembered, were the facts that extinguished CR's exhibited "spontaneous recovery", and that an extinguished CS exerted an inhibitory effect on other reflexes.

Without any reference to "cortical dynamics", we can describe the outcomes of our own experiments in two very different ways. The first is to state that the disinhibition effect occurred when a response which once had a much higher probability of occurrence had been reduced to a much lower probability. Perhaps, then, disinhibition will be observed whenever any operation (excluding injury, death, etc.) accomplishes this kind of change in response probability over time.[1] Whether or not all such operations share with experimental extinction the production of "cortical inhibition" might be left for the Pavlovians to ponder.

There is, however, another way of describing the outcome of our experiments. We can state simply that disinhibition occurred whenever probability of the baseline response was at a <u>very low</u> level (but greater than zero).[2] This statement makes no reference to the fact that the

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1. Valenstein (1959) studying the CER in guinea pigs found that with sufficiently heavy dosages of reserpine, baseline lever-pressing dropped virtually to zero. When this occurred the presentation of the three-minute "clicker" CS resulted in a burst of responding. In a similar vein we once observed (Brimer, 1962) a sick rat whose baseline response rate "spontaneously" dropped over VI training days from approximately 21 responses per minute to approximately 1 response per minute. When the response rate was at this latter low level the presentation of a 3-minute white noise CS gave rise to an inflection ratio of .75, a three-fold increase in rate.

2. Harrison and Abelson (1959), studying escape training in rats with noise termination as the reinforcer, found that the baseline rate of responding which developed even after extensive practice was low (apparently in the neighbourhood of one or two responses per minute). The authors report (1959, p. 29-31) that for five of the six animals studied it was clear that the introduction of "mild novel stimuli" (opening the door of the experimental chamber, clapping the hands, etc.) was "invariably followed by a period of regular rapid responding at a rate <u>higher</u> than that given at other times in the animal's history".

probability of response was once at a higher level, although, as was the case with Pavlov's demonstrations, this was true of our experiments. We can thus postulate that the disinhibition effect will occur whenever the probability of baseline responding is at the requisite low level, even if it has never been at a higher level. Thus, in this view, the opposed effects of extraneous stimuli depend exclusively on the current probability of response: if the probability is high, the stimulus produces a decrement, but if the probability is low, an increment. This view leads to a paradoxical conclusion, which suggests that the disinhibition phenomenon may be very misleadingly named. For the observable property shared by those operations defined as "inhibitory" is that they produce a decline in the probability of responding of an animal whose behavior is under continuous observation. Therefore, to say that the disinhibition effect depends only upon a given probability of response - without reference to the probability at an earlier time - is to say that disinhibition can occur without there being any inhibition to disinhibit! We ought to be able, if the present view is correct, to produce a "disinhibition effect" without employing any inhibitory operation.

The several studies described as Experiment 6 had as their focus the attempt to demonstrate a disinhibition effect in animals with the requisite low probability of response. The major effort was to obtain animals which met this criterion at the time of test, and which had never previously had a higher probability of response. The animals must not have been subjected to any inhibitory operation. This obviously suggested exploring the early acquisition of the bar-pressing response. We hoped to be able to "intercept" animals for testing at some point early in acquisition, when the probability of response had risen from operant level to the requisite "low level".

Put most simply the studies of Experiment 6 attempted to distinguish between two alternatives: whether the sufficient condition for a disinhibition effect is merely a critical low probability of response, or whether it is a probability of response which has declined to the same critical low level.

The first problem we face, experimentally, is to estimate what, under our assumptions, is the requisite low level of response probability which produces disinhibition. The decision was made, in these studies, to employ an 80 db., 3 minute white noise as the extraneous stimulus. Thus we must ask what was the baseline probability of bar-pressing in our earlier experiments with this stimulus which produced disinhibition.

Within Experiments 1 to 4, which shared the same basic experimental procedure, the baseline rates during disinhibition testing were *very* low. These experiments, however, are not the best possible guide, since their design guaranteed that the baseline for the three minutes *preceding* the disinhibitory stimulus would be zero. The rate observed during the *dummy* intervals, it should be noted, was greater than zero. For the 41 animals in Experiments 1 to 4 which received the 3 minute 80 db. noise stimulus, the mean rate during the dummy interval on trial 1 (when significant disinhibition was observed) was 0.7 responses per minute; the median rate was zero.

Experiment 5, in which presentation of a disinhibiting stimulus was not made contingent on the animal's response rate, makes clear that disinhibition can occur when the response rates, over a 3-minute interval, are higher than the very low rates in Experiments 1 to 4. In Experiment 5, with 3 minute 80 db. white noise, significant disinhibition was observed after 60 and 90 minutes of extinction training. The median response rates for the control group at these two stages of training were 1.7 and 0.3; the mean rates, 2.1 and 0.7.[1] However, after 3 minutes and 30 minutes of extinction, with median rates of 10.7 and 8.7 (means of 12.6 and 7.8), there was no sign of the disinhibition effect. It thus seems clear that while 8 responses per minute represents a rate too high to demonstrate disinhibition, the effect can be shown with response rates up to at least 2 per minute.

What was ideally desired, then, was to "intercept" during early acquisition a rate in the neighbourhood of 2, and test the effect of an extraneous stimulus at this point. We would not introduce any operation that might be termed "inhibitory"; rather, we would try to intercept a progressively increasing response rate at the requisite low level.

Study A of Experiment 6 was designed simply to obtain information on how rate changes develop during early acquisition of the bar-pressing response. No extraneous stimulus was employed.

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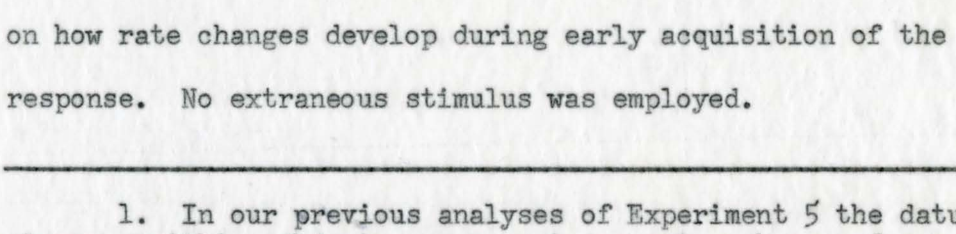
[1] In our previous analyses of Experiment 5 the datum employed was always a within-subject measurement assessing *change* of response rate. Thus, for example, the inflection ratio contrasted for each animal the number of responses in its 3-minute pre-stimulus interval to the number of responses in its 3-minute stimulus. This datum for experimental subjects was compared to the same datum for control subjects (whose "stimulus" was a dummy), to determine whether disinhibition occurred. In the present analysis the focus of attention is on the response rate, *per se*. Disinhibition can be demonstrated with this latter between-subject measurement by comparing the response rate of experimental animals during the 3-minute stimulus interval to the response rate of controls at *exactly the same* stage of training. The controls, of course, receive no stimulus presentation. Viewed diagrammatically the within-subject datum assesses disinhibition by comparing A':B' to A":B", the between-subject evaluation contrasts B" to B'. The same results are obtained in Experiment 5 employing *either* the between- or within-subject datum. The comparisons in Experiment 6, however, must of necessity be based on between-subject measurements. Thus, all of the cited control rate data from Experiment 5 is for the *B*" intervals.



STUDY A

METHOD

The general procedure adopted in the first study of Experiment 6, and employed in the two subsequent investigations, was as follows.

Animals were first put on a twenty-four hour feeding rhythm and reduced to about 75% of their ad lib body weight. The subjects were then introduced to the Skinner box, but <u>without</u> the response lever present, and given sixty "free" food pellets on a 1 minute variable interval schedule. This "magazine training" simply consisted of repeatedly presenting the audible magazine click followed by delivery of the food pellet. On the following day subjects were re-introduced to the Skinner box, where the response lever was now present and operative, and testing was carried out. Testing simply consisted of counting the number of continuously reinforced responses that were emitted in the <u>3-minute interval following some particular response criterion.</u>

There were 43 subjects in the first experiment, divided into four groups. The groups differed <u>only</u> with respect to the point at which the 3-minute interval during which responses were counted began. For different groups, the crucial count began after the animal had made 1, 3, 6, or 10 responses. Throughout Study A, all subjects were on a continuous reinforcement (CR) schedule for bar-pressing. That is, each bar press was followed immediately by both the magazine click and a food pellet. The numbers of subjects in the 1, 3, 6, and 10 response criterion groups were: 13, 9, 9, and 12.

RESULTS

Figure 15 presents the median response rate for the 3-minute interval following either 1, 3, 6, or 10 prior reinforced responses. Table 9 gives the means, medians, and ranges for the same data.

Of course, the effect of cumulative reinforcements is to progressively increase response rate ($S=372$, $p<.001$); the CR-1 animals have significantly lower response rates than either the CR-6 ($U=24$, $p<.05$) or the CR-10 ($U=13.5$, $p<.02$) subjects. Further, Figure 15 indicates that with a continuous reinforcement schedule, the response rate is asymptotic by the sixth bar press.

DISCUSSION

The critical question which Study A poses is whether, after one reinforcement, response rate is at the necessary low level for "disinhibition" testing. The rate is obviously higher than that in Experiments 1 to 4, and it is significantly higher ($U=31.5$, $p<.05$) than the 90 minute control group of Experiment 5, which showed maximal disinhibition. On the other hand, the rate is significantly lower ($U=28.5$, $p<.05$) than the 30 minute group of Experiment 5 which did not show disinhibition. Even though the median CR-1 rate is a little higher than that of the 60 minute subjects of the previous experiment (which did show disinhibition), there is no significant difference between the two groups ($U=48.5$, $.30<p<.40$).

Thus, although the response rate after one reinforcement is slightly higher than what would have been ideal, the level of responding is very close to a value which did produce disinhibition in a between-subject comparison in Experiment 5. At the other end of the scale, the rate after 10

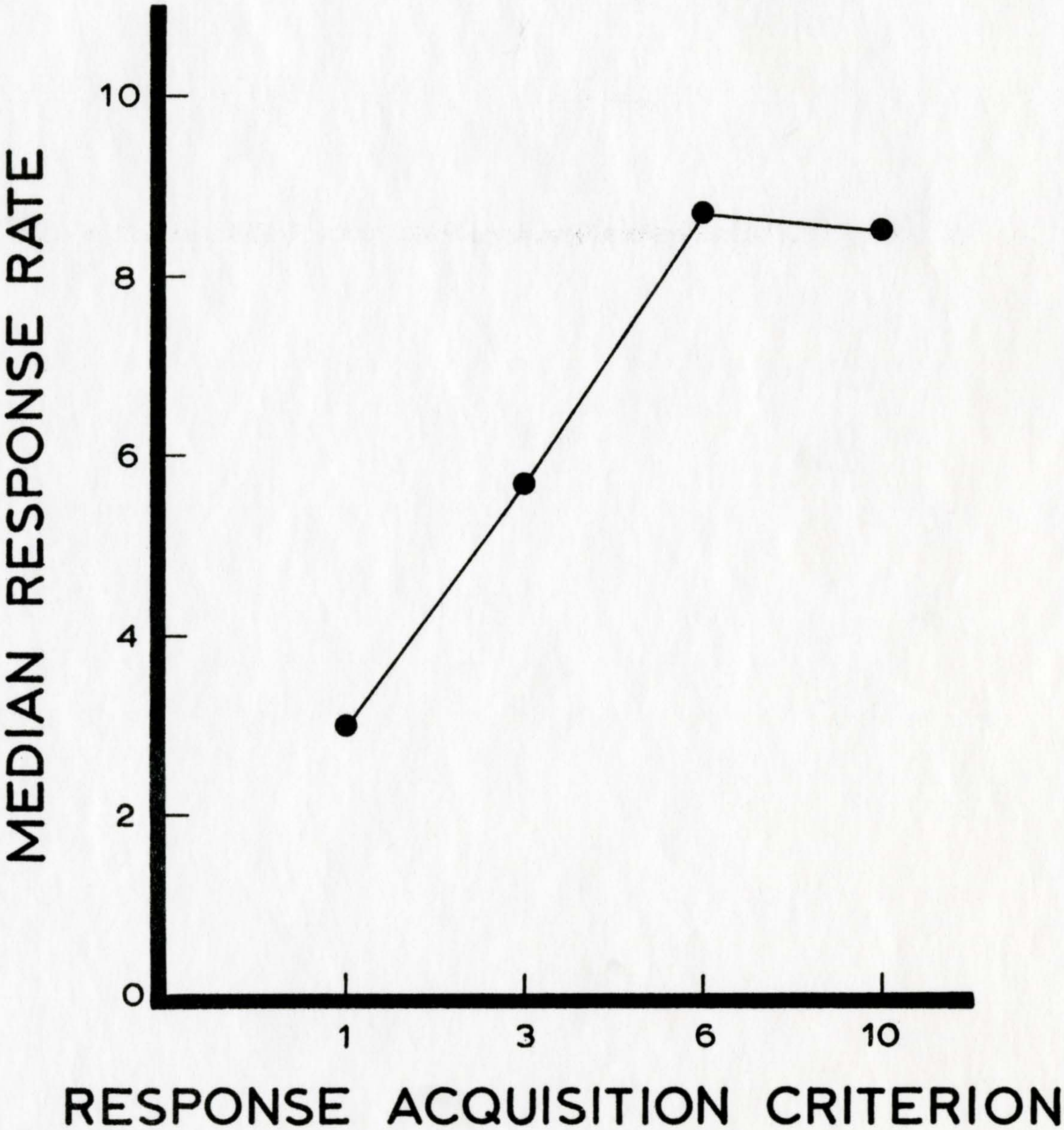


FIGURE 15. MEDIAN RATE (RESPONSES PER MINUTE) AS A FUNCTION OF RESPONSE ACQUISITION CRITERION (EXPERIMENT 6, STUDY A)

TABLE 9

RESPONSE RATES (PER MINUTE) FOR THREE-MINUTE PERIOD FOLLOWING CRITERIONAL RESPONSE (EXPERIMENT 6, STUDY A)

	CRITERIONAL RESPONSE			
	1	3	6	10
MEAN	3.6	6.0	7.3	9.0
MEDIAN	3.0	5.7	8.7	8.5
RANGE	0 — 8.7	2.7 — 12.7	.3 — 12.3	6.3 — 13.0

reinforcements is unquestionably outside the limits at which we have been able to produce disinhibition (the median is 8.5; the mean, 9.0 responses per minute).

Therefore, in the next study, we added two experimental groups which received the 3 minute noise stimulus immediately on making the first or the tenth reinforced response.

STUDY B

METHOD

In the second study there were two experimental groups of subjects, tested after either 1 or 10 responses. The procedure was identical to that previously employed, except that the noise stimulus was present throughout the 3-minute post-criterion interval. As in Study A, animals were on a continuous reinforcement schedule during the test. There were, in the present study, 13 subjects in the CR-1 experimental group and 14 in the CR-10 experimental group. In addition the 1-and 10-criterion groups of Study A were utilized as controls.

RESULTS

Figure 16 depicts the median response rate for the two experimental groups of the present study and the two corresponding control groups of the previous study. Table 10 gives the means, medians, and ranges for the response rates in the 3-minute post-criterion intervals.

It is clear,that after one reinforced response, there are no differences between the experimental and control groups ($U=77$, $p > .05$). The subjects receiving the test stimulus after the tenth response, however,

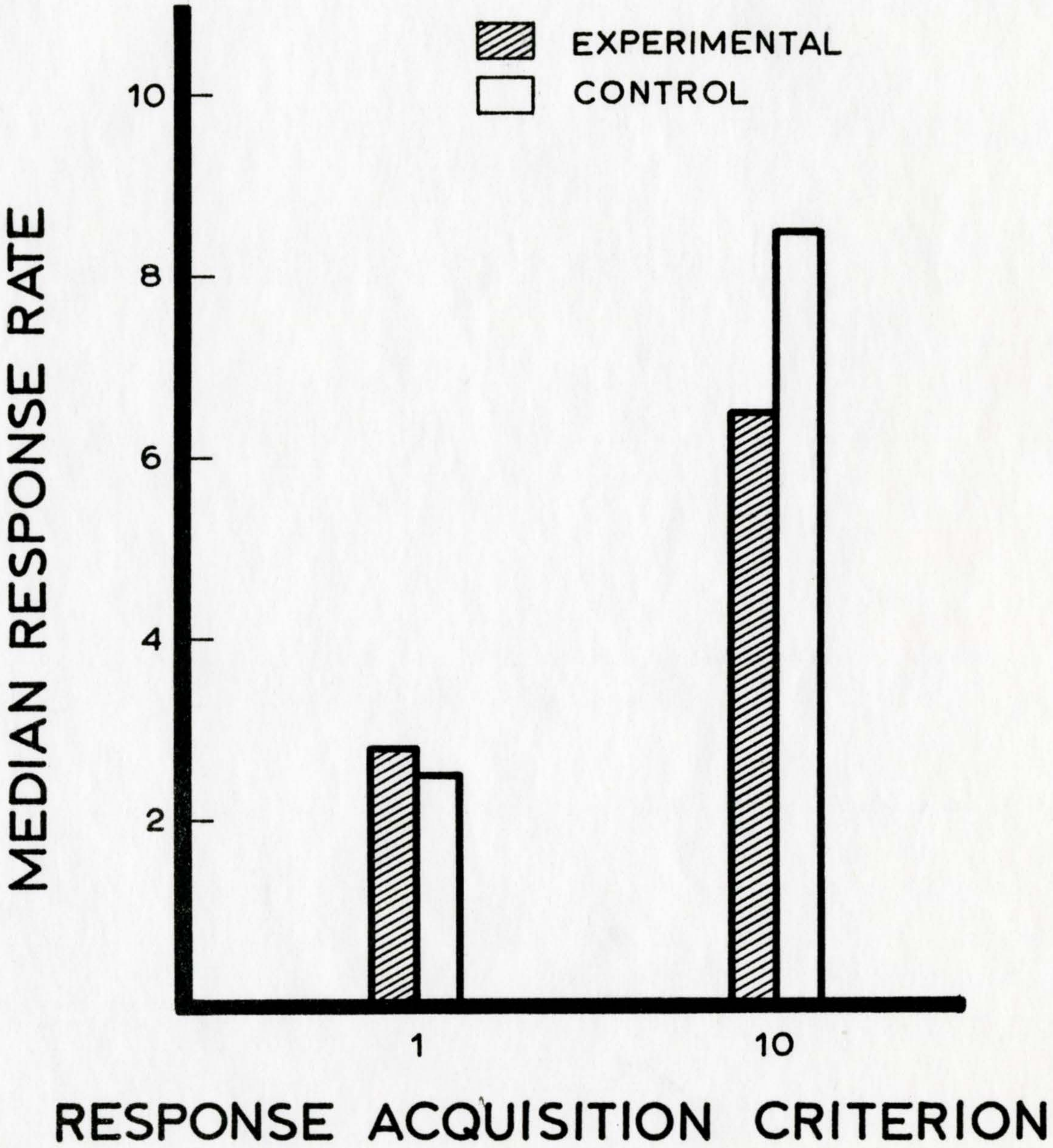


FIGURE 16. MEDIAN RATE (RESPONSES PER MINUTE) FOR EXPERIMENTAL AND CONTROL SUBJECTS, WITH RESPONSE CRITERIA OF 1 AND 10 (EXPERIMENT 6, STUDY B)

TABLE 10

EXPERIMENTAL AND CONTROL RESPONSE RATES (PER MINUTE) FOR THREE-MINUTE PERIOD FOLLOWING CRITERIONAL RESPONSE (EXPERIMENT 6, STUDY B)

EXPERIMENTAL

	CRITERIONAL RESPONSE	
	1	10
MEAN	3.1	6.8
MEDIAN	3.3	6.5
RANGE	0 — 7.3	1.7 — 11.3

CONTROL

	CRITERIONAL RESPONSE	
	1	10
MEAN	3.6	9.0
MEDIAN	3.0	8.5
RANGE	0 — 8.7	6.3 — 13.0

display a significantly lower response rate than the corresponding control animals (U=36, $p < .05$).

DISCUSSION

We find in this study that the noise stimulus does not produce "disinhibition" when the response rate is low, but, as might be expected, does produce inhibition, when the response rate is high. Thus, either a low response rate is not a sufficient condition for disinhibition, or possibly, the response rate after one reinforcement was not low enough. But, if the rate after one reinforcement is "too high", it is difficult to see how a lower rate could occur; obviously one reinforcement is the minimal condition for response acquisition.[1]

With the possibility in mind that the control response rate in Study B was too high to produce disinhibition, the final study attempted to produce a response rate below that observed in Study B. This was done by discontinuing reinforcement after the occurrence of the first reinforced response. The problem which this extinction procedure introduces is that an operation is employed that might be defined as "inhibitory". Under the particular experimental conditions adopted, however, the animal's response

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1. A number of pilot studies, which are not reported, were carried out in a series of unsuccessful attempts to secure a lower response rate during early acquisition. Two of the operations employed were; delaying the reinforcement of the first bar press, and satiating the animal prior to its bar-pressing session. Under these two conditions, the median response rates in the 3 minutes following the first reinforcement were 4.3 and 3.3. Thus the rates were not significantly different from the control rates in Study A and indeed, tended even to be slightly higher.

rate would never previously have been at a level higher than that in effect during the test. Thus, by our earlier definition of "inhibitory operation", it could be said that none was employed. We did expect, however, that the extinction operation employed would result in a lower response rate than what would have been observed, had the operation not been introduced. This, under a new definition, might be referred to as an "inhibitory operation".

STUDY C

METHOD

In Study C, all the subjects were tested after a response criterion of one, but only this first response was reinforced; subsequent bar presses produced neither the magazine click nor a food pellet. Thus, the subjects received extinction training during the 3-minute test interval. There were 18 subjects, divided into experimental and control groups of 9 subjects each. The experimental animals, in contrast to the controls, had 80 db. white noise presented throughout the test period. Response rates were recorded, as in the previous two studies, for the 3 minutes immediately following the criterion response.

RESULTS

Figure 17 presents the median response rates for the experimental and control groups of the present experiment, which were tested under an extinction procedure. The two corresponding groups from Studies A and B, which were tested under a continuous reinforcement schedule after meeting

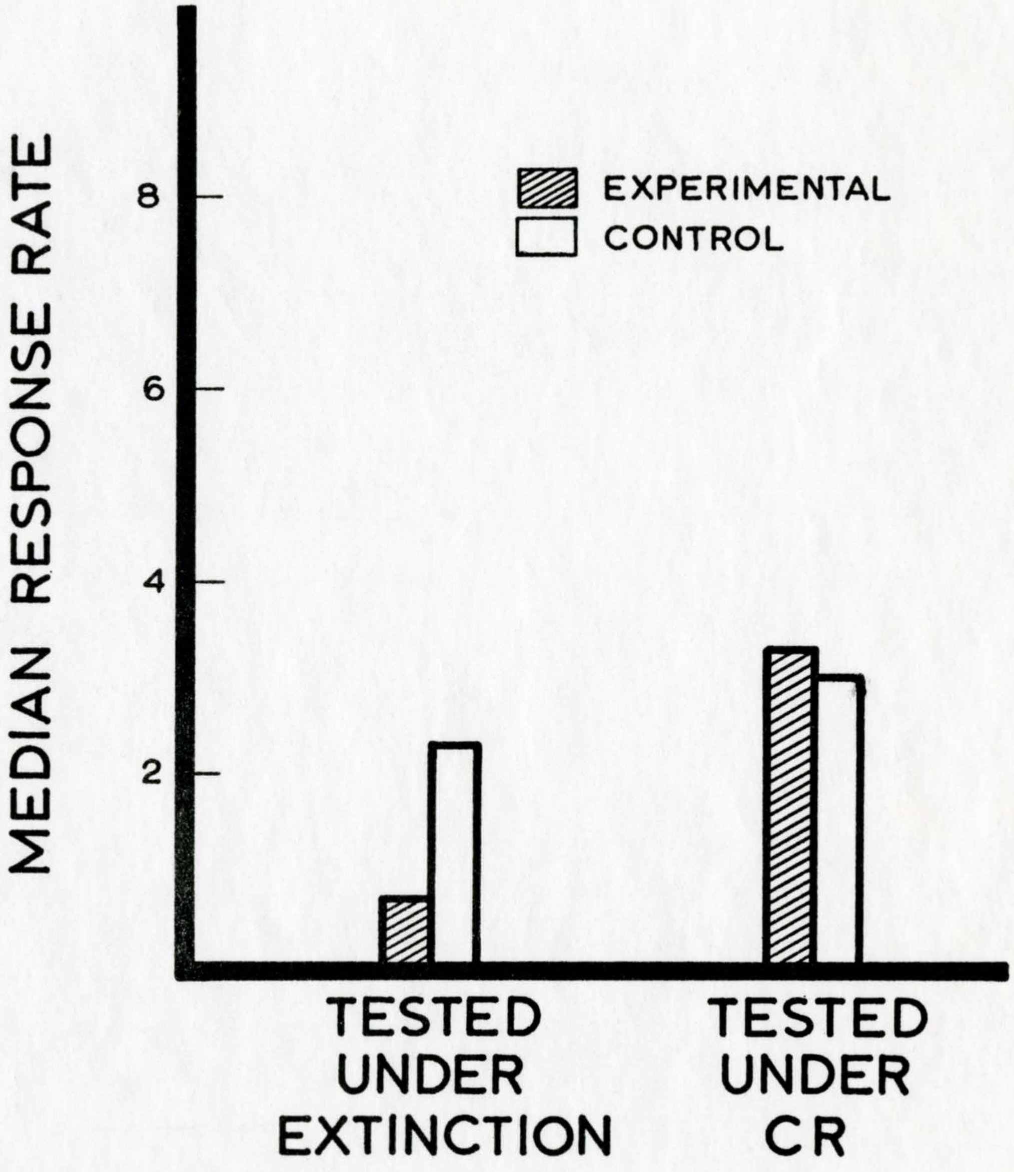

FIGURE 17. MEDIAN RATE (RESPONSES PER MINUTE) FOR EXPERIMENTAL AND CONTROL SUBJECTS TESTED UNDER EXTINCTION AND CONTINUOUS REINFORCEMENT SCHEDULES (EXPERIMENT 6, STUDY C)

the same response criterion, are also represented in the figure. Table 11 gives the means, medians, and ranges for the four groups. As is clear in Figure 17, within Study C, the extraneous stimulus had a marked inhibitory effect on response rate following one reinforcement ($U=12.5$, $p<.02$). Surprisingly, the control group of Study C did not differ from that of Study A ($U=48$, $p>.05$).

DISCUSSION

We find that in Study C, discontinuing reinforcement did not accomplish what was hoped for, viz., a lowering of response rate below the control level observed in Study A. This finding suggests that even a single reinforcement generates a considerable "reserve" of responses which are emitted in the following 3 minutes, regardless of whether the response continues to be reinforced. It is obvious that long-continued extinction, contrasted to long-continued reinforcement, would result in a lower response rate under the extinction than under the reinforcement condition; but such a difference cannot be demonstrated in the 3 minutes following the first reinforcement.[1]

We might argue that the response rate in Study C is still too high to provide a critical test. The extinction control animals of the present

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[1] Skinner (1938) long ago suggested that the first reinforcement might produce immediately the maximum response rate. Our data of Study A seems to contradict this, but the present study certainly supports the relative importance of the initial reinforcement, as it generates an impressively high level of responding, even in the absence of any further reward. The data of Study A might be interpreted, however, by assuming that a reinforcement either "works" or "doesn't work". If it works, it may produce the maximal rate immediately; and the more reinforcements that have been given to a particular animal, the more probable it is that one of them has "worked".

TABLE 11

EXPERIMENTAL AND CONTROL RESPONSE RATES (PER MINUTE) FOR THREE-MINUTE PERIOD FOLLOWING CRITERIONAL RESPONSE, UNDER EXTINCTION AND UNDER CONTINUOUS REINFORCEMENT (EXPERIMENT 6, STUDY C)

	EXTINCTION	
	EXPERIMENTAL	CONTROL
MEAN	.6	3.8
MEDIAN	.7	2.3
RANGE	0 — 1.7	.3 — 10.7

	CONTINUOUS REINFORCEMENT	
	EXPERIMENTAL	CONTROL
MEAN	3.1	3.6
MEDIAN	3.3	3.0
RANGE	0 — 7.3	0 — 8.7

study do not have a significantly lower rate than the 30 minute control group of Experiment 5, which failed to show disinhibition. But, on the other hand, the control group of the present study also does not differ significantly in rate from the 60 minute group of Experiment 5, which _did_ show disinhibition. The most impressive fact, however, is that an extraneous stimulus presented under the CR-1 extinction procedure, when the response rate was at least _relatively_ low, actually led to significant _inhibition_. Thus there is certainly no evidence to support the notion that a "low rate" is the sufficient condition to produce disinhibition. Perhaps, it is necessary that the response rate have been at one time at a higher level in order for the disinhibition phenomenon to occur.

The outcome of Study C presents us with a new problem. The response rates after one reinforced response are essentially the same whether or not extinction is introduced. In both instances the rates are relatively low. Why, we must now ask, does the stimulus have an inhibitory effect only under the extinction procedure? One possible answer would draw attention to the potency of the reinforcement variable. Continuing reinforcement during the test might well offset any basic inhibitory effect of the stimulus.[1]

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[1] Pretest trial inhibition in Experiments 1 to 5 was demonstrated while the rats worked under a $2\frac{1}{2}$ minute variable interval schedule. Thus, inhibition involved virtually no "cost" in terms of lost reinforcements. We must note, however, that in Study B rats working under a continuous reinforcement schedule did show inhibition after meeting a criterion of 10 reinforced responses. The control rate after 10 reinforcements, however, was very high; the statistically significant inhibition shown by the experimental subjects involved "giving up" only a small percentage of the reinforcements which would otherwise have been obtained. For continuously reinforced subjects tested for inhibition after meeting a 1 response criterion, _any_ lowering of response rate would have involved giving up a very large proportion of the reinforcements which would otherwise have been earned.

Perhaps the basic effect of an extraneous stimulus is always inhibitory, regardless of the baseline probability of response. The only exception to this possible rule in any of our data is the case where response probability is low, having once been at a higher level. This exception, of course, constitutes the phenomenon of disinhibition. The weight of the evidence seems to support the view that a "low rate" alone is not sufficient to produce disinhibition.

We have failed thus far to produce disinhibition when the response rate in acquisition is relatively low, without its once having been higher. The argument can always be made, however, that the rates actually obtained in Experiment 6 were not low enough. To guarantee obtaining a very low response rate we could utilize the "operant level".[1] Thus, in a final gesture, 7 rats were simply placed into the Skinner box, (with no prior magazine training) and after the first response, noise was presented for three minutes. No response was ever reinforced. The mean and median response rates during the 3-minute test interval were .4 and 0. This rate in a group of seven subjects, is not significantly greater than zero, and is in fact lower (though not significantly) than that of a control group of nine subjects which were similarly treated, but which received no noise presentation. Thus, once again, a very low probability of response does not in itself seem to be a sufficient condition to produce disinhibition.

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1. The operant rate is simply the number of responses that occur in the absence of any known reinforcement contingency. It would naturally be expected that such a rate would be very low. The response probability must be something greater than zero, or acquisition could never occur; the response must first be emitted in order to be reinforced.

CHAPTER NINE

DISCUSSION

The experiments which we have described have served to make clear a number of empirical facts with which any adequate theoretical interpretation of the effects of extraneous stimuli on behavior will have to deal. We shall briefly review these facts, before attempting a theoretical integration.

The results of the first experiment clearly demonstrated that the augmentation of response referred to by Pavlov as "disinhibition" may be reliably observed in the operant bar-pressing setting. The phenomenon appeared to be highly general. Extraneous stimuli of two different sensory modalities (visual and auditory), and of two different durations ($1\frac{1}{2}$ seconds and 3 minutes), increased responding which had been suppressed by two different types of "inhibitory operation" (extinction and food satiation). Further, even when responding was punished, an acceleration in responding took place during the presence of the extraneous stimulus.

The extraneous stimuli which, when presented during extinction, <u>increased</u> the response rate, had just the opposite effect when presented on pretest day, before any inhibitory operation was introduced. They reliably <u>decreased</u> the response rate on pretest day. Further, as Pavlov had suggested, there was a relationship between the magnitude of the inhibitory and disinhibitory effects. The relationship which we observed, however, was a simple one, rather than the curvilinear function suggested by Pavlov. The stimuli which were the more effective response inhibitors tended also to be the more potent disinhibitors.

The first experiment also made clear, since a 1½ second stimulus produced disinhibition, that the disinhibitory effect could manifest itself after termination of the extraneous stimulus.

We next examined in more detail some characteristics both of the "extraneous stimulus" and of the "inhibited response" that are involved in the disinhibition phenomenon.

The results of Experiment 2 revealed that varying the number of exposures to the extraneous stimulus prior to testing had substantially no effect on the disinhibition displayed. Thus we concluded that familiarity with the stimulus is relatively unimportant in the disinhibition phenomenon. This conclusion in turn suggests that the decrement in the disinhibition effect that was observed with repeated disinhibition testing in all of the first four experiments may be due to the growing level of inhibition of the response, rather than to increasing familiarity with, or adaptation to, the testing stimulus.

The inhibitory effect observed on pretest day, however, diminished rapidly with repeated testing, without any correlated change in the baseline response rate. With noise, for example, reliable response suppression was <u>never</u> observed in any of the experiments after the second pretest trial. Thus, if we identify the inhibitory effect with Pavlov's "orienting reflex", we must conclude that the orienting reflex adapts rapidly with repeated presentations of the stimulus.[1] Within Experiment 2, animals were tested for disinhibition with and without prior exposure to the extraneous stimulus. The fact that the various groups did not differ in amount of disinhibition displayed suggests that the disinhibitory effect can occur even when, as was presumably the case, the completely novel stimulus employed on the first disinhibition test trial elicits a concurrent orienting reflex.

Experiment 3 indicated that the relative effectiveness of turning a stimulus *on* or *off* varied with the stimulus modality. There was no difference between a light turned on and one turned off, in terms of amount of either inhibition or disinhibition. On the other hand noise-off, in distinction to noise-on, tended to be relatively ineffective both as an inhibitor and as a disinhibitor. These results closely paralleled findings by Kamin in CER conditioning. This need not, however, argue for any "central" similarity between conditioning and disinhibition, but may simply reflect the rat's "peripheral" sensitivity to different sensory inputs.

Although there was some slight suggestion in Experiment 4 that both inhibition and disinhibition increased with noise intensity, the most striking aspect of the data was the fact that the two effects were so relatively insensitive to the substantial differences in stimulus

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[1] We could not directly observe the behavior of our rats, which were trained in a completely dark box. The problem of direct observation in experiments of this sort is in any event difficult. The mere presence of the observer provides a confounding source of "extraneous stimulation".

intensity that were employed. This finding was in marked contrast to CER conditioning, where far smaller differences in intensity of a noise CS give rise to conspicuous differences in conditioning. Thus, in distinction to Experiment 3, a lack of parallelism between disinhibition and conditioning was revealed in Experiment 4. The results of Experiment 4 seem to suggest, unlike Pavlov, that conditioning and disinhibition are not identical in terms of their underlying processes. The noise intensity function in conditioning may derive from the fact that in that case stimulus events must be "bridged" in time. The more intense stimulus may thus facilitate conditioning through providing a longer-lasting "trace" of the CS, to be associated with the US. Disinhibition, on the other hand, seems more nearly an all-or-none phenomenon, with respect to intensity of the disinhibiting stimulus. Providing the extraneous stimulus is above some critical threshold, disinhibition appears to occur at about its maximal level.

The first four experiments demonstrated that an extraneous stimulus would increase response rate when response probability was at a very low level. When "inhibition" is defined simply as referring to an observed decrease in response rate, this amounts to saying that the first four experiments all involved a substantial degree of inhibition prior to the test for disinhibition. Thus, in Experiment 5, we asked how degree of disinhibition varied with the amount of inhibition of the response. This meant, empirically, holding constant the degree of previous acquisition of bar pressing, while varying the degree of extinction training (and thus the baseline response probability) prior to disinhibition testing. The results suggested that disinhibition only occurred when responding had been reduced to some critically low value. On the one hand, response rate could be substantially reduced without any accompanying tendency for the rate to increase in the presence of the extraneous stimulus. On the other hand, with the continuation of extinction "below zero", the evidence suggested that animals would also fail to respond to the stimulus. It was only at a particular low response rate (under the conditions of Experiment 5, a rate of about two responses per minute) that the response acceleration tendency was clearly evident. This notion of a "critical" response probability can be utilized to explain both the decreasing amount of disinhibition with repeated testing during extinction, and the large amount of disinhibition shown by food-satiation subjects in Experiment 1.

The final series of experiments sought to determine whether or not the critically low response probability previously referred to was the *sufficient* condition for the disinhibition effect. The experimental evidence, viewed in total, offered no support whatever to the idea that a low response probability was the sufficient condition for disinhibition; it suggested instead that the response rate must be *reduced* to the requisite low level from a previously higher value. Thus, without endowing the term "inhibition" with the quasi-neurophysiological properties assigned to it by Pavlov, we finally seem to agree with him that the acceleration in responding produced by extraneous stimuli during extinction is a case of "*dis*-inhibition". Whenever we presented the extraneous stimulus to animals with the requisite low response rate - which had never been at a higher level - we observed either no effect, or actually a *decrement* in rate of responding.

The only serious attempt to develop a theory accounting for the opposed effects of an extraneous stimulus during acquisition and extinction has been Pavlov's. We have already seen how his effort to incorporate the facts of conditioning, of extinction, and of extraneous stimulation within a single system failed. The theory of "cortical functioning" elaborated into a web of polar variables, capable of postdicting any empirical outcome. The Pavlovian theory at the same time generated mutually contradictory conclusions. We must ask, however, whether any other available psychological theory *can* incorporate the facts. We state at the outset our conviction that no present theory can do this, but we shall examine briefly the deficiencies of available formulations.

We can describe as "single factor" theories those which attempt to explain some part of the relevant phenomena by attributing only *one* function to the extraneous stimulus. The crudest form of single factor theory can be speedily dismissed. Thus, Wendt, momentarily ignoring the empirical phenomenon of disinhibition, attributed the empirical phenomenon of inhibition to elicitation by the stimulus of a response incompatible with the CR; while Skinner, momentarily ignoring inhibition, attributed disinhibition to a "facilitating" effect of the stimulus! This particular game comes to an ignominious end when the two theorists meet head-on; neither has attempted to deal with all the facts, and each postulates a mechanism which contradicts the ignored facts.

There may, however, be some hope for a single factor theory which postulates an interaction between a constant effect of the stimulus on the organism, and the organism's momentary state, in determining the behavioral outcome. That is, such a theory would assert that, though the effect on a specified behavior may vary with the animal's state, at some more "central" level the stimulus has an invariant effect. The most obvious candidate for such a theory is the "level of arousal" or "level of activation" formulation

(Duffy, 1957; Malmo, 1959).[1] The essence of this notion is that there is an "optimum" level of arousal or activation for any particular task; "... the relation between activation and behavioral efficiency ... is described by an inverted U curve" (Malmo, 1959, p. 368). The theory asserts further that "... activation level is in large part a function of environmental stimulating conditions" (Malmo, 1959, p. 374).

Thus, we might assert that an extraneous stimulus _always_ increases the animal's "level of arousal", but that the effect of this increase on _behavior_ would depend on the animal's level of arousal at the moment the stimulus is delivered. Perhaps, e.g., the animal is already optimally aroused on pretest day, and at a very low level of arousal during extinction; in this way bar pressing might be appropriately impeded, or facilitated. The most obvious difficulty with invoking arousal theory in the present context is the absence of any independent measurement of the animal's arousal level. Further, some of the behavioral facts seem embarrassing. Why should not the stimulus "over-arouse" on pretest trials following the first presentation? Why, early in extinction, when the arousal level has presumably dropped sufficiently to produce a substantial lowering of response rate, does the stimulus have no detectable effect? We should have finally to make the implausible assumption that, when the animal has received either free or response-contingent shock, he is so _under_-aroused that the stimulus will have facilitating effects!

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[1] The concept of arousal or activation is roughly comparable to that of general "drive" in learning theory. It is usually assumed, however, that arousal may be directly measured by some physiological index. Malmo has suggested that the central mechanism of activation is the ascending reticular system.

The kinds of physiological reactions characteristically used to index arousal (e.g., increased heart rate) are known to be elicited by electric shock.

We could, of course, invent a single convenient "function" for the extraneous stimulus - that of facilitating whichever is the weaker of two opposing "tendencies". Whether we name the tendencies "competing responses", or "intentions", or "excitation vs. inhibition", we should have to assume that two competing tendencies are operative *both* during acquisition and during extinction, with a different one predominant at each stage. This is not wholly implausible; the animal may be "in conflict" about whether to press the bar, or whether *not* to press the bar, both during acquisition and extinction. The problem again lies in the lack of any independent measure of the opposed tendencies. We see only their *resultant* - the rate at which the animal in fact presses the bar. We should note also that the convenient function of facilitating the *weaker* of two tendencies appears to be in direct opposition to the most fully developed of relevant psychological theories (Hull, 1951). Within Hullian theory, the extraneous stimulus in such a situation might presumably act like "irrelevant drive", contributing toward the total of "generalized drive". The generalized drive in turn multiplies the "habit strengths" of *all* existing response tendencies in determining behavior. The response most facilitated would thus be that with the *higher* strength before introduction of the extraneous stimulus!

With so little comfort derived from single factor theories, we can now examine a class of "two factor" theories. While no such theory has in fact been proposed, any such theory would assign *two* separable functions to an extraneous stimulus; a "distracting" function to account for the effect during acquisition, and some other function to account for the opposite effect during extinction. To any such theory there must be appended some explanation of why one of these functions predominates during acquisition, and the other during extinction.

We can deal first with the "distracting" function. This can reasonably be identified with Pavlov's "orienting reflex", or, much the same thing, with Bindra's (1959) "novelty reactions". These notions involve the simple proposition, which has substantial empirical support, that a novel or unexpected stimulus elicits "orienting" or investigatory" reactions. These in turn might well be incompatible with the conditioned response. We should note that such orienting reactions are known to extinguish rapidly, and this is consistent with the fact that with the noise stimulus, we detected observable interference with the response only on the <u>first</u> pretest trial.

But what now of the second, facilitating function? We might try to place the burden on an associative, or "cueing" function of the stimulus. The stimulus, in most of our experiments, was temporally associated (on pretest days) with food reinforcement for bar pressing. Perhaps this association between stimulus and food-for-bar-pressing is responsible for the resumption of bar pressing when the stimulus is now presented during extinction. This, however, cannot explain why the stimulus had a facilitating effect on animals which had <u>never</u> previously experienced it. Perhaps, on the other hand, the important fact is that every other aspect of the experimental situation <u>but</u> the stimulus has been associated with non-reinforcement. Thus, adding a new element to the extinction situation might produce a "generalization decrement" of <u>extinction</u>, permitting bar pressing to occur. The problem here is the facilitating effect of the stimulus on food-satiated animals, for whom bar pressing has *not* been associated with non-reinforcement. Thus, an associative interpretation of the effects of the stimulus during extinction seems wholly inadequate.

We are left, finally, with an attempt to combine some variant of a Hullian "generalized drive" facilitating effect with the distracting effect, in a two-factor theory. We might argue that there is only one relevant response tendency - the tendency to bar press. We now ignore the possibility of a competing tendency *not* to bar press (or of "inhibitory processes" in general), and focus solely on the existing tendency to bar press, as indexed by the observed rate of bar pressing. We can assert that, in *addition* to exerting a distracting effect which will impede bar pressing, any extraneous stimulus also "energizes" bar pressing. Why, however, should sometimes one and sometimes the other effect predominate?

The fact that a decremental effect was observed only on the first pre-test trial can be attributed to the rapid diminution of the "distracting" property of the stimulus. We might also argue that no incremental effect was observed on subsequent pretest trials because the response was already at asymptotic strength. We should note that the failure to detect a decremental effect in animals which were presented with the novel stimulus for the *first* time during extinction is not a telling blow against this formulation. There is no logical reason why *both* a distracting effect (orienting reflex) *and* a substantial energizing effect could not occur consecutively within the three-minute time span during which we measured bar pressing. The repeated observation in early studies of disinhibition that a facilitative effect often occurs some considerable time *after* termination of the stimulus in fact encourages speculation that the *first* reaction to the stimulus is an impeding "orienting reaction". Thus, the net total of two consecutive effects might well have been, as the data would force us to assert, facilitative.

To this point, by stringing together a number of not wholly implausible assumptions, we have managed in our latest theoretical effort to post-dict part of the data. This effort, however, must also come to grief at at least two points. There is no obvious reason why the net effect of a stimulus presented early in acquisition (Experiment 6) should not have been facilitative, rather than decremental. Then, we must finally ask, why did not the stimulus have a facilitating effect after 30 minutes of extinction in Experiment 5? The critical fact here is that, at this point, the response was shown to be significantly below its asymptotic strength, and there was considerable "room" for facilitation. The stimulus, further, was not novel for these animals, which had been pre-tested.

Thus, we must reluctantly abandon the attempt to fit the facts to existing theories, or even to esoteric combinations of selected parts of existing theories. The major stumbling block for all theoretical efforts is the paradox to which Pavlov first called attention: why does the same stimulus have different effects at different stages of training?

We turn finally to one last empirical scan of the data. What, after all, do all the cases of "disinhibition" which we have demonstrated have in common? The irreducible minimum derivable from our experiments seems to be this: disinhibition occurred only when a response which was once at a higher level of probability had been reduced to a "critical" lower level of probability. What further empirical questions might we ask in new experiments? We could at least ask whether the level from which the response probability is reduced is a relevant variable. That is, we might test two groups of animals at that point in extinction when the response rate of each is about two per minute; but one group of animals would have been previously trained to a much higher rate of responding than the other. We could also test animals with previous histories of two separate responses, each of which, after original acquisition, had been reduced to the same critically low level of response probability. The critical test would be made when the manipulanda for both responses were available to the animal. Which response would be "disinhibited" by an extraneous stimulus -- the first learned, or the last learned? Were both to be learned before either was extinguished, which would be disinhibited - the first or the last extinguished? Would amount of disinhibition vary with the time interval (during which no experimental treatment was given) between extinction of a response to the critical level, and the subsequent test? Perhaps a series of such empirical studies, asking fresher questions in newer contexts, would provide enough answers to enable more informed guesses as to the processes underlying the disinhibition phenomenon.

The phenomenon is certainly real, and reliably obtainable in the vast majority of subjects. Thus it is certainly susceptible to continued parametric study, toward which the present experiments have attempted to contribute. Possibly the effect is attributable to a heterogeneous concatenation of processes already understood, but this has not been demonstrated. Possibly again, a new theory arising out of the study of disinhibition might be of major importance for behavior theory. The events subsumed under such terms as inhibition, conflict, and regression are not of trivial significance - and each of these concepts may well be involved in an ultimately satisfactory theoretical interpretation of disinhibition.

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APPENDICES

APPENDIX A

RAW DATA FOR EXPERIMENT 1

INFLECTION RATIOS FOR EACH EXPERIMENTAL EXTINCTION SUBJECT ON PRETEST TRIALS 1 - 4

		Pretest Trial			
	Subject	1	2	3	4
1½ SECOND NOISE	1	.40	.49	.24	.54
	2	.33	.48	.51	.57
	3	.40	.43	.45	.39
	4	.39	.57	.51	.43
	5	.37	.46	.58	.57
	6	.47	.37	.51	.58
	7	.54	.37	.47	.52
	8	.49	.40	.51	.48
3 MINUTE NOISE	1	.43	.40	.55	.44
	2	.46	.47	.49	.53
	3	.41	.51	.51	.46
	4	.41	.56	.56	.48
	5	.42	.45	.48	.44
	6	.53	.52	.50	.62
	7	.33	.45	.47	.48
	8	.54	.55	.48	.42
1½ SECOND LIGHT	1	.54	.31	.49	.60
	2	.49	.55	.53	.56
	3	.50	.38	.54	.50
	4	.49	.53	.47	.48
	5	.48	.56	.54	.48
	6	.49	.55	.59	.69
	7	.61	.42	.51	.56
	8	.37	.48	.47	.50
3 MINUTE LIGHT	1	.36	.45	.49	.34
	2	.45	.39	.54	.49
	3	.22	.33	.32	.45
	4	.33	.39	.38	.37
	5	.20	.37	.42	.45
	6	.25	.24	.20	.21
	7	.25	.35	.34	.37
	8	.47	.48	.45	.39

INFLECTION RATIOS FOR EACH FOOD SATIATION SUBJECT

ON PRETEST TRIALS 1 - 4

		Pretest Trial			
	Subject	1	2	3	4
1½ SECOND NOISE	1	.41	.52	.31	.48
	2	.38	.60	.45	.45
	3	.32	.40	.53	.55
	4	.51	.51	.50	.60
	5	.49	.46	.38	.51
	6	.48	.33	.44	.78
	7	.42	.44	.44	.44
	8	.45	.49	.53	.60
3 MINUTE NOISE	1	.45	.50	.45	.52
	2	.59	.57	.65	.57
	3	.46	.53	.60	.58
	4	.46	.60	.47	.46
	5	.32	.49	.43	.56
	6	.55	.61	.49	.54
	7	.25	.49	.51	.52
	8	.34	.54	.63	.62
1½ SECOND LIGHT	1	.46	.50	.63	.45
	2	.41	.42	.42	.49
	3	.52	.33	.49	.51
	4	.41	.46	.41	.57
	5	.53	.33	.62	.45
	6	.51	.46	.47	.44
	7	.50	.29	.41	.56
	8	.56	.47	.47	.53
3 MINUTE LIGHT	1	.26	.37	.46	.52
	2	.36	.38	.37	.51
	3	.21	.28	.31	.34
	4	.22	.35	.34	.52
	5	.29	.37	.42	.37
	6	.30	.42	.44	.38
	7	.32	.48	.37	.45
	8	.30	.46	.29	.48

NUMBER OF RESPONSES FOR EACH EXPERIMENTAL EXTINCTION

SUBJECT ON TEST DAYS 1 - 4

		Stimulus Presentation				Dummy Presentation			
		1	2	3	4	1	2	3	4
	Subject								
1½ SECOND NOISE	1	2	0	0	0	0	0	1	0
	2	1	5	0	1	0	0	0	0
	3	3	0	0	0	0	1	0	1
	4	6	3	0	2	3	0	0	0
	5	7	1	0	1	0	0	0	0
	6	0	0	0	0	0	0	0	0
	7	0	6	1	0	1	0	0	0
	8	0	0	0	2	2	0	0	0
3 MINUTE NOISE	1	4	2	0	0	2	2	0	0
	2	2	0	0	0	0	0	0	0
	3	20	1	0	0	1	6	0	0
	4	1	0	0	4	0	0	0	0
	5	0	1	2	0	0	0	0	1
	6	5	2	0	0	7	2	1	1
	7	5	0	2	0	0	0	0	0
	8	8	3	0	0	5	1	0	0
1½ SECOND LIGHT	1	0	0	0	0	0	0	0	0
	2	2	0	0	0	0	0	0	2
	3	0	2	0	1	0	0	0	0
	4	0	0	1	0	0	1	0	0
	5	0	2	2	0	1	0	1	0
	6	11	3	16	1	4	1	0	0
	7	2	5	0	1	0	0	0	0
	8	2	0	2	2	8	0	0	1
3 MINUTE LIGHT	1	4	6	2	2	7	0	1	0
	2	7	2	2	0	0	0	0	0
	3	2	3	1	2	3	1	0	0
	4	2	1	0	1	0	0	0	2
	5	7	6	1	0	1	0	0	0
	6	7	0	1	0	1	0	0	0
	7	10	5	3	2	2	0	1	0
	8	14	4	4	0	1	1	0	0

NUMBER OF RESPONSES FOR EACH FOOD SATIATION SUBJECT ON TEST DAYS 1 - 4

		Stimulus Presentation				Dummy Presentation			
	Subject	1	2	3	4	1	2	3	4
1½ SECOND NOISE	1	0	8*	-	10	0	-	19*	0
	2	10*	10	10	3	-	0	3	10
	3	5	2	-	9	0	5	9*	7
	4	-	7	2	1	-	4	1	4
	5	-	-	0	5	-	-	0	4
	6	2	0	1	0	0	2	0	1
	7	-	15	-	4	-	10	-	6
	8	-	-	8	9	-	-	1	0
3 MINUTE NOISE	1	5	0	0	**	0	1	0	**
	2	7	4	3	-	2	0	2	4*
	3	-	6	13	13	-	0	0	0
	4	2	1	5	2	0	3	0	1
	5	18	24	13	20	5	0	0	3
	6	5	10	12	11	0	5	4	3
	7	1	10	9	16	0	0	0	0
	8	-	-	14	-	-	-	8	0*
1½ SECOND LIGHT	1	0	1	5	2	0	3	2	0
	2	4	3	3	0	2	2	1	0
	3	5	6	5	8	0	0	4	10
	4	-	2*	4	0	5*	-	2	11
	5	-	11	2	0	-	0	0	0
	6	-	0	4	0	-	12	0	0
	7	0	0	0	2	0	0	0	0
	8	-	18	2	5	9*	5	4	3
3 MINUTE LIGHT	1	10	1*	13	9	1	-	2	6
	2	-	-	10	8	-	-	1	0
	3	8*	-	14	22	-	-	11	1
	4	3	5	9	6	11	2	0	1
	5	15	8	7	5	8	2	2	3
	6	21	15	12	13	0	0	10	5
	7	6	4	8	7	22	8	8	0
	8	-	11	26	-	-	6	1	12*

* Response excluded from analysis because animal failed to receive both stimulus and dummy presentation

\- Presentation not given due to failure to meet 3 minute no-response criterion

** Subject died

INFLECTION RATIOS FOR EACH SHOCK PUNISHMENT SUBJECT ON PRETEST TRIALS 1 - 4

		Pretest Trial			
	Subject	1	2	3	4
3 MINUTE LIGHT	1	.28	.35	.29	.41
	2	.38	.52	.45	.51
	3	.37	.53	.30	.32
	4	.44	.36	.33	.34
	5	.25	.37	.47	.44
	6	.44	.36	.44	.48
	7	.22	.17	.15	.19
	8	.36	.45	.40	.52

NUMBER OF RESPONSES FOR EACH SHOCK PUNISHMENT SUBJECT ON TEST DAYS 1 - 4

		Stimulus Presentation				Dummy Presentation			
	Subject	1	2	3	4	1	2	3	4
3 MINUTE LIGHT + NOISE	1	3	1	0	0	1	0	0	0
	2	1	1	0	0	0	0	0	1
	3	1	2	0	1	1	0	0	0
	4	3	4	3	1	1	3	3	0
	5	1	1	2	1	1	0	1	0
	6	5	0	1	4	0	2	5	2
	7	0	1	0	9	0	0	0	3
	8	1	0	0	0	0	0	0	0

APPENDIX B

RAW DATA FOR EXPERIMENT 2

PRETEST INFLECTION RATIOS FOR SUBJECTS GIVEN 4 AND 16 NOISE PRETEST TRIALS

	Pretest Trial			
Subject	1	2	3	4
9	.45	.48	.53	.56
10	.58	.59	.49	.56
11	.48	.26	.54	.50
12	.36	.46	.30	.45
13	.28	.55	.46	.51

	Pretest Trial							
Subject	1	2	3	4	5	6	7	8
1	.54	.47	.53	.64	.58	.53	.44	.56
2	.46	.52	.48	.50	.56	.58	.51	.46
3	.42	.45	.58	.56	.61	.49	.57	.47
4	.23	.35	.33	.40	.29	.43	.46	.47
5	.23	.41	.35	.40	.42	.51	.55	.40
6	.41	.45	.39	.56	.48	.45	.43	.54
7	.33	.46	.63	.58	.47	.50	.55	.61
8	.38	.50	.57	.54	.46	.45	.55	.51

	Pretest Trial							
Subject	9	10	11	12	13	14	15	16
1	.54	.63	.59	.58	.60	.55	.54	.51
2	.43	.44	.58	.44	.56	.48	.53	.53
3	.61	.56	.54	.54	.50	.53	.53	.60
4	.42	.51	.45	.48	.53	.61	.47	.50
5	.46	.51	.63	.51	.51	.70	.44	.60
6	.46	.47	.47	.58	.41	.45	.55	.47
7	.56	.59	.56	.45	.47	.49	.57	.53
8	.48	.47	.70	.50	.53	.54	.53	.42

NUMBER OF RESPONSES FOR EACH SUBJECT ON TEST DAYS 1-4

		Stimulus Presentation				Dummy Presentation			
	Subject	1	2	3	4	1	2	3	4
NOISE- 0 PRETEST	1	1	1	0	0	0	0	0	0
	2	5	0	4	1	4	0	0	19
	3	0	0	0	0	0	0	0	0
	4	0	0	0	0	0	0	0	0
	5	19	6	4	1	6	3	0	0
	6	11	8	0	5	2	0	0	1
	7	0	0	1	0	1	0	0	0
	8	2	0	0	0	0	0	0	0
	9	7	1	0	0	2	0	0	0
	10	1	0	1	0	0	0	1	1
	11	3	3	0	0	1	0	0	0
	12	3	0	2	0	0	1	1	0
NOISE- 4 PRETEST	9	4	4	7	0	4	1	0	0
	10	1	8	1	1	0	0	0	0
	11	0	0	0	0	0	0	0	0
	12	3	2	0	11	7	0	0	0
	13	0	0	1	1	0	0	0	0
NOISE-16 PRETEST	1	2	3	4	0	2	0	0	0
	2	10	2	0	6	6	0	4	1
	3	10	6	0	2	3	2	1	0
	4	2	3	0	1	22	3	0	0
	5	0	8	0	2	0	0	0	0
	6	6	0	2	0	0	1	0	0
	7	12	0	0	0	0	0	0	0
	8	6	1	2	0	0	1	1	1
LIGHT- 0 PRETEST	1	6	2	2	0	0	0	0	0
	2	3	4	1	1	0	1	0	0
	3	5	5	4	2	3	1	0	0
	4	10	3	8	1	3	1	0	1
	5	20	7	0	0	0	0	0	0
	6	6	4	3	4	0	0	0	2
	7	0	4	0	0	2	2	0	0
	8	5	1	1	1	1	0	0	0
	9	5	0	0	4	6	0	1	0
	10	5	4	2	5	0	5	0	0
	11	6	0	0	2	0	1	0	0
	12	13	4	6	6	0	0	0	2

APPENDIX C

RAW DATA FOR EXPERIMENT 3

INFLECTION RATIOS FOR EACH SUBJECT ON PRETEST TRIALS 1 - 4

		Pretest Trial			
	Subject	1	2	3	4
NOISE-OFF	1	.43	.47	.49	.38
	2	.50	.47	.46	.56
	3	.56	.46	.49	.44
	4	.49	.45	.33	.31
	5	.44	.32	.46	.47
	6	.43	.45	.42	.50
	7	.48	.49	.53	.35
	8	.46	.46	.51	.40
	9	.45	.47	.46	.42
	10	.52	.43	.43	.48
	11	.47	.55	.56	.40
	12	.36	.40	.37	.50
LIGHT-OFF	1	.32	.46	.42	.56
	2	.22	.34	.45	.40
	3	.22	.40	.52	.43
	4	.28	.40	.42	.50
	5	.34	.47	.35	.47
	6	.28	.28	.44	.40
	7	.27	.41	.45	.48
	8	.20	.42	.40	.50

NUMBER OF RESPONSES FOR EACH SUBJECT ON TEST DAYS 1-4

		Stimulus Presentation				Dummy Presentation			
	Subject	1	2	3	4	1	2	3	4
NOISE-OFF	1	0	0	0	0	0	1	1	1
	2	0	0	0	0	0	0	0	0
	3	4	6	0	0	0	0	0	0
	4	2	0	0	0	0	0	0	0
	5	0	0	0	1	0	0	0	0
	6	0	0	0	0	0	0	0	0
	7	0	0	0	0	0	0	0	0
	8	0	0	0	4	0	0	0	0
	9	2	0	0	0	0	0	0	0
	10	5	0	2	0	0	0	0	0
	11	0	0	0	0	1	0	3	0
	12	6	0	0	1	2	0	0	0
LIGHT-OFF	1	5	4	0	0	7	0	0	17
	2	6	6	3	2	1	0	1	0
	3	1	2	2	2	0	0	0	0
	4	16	3	0	8	1	0	0	0
	5	25	8	2	0	13	0	1	0
	6	6	0	7	0	3	0	0	0
	7	4	0	0	0	0	0	0	0
	8	2	5	1	1	4	0	0	0

APPENDIX D

RAW DATA FOR EXPERIMENT 4

INFLECTION RATIOS FOR EACH SUBJECT ON PRETEST TRIALS 1-4

		Pretest Trial			
	Subject	1	2	3	4
45 DB. NOISE	1	.14	.43	.54	.38
	2	.38	.46	.48	.47
	3	.38	.52	.43	.50
	4	.52	.50	.58	.57
	5	.42	.37	.44	.52
	6	.39	.41	.51	.51
	7	.48	.48	.46	.42
	8	.44	.66	.60	.36
	9	.48	.52	.41	.52
	10	.42	.46	.47	.49
	11	.46	.44	.49	.52
	12	.54	.47	.48	.46
	13	.49	.52	.52	.53
65 DB. NOISE	1	.37	.38	.25	.48
	2	.43	.45	.58	.51
	3	.53	.53	.52	.40
	4	.35	.47	.43	.59
	5	.45	.51	.49	.43
	6	.50	.46	.52	.52
	7	.43	.67	.39	.58
	8	.44	.50	.33	.24
	9	.50	.52	.60	.46
	10	.63	.41	.71	.44
	11	.47	.41	.45	.49
	12	.47	.50	.38	.39
	13	.43	.51	.49	.44
100 DB. NOISE	1	.57	.38	.47	.36
	2	.41	.47	.50	.47
	3	.14	.42	.53	.33
	4	.11	.35	.41	.41
	5	.24	.30	.40	.42
	6	.09	.32	.37	.40
	7	.17	.60	.39	.59
	8	.41	.54	.50	.55
	9	.58	.50	.50	.63
	10	.15	.11	.31	.43
	11	.49	.57	.51	.58
	12	.53	.51	.49	.54
	13	.33	.48	.54	.48

NUMBER OF RESPONSES FOR EACH SUBJECT ON TEST DAYS 1-4

		Stimulus Presentation				Dummy Presentation			
		1	2	3	4	1	2	3	4
	Subject								
45 DB. NOISE	1	2	1	3	0	1	0	0	1
	2	0	0	0	0	0	0	0	0
	3	0	7	0	0	0	0	2	0
	4	4	0	0	1	0	0	0	0
	5	4	0	0	0	0	0	2	0
	6	3	3	2	2	2	0	1	2
	7	0	4	0	3	0	1	1	0
	8	0	0	0	3	0	0	3	0
	9	5	12	2	0	0	0	0	0
	10	0	0	0	0	6	0	0	0
	11	0	1	0	0	0	1	0	0
	12	1	7	4	4	0	0	0	0
	13	2	0	0	0	0	2	0	0
65 DB. NOISE	1	1	2	1	0	0	2	0	6
	2	2	0	2	0	0	0	3	0
	3	0	2	2	2	0	0	1	0
	4	4	1	0	0	1	0	0	1
	5	5	2	6	0	5	0	2	0
	6	11	0	1	1	3	0	0	0
	7	2	2	1	0	0	2	1	0
	8	1	4	0	0	0	0	0	0
	9	9	0	0	0	0	0	0	0
	10	4	0	0	3	0	0	1	0
	11	0	0	0	6	0	0	0	0
	12	4	5	1	0	0	0	0	0
	13	0	2	0	4	0	0	2	0
100 DB. NOISE	1	4	1	0	0	0	0	0	0
	2	1	0	0	0	0	0	0	0
	3	6	0	0	0	0	0	0	0
	4	16	2	1	5	2	2	0	0
	5	5	0	0	0	1	0	0	0
	6	4	1	0	2	0	0	0	0
	7	1	0	3	0	6	0	0	1
	8	7	0	0	0	10	0	2	0
	9	0	4	1	0	0	0	2	0
	10	1	0	1	1	1	0	0	0
	11	22	8	11	4	3	1	1	2
	12	4	2	0	0	0	0	0	0
	13	3	3	2	0	0	0	0	0

APPENDIX E

RAW DATA FOR EXPERIMENT 5

INFLECTION RATIOS FOR EACH SUBJECT ON PRETEST TRIALS 1 - 4

		Pretest Trial			
	Subject	1	2	3	4
3 MINUTE EXTINCTION	1	.30	.47	.81	.43
	2	.34	.51	.45	.43
	3	.46	.48	.62	.46
	4	.49	.58	.42	.41
	5	.49	.37	.54	.41
	6	.41	.56	.49	.44
	7	.39	.59	.49	.27
	8	.54	.42	.51	.48
	9	.53	.46	.46	.52
	10	.45	.52	.53	.59
30 MINUTE EXTINCTION	1	.48	.62	.59	.54
	2	.35	.37	.53	.56
	3	.48	.58	.68	.53
	4	.32	.38	.38	.52
	5	.50	.58	.48	.55
	6	.49	.49	.44	.50
	7	.42	.39	.61	.62
	8	.49	.48	.48	.50
	9	.42	.43	.52	.49
60 MINUTE EXTINCTION	1	.65	.59	.63	.44
	2	.51	.54	.57	.49
	3	.36	.64	.59	.58
	4	.48	.47	.47	.55
	5	.38	.59	.62	.46
	6	.39	.48	.40	.52
	7	.31	.50	.63	.54
	8	.56	.42	.46	.48
	9	.46	.52	.44	.50
	10	.20	.31	.32	.36
90 MINUTE EXTINCTION	1	.47	.50	.55	.45
	2	.39	.38	.48	.40
	3	.51	.59	.56	.45
	4	.54	.42	.55	.35
	5	.86	.54	.51	.41
	6	.44	.48	.57	.25
	7	.50	.56	.33	.60
	8	.77	.59	.52	.60
	9	.54	.54	.46	.51
	~~10~~				
120 MINUTE EXTINCTION	1	.39	.53	.40	.44
	2	.42	.53	.67	.56
	3	.23	.42	.44	.50
	4	.76	.37	.41	.47
	5	.38	.47	.57	.64
	6	.43	.46	.54	.56
	7	.27	.50	.50	.73
	8	.42	.47	.43	.35
	9	.20	.45	.52	.52
	10	.51	.54	.57	.52

NUMBER OF PRE-TEST AND TEST RESPONSES AND INFLECTION RATIOS FOR EXPERIMENTAL SUBJECTS AT DIFFERENT STAGES OF EXTINCTION

	Subject	3' Pre	3' Stimulus	Ratio
3 MINUTES EXTINCTION	1	47	71	.60
	2	41	28	.41
	3	12	13	.52
	4	39	39	.50
	5	51	56	.52
	6	24	38	.61
	7	12	23	.66
	8	35	43	.55
	9	28	32	.53
	10	32	36	.53
30 MINUTES EXTINCTION	1	13	16	.55
	2	16	11	.41
	3	22	23	.51
	4	26	30	.54
	5	12	12	.50
	6	17	14	.45
	7	17	16	.48
	8	48	32	.40
	9	12	26	.68
60 MINUTES EXTINCTION	1	6	19	.76
	2	14	19	.58
	3	6	18	.75
	4	6	38	.86
	5	11	18	.62
	6	4	3	.43
	7	15	19	.56
	8	14	15	.52
	9	4	8	.67
	10	5	1	.17

	Subject	3' Pre	3' Stimulus	Ratio
90 MINUTES EXTINCTION	1	0	6	1.00
	2	23	20	.47
	3	1	16	.94
	4	3	5	.63
	5	5	7	.58
	6	3	4	.57
	7	0	11	1.00
	8	0	6	1.00
	9	0	3	1.00
120 MINUTES EXTINCTION	1	0	1	1.00
	2	0	8	1.00
	3	0	0	*
	4	0	0	*
	5	0	0	*
	6	0	21	1.00
	7	1	8	.89
	8	0	0	*
	9	4	6	.60
	10	0	8	1.00

* Ratio indeterminate

NUMBER OF PRE-TEST AND TEST RESPONSES AND INFLECTION RATIOS

FOR CONTROL SUBJECTS AT DIFFERENT STAGES OF EXTINCTION

	3 Minutes Extinction			30 Minutes Extinction		
Subject	3' Pre	3' Dummy	Ratio	3' Pre	3' Dummy	Ratio
1	55	43	.44	55	38	.41
2	5	22	.81	8	14	.64
3	44	30	.41	41	40	.49
4	18	21	.54	5	0	.00
5	19	19	.50	10	9	.47
6	60	77	.56	38	27	.42
7	20	34	.63	13	25	.66
8	29	22	.43	16	18	.53
9	32	67	.68	39	36	.48
10	30	43	.59	23	27	.54

	60 Minutes Extinction			90 Minutes Extinction		
	3' Pre	3' Dummy	Ratio	3' Pre	3' Dummy	Ratio
1	27	5	.16	0	0	*
2	0	0	*	0	0	*
3	21	9	.30	6	1	.17
4	5	5	.50	0	3	1.00
5	1	0	.00	0	2	1.00
6	13	14	.52	2	0	.00
7	0	0	*	0	0	*
8	9	2	.18	2	1	.33
9	25	21	.46	6	6	.50
10	8	7	.47	5	9	.64

* Ratio indeterminate

APPENDIX F

RAW DATA FOR EXPERIMENT 6

NUMBER OF RESPONSES FOR EACH SUBJECT IN THREE-MINUTE POST-CRITERION PERIOD

CONTINUOUS REINFORCEMENT

Criterional Response

CONTROL SUBJECTS				EXPERIMENTAL SUBJECTS	
1	3	6	10	1	10
25	17	1	33	1	23
13	9	26	22	20	21
0	8	29	25	4	34
16	18	13	27	14	21
7	11	27	22	5	20
19	21	27	19	0	18
26	17	18	39	0	15
3	23	18	26	0	12
0	38	37	24	10	18
21			23	12	15
9			32	22	31
2			31	18	19
0				17	5
					32

EXTINCTION

Criterional Response

CONTROL SUBJECTS	EXPERIMENTAL SUBJECTS
1	1
2	3
0	32
0	26
0	12
0	14
5	1
3	7
5	1
2	7