An investigation of Eysenck's incubation of fear theory of anxiety and neurosis

by

Terrence P. Nicholaichuk

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requirements for the degree of Master of Arts
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Abstract

The purpose of this investigation was to examine one of the basic assumptions of H. J. Eysenck's (1968) behavioral theory of neurosis. The basic tenets of the theory state that neurotic behavior is acquired through Pavlovian conditioning, is subject to "spontaneous remission" in some cases, while in others, it continues to gain in strength in the absence of the original eliciting stimuli. Specifically it is postulated that after a single conditioned stimulus (CS), intense unconditioned stimulus (US) pairing, repeated CS alone presentations may result in an increase in fear (conditioned response; CR).

In the present study, rats were given a single tone CS - 3.5 mA shock US pairing and tested with CS alone trials over the next ten days. Major response measures were duration of freezing upon CS onset, latency to escape the portion of the apparatus in which the CS was presented and level of activity. In addition, several indices of activity were taken before the CS was introduced into the test situation. Unpaired and CS alone control groups were employed to assess the nonassociative effects of CS and US presentation, and a tone CS-1.05 mA shock paired group was employed to dimensionalize the incubation effect.

The major result of the procedure employed here was that rather than increasing, the levels of conditioned fear decreased with repeated CS alone presentations, for both the high and low shock intensity paired groups. The unpaired and CS alone control

groups demonstrated little or no associative effect of CS-US presentation. Taken together, these data question the validity of Eysenck's (1968) incubation of fear construct.

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An investigation of Eysenck's incubation of fear theory of anxiety and neurosis.

The purpose of the present investigation was to examine one of the basic assumptions of Eysenck's (1968) behavioral theory of neurosis. The basic tenets of the theory state that neurotic behavior is acquired through learning, is subject to spontaneous remission in some cases; while in other instances, it continues to gain strength in the absence of eliciting stimuli. The last assumption will be the major focus of this study.

Although psychologists, as yet, have not adopted a universally agreed upon theory of human neurosis (Eysenck, 1976), there does appear to be general agreement upon the description of the condition. Neurosis is a term often used to describe behavior which is associated with strong emotions such as anxiety or fear (Eysenck, 1977; Martin, 1972). For the purposes of the present discussion, we will adopt Wolpe's definition of neurosis.

"Neurotic behavior is any persistent habit of unadaptive behavior acquired by learning in a physiologically normal organism. Anxiety is usually the central constituent of this behavior, being invariably present in the causal situations." (Wolpe, 1958 P.32).

To elucidate the meaning of "unadaptive behavior", Wolpe (1958) defined it in terms of behavior which leads either to an unnecessary expenditure of energy, or to damage to the person's state of emotional or mental well being. Accordingly, for the neurotic, the feeling of anxiety (used synonymously with fear) may lead to the needless expenditure of energy required to avoid anxiety-provoking situations. Alternatively, the neurotic's anxiety may be debilitating in itself.

Although no characteristic behavioral pattern has yet been identified for the neurotic individual, the range of unadaptive behavior that might be displayed varies considerably. For some individuals, the unadaptive behaviors may comprise the majority of the individual's behavioral patterns as in the case of pervasive anxiety. For others, the behavior may appear only in quite specific situations as in instances of monosymptomatic phobias. Nonetheless, while the unadaptive behavior does not conform to a set pattern, "the unadaptiveness of neurotic reactions is usually overwhelmingly obvious" (Wolpe, 1958 P.33).

In Wolpe's definition of neurosis, the emphasis is upon the learning of unadaptive behavior. However, to provide an adequate account of neuroses, there is another aspect to neurotic behaviors which must be dealt with, and that is the evidence that suggests that neurotic disorders are subject to "spontaneous remission". For example, Eysenck (1952) concluded, on

the basis of published reports encompasing some 8,053 cases, that approximately two-thirds of neurotics recover or show marked improvement within two years of the onset of their condition whether they are treated by psychotherapy or not. Furthermore, Eysenck and Rachman (1965) concluded that the rate of spontaneous remission was a negatively decelerating curve (ie. resembled a normal extinction curve) and predict that over 90 per cent of neurotics will be recovered or markedly improved five years after the onset of their disorder. Any theory of neurosis will have to account for the fact that "spontaneous remission" does occur in a large number of neurotics.

On the other hand, it has been noted that in some cases, neurotic fears appear to increase rather than remit (Eysenck, 1968). In these cases, the neurosis becomes increasingly debilitating even in the absence of the initial fear inducing stimulus. Any theory of neurosis will also have to explain this important observation.

Eysenck, (1968) has proposed a theory of neurosis which provides a mechanism for the learning of anxiety, the "spontaneous remission" of neurotic behavior, as well as the increase in severity. For Eysenck, the development of anxiety is based on the acquisition processes of Pavlovian conditioning, while instances of "spontaneous remission" can be accounted for by

the extinction processes of Pavlovian conditioning. The Pavlovian conditioning paradigm involves an unconditioned stimulus (US) which reliably produces a measurable unconditioned response (UR), and a conditioned stimulus (CS) that does not initially produce a response resembling the UR. In the acquisition phase of conditioning, the CS and US are presented to the subject in a specified order and temporal spacing and independent of the subject's behavior.

With repeated stimulus presentations, a response that is similar to the UR, called the conditioned response (CR), develops to the CS. Over acquisition, the CR increases in strength to some asymptotic value (Gormezano & Kehoe, 1975). In the extinction phase of conditioning, the CS is presented unaccompanied by the US. Following repeated CS alone presentations, the CR can be expected to decrease in strength and eventually disappear (Kimble, 1961).

According to Mowrer (1939), while the classical conditioning paradigm is specified in terms of overt measurable responses, there is a long history of applying the paradigm to the acquisition and extinction of inferred emotional states such as fear (eg. Mowrer, 1939; Watson & Rayner, 1920). For example, Mowrer (1939) stated "anxiety or fear is a learned emotional response (CR) to stimuli (CS) denoting the advent of a painful or noxious event (US)[1].

1. Expressions in parentheses inserted by the author.

It is a response acquired in accordance with the associative laws of classical conditioning". In addition, it is assumed that conditioned fear acquires drive properties which when reduced can serve to reinforce a contingent response (Brown, 1961; Gormezano & Moore, 1969). The learned and drive like properties of conditioned fear have been inferred from studies which have monitored conditioned autonomic responses (cf. Rescorla & Solomon, 1967) or CS produced changes in instrumentally conditioned behavior (cf. Estes & Skinner, 1941), consummatory responses (eg. Amsel, 1950) or general activity (eg. Brown, Kalish & Farber, 1951).

Eysenck's theory of neurosis.

To account for the development of neurotic behaviors, Eysenck's theory (1968, 1976, 1977), paralleling the traditional accounts of conditioned fear (c.f. Brown, 1961; Rescorla & Solomon, 1967), assumed that when a CS signals the occurrence of a noxious US, the CS by means of the laws of classical conditioning, becomes a signal for impending danger and pain and elicits discomfort, fear, and annoyance (the CR's were termed nocive responses or NR's by Eysenck, 1968). In addition, it is assumed that the association between the CS and CR is strengthened with each successive CS - US pairing. At this point, Eysenck deviates from traditional learning theory (although a similar type of mechanism has been suggested by Solomon & Wynne (1953) to account for the seeming irreversibility of avoidance responding in dogs)

by assuming that the conditioned fear (a CR) should be considered to be a painful event, and therfore should increase fear in the situation and further strengthen the CS - CR bond. Specifically, Eysenck asserts that "CS (CS alone)[1]. although unaccompanied by UCS or UCR, is in fact accompanied by CR, which is a partial, possibly weak but real NR. Hence some reinforcement is provided, although perhaps this is so much weaker than that accompanying the UCS that its presence may not be very important under certain circumstances. Yet in principle it is always present, and its presence would theoretically lead to a strengthening of the CS/NR bond, and hence to some form of incubation. What is being suggested, in other words, is that conditioning sets in mothion a positive feed-back cycle in which the CR provides reinforcement for the CS. Usually the extinction process will be stronger than this form if reinforcement, leading to overall extinction, but under certain circumstances (e.g. when the UCS is exceptionally strong) the extinction process may be weaker than the CS/NR reinforcement process and observable incubation will result. What we propose to add is that fear, so generated, is itself a painful event, and the stimuli associated with it (i.e. CS) therefore by classical conditioning, come to evoke more fear thus producing a positive feed-back." (1968, P.313).

Therefore, according to Eysenck, once conditioning has commenced, two processes will affect the strength of the CS - CR

association when the CS is presented without the US. The first process is the traditional decremental CS - CR association. The second process is the presumed CR produced augmentation of fear which strengthens the CS - CR association. Consequently, the net effect of a CS alone presentation will be determined by the strongest of the two processes. If the decremental process is stronger than the incremental process, a series of CS alone presentations should produce extinction. This process is presumed to underly the "spontaneous remission" of neurotic disorders. On the other hand, if the incremental process is greater than the decremental process, the CS - CR association will become stronger and the neurotic disorder should worsen.

The CR produced augmentation of the CS - CR association on CS alone presentation was called incubation of fear by Eysenck (1968). As an aside, it should be pointed out that the term "incubation of fear" has been used in a different context with a different meaning. If an organism is exposed to an aversive event, and the level of resulting fear is subsequently monitored without the CS being presented in the interval it is observed (eg. Kamin, 1957) that the level of fear first decreases from, but then later returns to its initial value. This time dependent change in the strength of the fear reaction has also been called the incubation of fear. It is obvious that this use of the term differs in process from the augmentation of fear on CS alone trials proposed by Eysenck (1968). Throughout

this study, the term "incubation of fear" will be applied to Eysenck's hypothesized mechanism, while the term "Kamin effect" will be applied to the observed phenomenon of time dependent changes in the strength of fear.

While the postulation of incubation of fear allowed Eysenck (1968) to apply conditioning theory to account for neurosis, the concept must be considered to be circularly defined unless several issues can be clarified. Specifically, the factors controlling the incubation of fear must be delineated and the phenomenon must be observed independent from its application to neurotic disorders. Eysenck (1968, 1976, 1977) recognizing this potential problem postulated several variables which should affect the incubation of fear process and summarized several studies which appeared to demonstrate the phenomenon.

Theoretically, Eysenck (1976) asserted that the incubation process only occurs when the CS elicits CR's with aversive drive properties. Presumably there are no incubation effects with appetitive US's. When a subject experiences painful shock, it is this experience which comes to be associated with the CS and to which is added another increment of pain and fear in the form of the CR (Eysenck, 1968). Under conditions where an extremely aversive US is employed, Eysenck proposed that the acquired CR (fear) will be of sufficient magnitude so that it will operate in a manner similar to the US and counteract the effects of

extinction. The process is thought to set up a positive feedback cycle with each presentation of the CS alone accompanied by and incrementing the CR (fear). Eysenck (1968) further proposed that the resulting incubation of fear could result after a single CS, intense US pairing.

Eysenck (1976) went on to specify some conditions which may serve to facilitate the incubation of fear with CS alone presentations. These are, CS presentations of short duration (cf. Rohrbaugh & Riccio, 1970; Silvestri, Rohrbaugh & Riccio, 1970), extremely aversive US's (cf. Campbell, Sanderson & Laverty, 1964), and individual characteristics of the subjects. He did report, however, that evidence for these last two, particularly subject characteristics, was weak.

Eysenck (1968, 1976) has cited data from both the human and animal learning literature that purportedly supports his incubation of fear hypothesis. This evidence will be briefly reviewed in the following paragraphs.

The first group of studies reported (Campbell, Sanderson & Laverty, 1964; Dykman & Gantt, 1960; Lichtenstein, 1950; Napalkov, 1963) purportedly demonstrate an increase in anxiety following repeated CS alone presentations. The next group of studies (Allen & Mitcham, 1970; Bindra & Cameron, 1953; Denny & Ditchman, 1962; Desidorato & Wassarman, 1967; Golin, 1961; Golin & Golin, 1966; Kamin, 1957, 1963; Kumar, 1970; McAllister & McAllister, 1963; McMichael, 1966; Solomon, Kamin & Wynne, 1953) although cited by

Eysenck (1968, 1976) as support for the incubation of fear hypothesis, do not deal with the hypothesis, but rather are directed at the retention and generalization of fear over time. Finally, recent experiments, cited by Eysenck (1968, 1976) to support the presumed parametric control of the incubation of fear process will be reviewed (ie. Reynierse, 1966; Rohrbaugh & Riccio, 1970; Rohrbaugh, Riccio & Arthur, 1972; Silvestri, Rohrbaugh & Riccio, 1970).

Studies demonstrating an increase in fear.

Napalkov (1963; cf. Eysenck, 1967) in studies of hypertension in dogs paired various nocive stimuli such as strong shock, flashes of light and firing of toy pistols with a neutral CS. The dogs which received many such pairings experienced a rise in blood pressure of 30 - 50 mm. upon presentations of these stimuli. After 20 - 30 pairings these dogs habituated to the stimuli and no further increase in blood pressure was observed. Several animals, however, received only one such pairing which was followed by CS alone presentations of unspecified length. Blood pressure rose to 190 - 230 mm. and did not fall for many months. Hypertension persisted in these dogs for the entire observation period of 16 months.

Lichtenstein (1950) in a study of feeding inhibition in dogs reported instances of the failure of this feeding inhibition to extinguish for months after shock was discontinued.

The subjects were given 85 V. shocks of 2 sec. duration when a feeding response was made. There were a total of 60 trials given before shock was discontinued. When the dogs were reintroduced to the feeding situation, they struggled, resisted, vomited and developed tremors, tic-like movements and disturbed respiration. Depression in general activity level and increased aggression against cage mates were also noted. In another experiment with dogs, Dykman & Gantt (1960), in an attempt to condition an orienting response, delivered three 120 V. shock US's, each preceded by a tone CS. The intertrial interval (ITI) was five minutes. Over the next 24 days repeated attempts to extinguish the CR were made. In this time period heart rate increased, tremors developed, the dog showed signs of fearing the experimenter and began to defecate, urinate and vomit. It was noted that these behaviors intensified once they appeared. Campbell, Sanderson & Laverty (1964) paired a 5-sec. tone CS to a 100-sec. drug induced interruption of respiration US in human subjects. GSR readings were taken as the major measure of CR strength. Thirty CS alone presentations were given 5 min. after conditioning, again one week later, and 40 more CS alone trials were given three weeks later. GSR continued to grow in strength despite the repeated extinction trials.

While it would appear that each of these studies demonstrates the incubation of fear effect, acceptance of this

assertion is premature since the following methodological considerations mitigate against the conclusion.

First, in the Dykman & Gantt (1960) and Lichtenstein (1950) studies, the noted physiological changes were not systematically recorded, but were based on unsystematic observations of the subjects. Whether these observations would be confirmed under systematic examination is an open empirical question. Second, the Campbell et al (1964), Dykman & Gantt (1960) and Lichtenstein (1950) papers did not employ a discrete CS, but inferred that apparatus cues served as the CS for conditioning. Accordingly, it is not possible to separate the presumed CR produced increase in CS - CR strength from non-associative factors which might also augment a fear reaction. Without a discrete CS, and without the appropriate control group methodolgy it is not possible to attribute the behavioral changes to incubation of fear. Third, a careful analysis of the Lichtenstein (1950) experiment clearly indicates that Lichtenstein's procedure was one of punishment (i.e. aversive stimulus was response contingent), not Pavlovian conditioning. Consequently, only if punishment and Pavlovian conditioning are controlled by the same processes, can the Lichtenstein experiment be used to support the Pavlovian process of incubation of fear. Fourth, the Campbell et al (1964), study employed the GSR as a dependent variable, but did not control for the known sensitization effects on GSR (c.f., Stewart, Stern, Winokur & Fredman, 1961), the methodological hazards associated with the GSR response system (c.f. Venables

& Martin, 1967) or the known ideational control of the GSR (c.f. Cautela, 1967; McAllister & McAllister, 1967; Woods, 1974). Thus, only Napalkov (1963), appears to have produced a genuine incubation effect. Unfortunately, the account of his experimental procedure is so vague that it defies any attempt at direct interpretation or replication.

Evidence for the retention of fear.

Of the next group of studies presented by Eysenck (1968, 1976), to support his incubation of fear hypothesis, only one, a study by Solomon, Kamin & Wynne (1953), is actually relevant. The remaining studies examine the retention of fear over time rather than an effect of CS alone trials on levels of fear. An examination of the relevant study will be presented first, and this will be followed by a summary of the fear retention literature.

Solomon, Kamin & Wynne (1953) paired a tone CS accompanied by raising a gate separating two sides of a shuttle box with an intense shock (US). After the subjects (dogs) had learned to avoid the shock by jumping to the other side of the apparatus, they were tested in extinction. When the CS was repeatedly presented alone the latencies to jump the hurdle became shorter and the response failed to extinguish for up to 490 trials. The jumping response was finally eliminated after the experimenters punished it (the dogs jumped into shock) and prevented it

(a glass barrier was raised to prevent the dogs from shuttling). Since avoidance behavior was maintained on CS alone trials, the Solomon et al (1953) study is consistent with an incubation of fear hypothesis. However, since there was no clear evidence of an augmentation in avoidance responding, the experiment can not be employed to support a CR produced increment in CS - CR associative strength.

In the animal literature, the retention of fear is examined by assessing the level of fear at some time interval following a fear training regime (e.g. avoidance conditioning, passive avoidance, or Pavlovian fear conditioning). In the prototype experiment, Kamin (1957) gave rats avoidance training in a shuttle box and tested his group for retention of the avoidance response. Different groups were tested 0 hours, 0.5 hours, 1 hour, 6 hours, 24 hours, and 19 days after conditioning. Kamin reported that the magnitude of fear decreased from the levels observed with the 0 hour delay group, to a minimum for the 1 hour delay group, and then for the longer delay groups, returned to the levels of the 0 hour delay group. While the temporal location of the minimum level of fear is a contentious issue, the U-shaped fear retention curve is typically observed in avoidance situations (e.g. Denny & Ditchman, 1962; Kamin, 1957, 1963; McMichael, 1966, experiment I) passive avoidance situations (e.g. Tarpy,

1966). Failures to observe a U-shaped function have been reported by Allen & Mitcham (1970), in passive avoidance, and by McMichael (1966, experiment 2), in Pavlovian conditioned fear. In both studies the level of fear increased with the passage of time.

In the human research on fear retention, there has been a wide range of procedures employed. However, typically the retention interval has been relatively short (less than 30 minutes) and the dependent measure monitored, has been the GSR. In all studies, the index of fear has increased over the retention interval. For example, Bindra & Cameron (1953) gave human subjects shock paired with a discriminative stimulus. GSR scores were greater after a 10 minute rest period given during training. While the subjects were not tested in extinction, the enhancement of GSR scores in the post rest session were taken by Eysenck (1968, 1976) as evidence of a time dependent increase in fear. In a somewhat different paradigm, Golin (1961) paired a shock US with stimulus words. Subjects were aware of the shock contingency for one word but unaware of it for the others. After acquisition (6 trials) half of the subjects experienced a 30 minute post-shock delay before extinction began. The amplitude of the GSR decreased over acquisition trials and increased dramatically on the first trial after the delay. The group given the extinction condition immediately did not display a similar increase in GSR amplitude. Golin &

Golin (1966) gave human subjects light-shock pairings for eight acquisition trials using 0.5 minutes, 2.0 minutes, and 4.0 minutes intertrial intervals (ITI). GSR amplitude, in extinction was greatest for the 0.5 minute ITI group than the other groups after 30 minutes post-conditioning delay. And finally, Desidorato & Wassarman (1967) trained subjects to label a visual stimulus (a 104 mm 2 black square projected on a white ground). Subjects were then tested for generalization either immediately, or after a 15 minute delay. For half the subjects the stimulus was paired with an aversive ton US. The other half received non-aversive tone. Each group was further divided in to low and high anxiety subjects. Their major finding was that highly anxious subjects gave fewere gerneralized responses after the delay. The groups tested immediately showed no differences. The results were deemed compatible with the incubation notion because it was assumed that the 15 minute delay produced an increase in emotionality during aversive training and gave rise to the elevated generalization gradient.

Although Eysenck (1968, 1976) claims that the fear retention studies are consistent with the incubation of fear hypothesis, a comparison of the independent variable actually manipulated (time) to the theoretically required independent variable manipulation (CS alone presentations) clearly indicates that the studies are not pertinent to the hypothesis. However, it could be

argued, that the studies (Allen & Mitcham, 1970; Bindra & Cameron, 1953; Desidorato & Wassarman, 1967; Golin, 1961; Golin & Golin, 1966; McMichael, 1966, experiment 2) that showed an increase in fear over time might support a different type of theoretical mechanism which increases the amount of fear in the absence of the US; or CS - US pairings. This would also be a tenuous argument, since the bulk of the studies employed the GSR as a dependent measure without considering the possibility of ideational control (Cautela, 1967; Woods, 1974), methodological limitations (Venables & Martin, 1967), and behavioral controls (Stewart, Stern Winokur & Fredman, 1961). Particularly critical is the absence of behavioral controls. For it is known (c.f. Venables & Martin, 1967) that if a passage of time follows the habituation of the GSR response, then on subsequent stimulus presentations the magnitude of the GSR will recover to pre-habituation levels.

Recent attempts to determine the boundary conditions of incubation of fear.

More recently, Eysenck (1976) reported four studies (Silvestri, Rohrbaugh & Riccio, 1970; Reynierse, 1966; Rohrbaugh & Riccio, 1970; Rohrbaugh, Riccio & Arthur, 1972) which were interpreted as directly supporting the incubation of fear hypothesis. In addition, these studies, by manipulating either the number of CS alone trials that followed fear training

(Reynierse , 1966) or the duration of the CS on CS alone trials (Silvestri, Rohrbaugh & Riccio, 1970; Rohrbaugh & Riccio, 1970; Rohrbaugh, Riccio & Arthur, 1972) provided the first parametic investigation of factors which should regulate the incubation of fear process.

Reynierse (1966) trained rats to avoid shock signalled by a tone CS and presented either one, five or twenty CS only presentations in the 40 minutes period immediately after the avoidance response was acquired. All animals were then given additional extinction trials 24 hours later. Rats which received one CS only trial immediately after training extinguished rapidly. Animals which received 20 CS alone trials and those which received a single CS exposure 40 minutes after acquisition displayed much greater resistance to extinction. Thus it would appear that an increase in both number of CS alone trials and time from training to CS alone trials increase the magnitude of the incubation process.

Rohrbaugh & Riccio (1970, experiment 1) shocked food and water deprived rats in an experimental chamber and subsequently indexed fear by the latency the subjects took to approach food and water in the chamber. Prior to the evaluation of fear levels, subjects were given CS only exposure of 0., .5, 5, 15, or 50 minutes in length, temporally centered in the one hour interval following conditioning. Rats in the 5 minute

CS exposure group had the longest latency of approach to food and water while groups 0 minutes, .5 minutes, 15 minutes, and 50 minutes CS only exposure followed, in order of decreasing latencies of approach. In a second experiment, rats were shocked in one side of a compartment and tested for spacial avoidance of that side two weeks later. In the interval between shocking and testing, three CS only exposures of either 0, .5, 1, and 5 minutes durations were administered. In the subsequent test, animals in the .5 and 1 minute groups avoided the shock side significantly more than the other groups ($p < .01$).

Silvestri, Rohrbaugh & Riccio (1970) went on to compare CS only exposures with shock reinstatement which were interpolated in the four week period between conditioning and testing. Rats were given 200 V. shock in a black colored segment of a two-compartment chamber in the conditioning phase. The "safe" side was colored white. Group 1 was given three reinstating shocks, in the black side, at weekly intervals after conditioning. Group 2 was given three CS only exposures (in the form of apparatus cues) in the same period. Group 3 served as a retention control and Group 4 received reinstating shock in the black segment but no initial conditioning. Group 1 avoided the shock side of the apparatus considerably more than Group 2, which in turn avoided more than Group 3. Group 4 demonstrated the poorest avoidance of the black side. Thus, while CS alone presentation increases

fear over no CS alone conditions, the increase in fear was less than would occur with continued CS - US pairings (Group 2 vs Group 1).

A more refined investigation of the incubation of fear phenomenon was conducted by Rohrbaugh, Riccio & Arthur (1972). Water deprived rats received six, one second 175 V. shocks paired with a tone CS. In the 12 minute period following conditioning the animals received 0 seconds, 15 seconds, or 10 minute exposure to the CS only. Suppression of the licking response was taken as a measure of CR strength. Rats in the 15 second CS group displayed the greatest suppression of licking on the first test trial. Suppression was, however, less pronounced in the second trial (approximately 20 licks in the last minute of the first test and 115 licks in the last minute of the second trial).

The preceding studies suggest that CS alone presentations may augment the level of fear in a situation. They also suggest that the magnitude of fear is directly related to the number of CS alone presentations (Silvestri et al, 1970). In addition, it would appear that a short CS exposure produces increments of larger magnitude than either very brief or long CS exposures (Rohrbaugh & Riccio, 1970; Rohrbaugh et al, 1972). However, it is not clear how Eysenck's theory accounts for Reynierse (1966) finding that 20 CS only exposures produced enhancement equal

to a single CS only exposure 40 minutes after conditioning. Rohrbaugh & Riccio (1970) and Silvestri et al (1970) while demonstrating an incubation effect, failed to perform repeated extinction trials to determine whether CR strength continued incrementing and finally, although Rohrbaugh et al (1972) produced an enhancement of CR strength following CS only exposure, the effect deteriorated on the second test trial. Eysenck's incubation of fear hypothesis would predict that given CR enhancement initally occurs, it should continue to occur in extinction. These studies are further confounded by procedural dissimilarities and lack of adequate controls. Thus, these studies give mixed support to Eysenck's position.

In summary, the preceding analysis does not lead to Eysenck's (1968, 1976) conclusion that the incubation of fear is a well documented phenomenon. Eysenck's referent experiments (Campbell et al, 1964; Dykman & Gantt, 1960; Lichtenstein, 1950; Napalkov, 1963) are methodologically inadequate. A large number of the cited experiments are directed at the retention of fear, not the incubation of fear. And the few remaining experiments give only mixed support to the hypothesis. Since Eysenck's (1968, 1976, 1977) theory of neurosis requires the incubation of fear hypothesis to fully account for the behavior associated with neurotic disorders, the lack of confirmation of the hypothesis questions the tenability of Eysenck's theoretical account. Accordingly, the present study will attempt to provide an un-

ambiguous examination of the incubation of fear hypothesis.

The incubation of fear hypothesis states that on CS alone trials that follow fear conditioning, the CS - CR association is strengthened by the occurrence of the fear CR. Consequently, performance on the CS alone trial will be determined by the subtractive interaction between the incubation effect and extinction. For an increase in the observed fear reaction to occur, the magnitude of the incubation effect must be greater than the magnitude of the extinction effect. Since Eysenck (1968, 1976) claims that the magnitude of the incubation effect is directly related to the intensity of the US and the accompanying UR, manipulations of US intensity should maximize the probability of observing an incubation effect. Thus, it would be expected that the level of conditioned fear, and hence the magnitude of the incubation effect should be a simple direct function of US intensity.

However, this expectation does not appear to be entirely confirmed in the fear conditioning literature. In classical aversive conditioning (c.f. Gormezano & Moore, 1969) and in passive avoidance (e.g. TenHave, 1977; Zammit - Montebello, Black, Marquis & Suboski, 1969) shock intensity directly controls the situations, increases in US intensity facilitate avoidance indices of conditioning. Similarly, in one-way avoidance (e.g. Moyer & Korn, 1966; Theios, Lynch & Lowe, 1966). In

contrast, increases in shock intensity in two-way avoidance often tends to impair performance, rather than increase it (e.g. Levis & Boyd, 1973; McAllister & McAllister, 1971; Moyer & Korn, 1964; Theios et al, 1966). In addition when fear is measured by superimposing the CS on instrumental or consummatory behavior, the index of conditioning (changes in behavior during the CS) may be directly related to US intensity (c.f. Gormezano & Moore, 1969) but the uninterpretable, because the US intensity manipulation interrupts all responding in the situation (e.g. Church, 1969). These complex outcomes led Eysenck to conclude that the effect of shock intensity is not clear.

However, Blanchard & Blanchard (1968 a, 1968 b, 1969) Bolles (1971) and Myer (1971) have reported observations which may help explain these complex results. These authors tested rats in a variety of avoidance and escape situations using shock US's up to a 3.0 mA in intensity. They reported that intense shock evokes "pre-experimentally acquired responses" such as freezing and crouching which interfere with instrumental escape and avoidance responding. They concluded that avoidance is the major reaction to approaching aversive stimuli, but freezing, in whatever posture the rat has attained, is the characteristic response to aversive, static, situational stimuli (Blanchard & Blanchard, 1969). Bolles (1971) further concluded that a rat will learn to escape quickly if the escape response does not interfere with freezing (ie. the animal will freeze on a bar if a bar-press

response is needed to terminate shock) but the acquisition of the escape response will suffer if it competes with freezing. Accordingly, if fear is assessed with a univariate experimental design, as is typically the case, and if the reaction to a fear stimulus may be one of several competing response tendencies, the empirical observations of independent variable manipulations should be complex, since the dependent measure selected by the experimenter may or may not be the subject's dominant response to the experimenter's selected task. Since Eysenck (1968, 1976) does not specify the fear behavior, a multivariate approach should maximize the opportunity of observing the incubation of fear phenomenon.

While Eysenck's (1968) incubation of fear hypothesis is primarily based upon results reported in the learning literature, it has been previously noted that those studies cited as support for the theory are subject to a number of criticisms. The present investigation proposes to answer these criticisms and provide a more valid examination of the incubation of fear phenomenon. First, rather than conditioning a response not normally in the subject's behavioral repetoire and then taking changes in that response as a measure of Pavlovian fear conditioning, the subject's innate responses to anxiety-provoking stimuli (both freezing and escape) were taken as indices of

acquired fear. Second, conditioning and testing took place in two dissimilar chambers in order to minimize the effect of apparatus cues on behavior (McAllister & McAllister, 1971), so that the associative effects to the CS could more readily be observed. Third, rats were given a single CS, intense shock US pairing. Eysenck's (1968, 1976) model predicts that such pairing, if extremely traumatic, will be sufficient to produce incubation of fear. Fourth, high and low shock intensities were present to separate groups of subjects. Presumably, incubation of fear should be demonstrated with high intensity shock, but extinction should be observed with the lower intensity shock. Fifth, by presenting CS alone trials once per day over ten consecutive days, the time course of the proposed investigation was well beyond the temporal range of the "Kamin effect" (eg. Kamin, 1957), and therefore allowed a more rigorous examination of the incubation of fear phenomenon. Sixth, the animals were given a number of CS alone trials to determine whether the fear response would incubate (increase in magnitude) or extinguish. And finally, control groups were employed to separate the associative effects from the non-associative effects of the shock US. Eysenck's model predicts that the incubation of fear should result from a pairing operation and therefore should be observed only in the associative (paired) groups.

Method

Subjects.

The subjects were 50 hooded rats, weighing from 200 - 300 gm. on arrival, obtained from the University of Manitoba's Dentistry Department breeding colony. They were housed in individual cages with food and water freely available.

Apparatus.

The apparatus consisted of two chambers: a conditioning chamber and a test chamber. Conditioning took place in a brushed aluminum Coulbourn Instruments Inc. Model E 10-10 operant chamber (30 cm. X 24 cm. & 29 cm. in height) equipped with a house light, a grid floor and a 6.6 cm. diameter 8 ohm speaker. The grid floor was composed of 7 mm. diameter stainless steel bars 1.8 cm. apart (centre to centre). Scrambled shock was delivered to the floor of the chamber by a Coulbourn Instruments Inc, Solid State, Shock Generator. The conditioning chamber was housed in a sound and light attenuating fibre-glass box equipped with an exhaust fan. The test chamber, constructed of wood, (60 cm. X 30 cm. & 20 cm. in height) was divided into two 30 cm. X 30 cm. sections by a wall. A door 7.0 cm. wide and 7.0 cm. high was centred at the base of the wall. The floor and walls were painted white and an 8 ohm speaker was centred on the wall opposite the door in each section of the chamber. Each side was also equipped

with a house light situated on the same wall as, but to the left of of the speaker and centred 3.0 cm. down and 3.0 cm. over from the top, left hand corner. In addition, the floor of each section was divided into nine 10 cm. by 10 cm. squares by black lines. The ceiling of the chamber was covered with clear plexiglass. An adjustable mirror was mounted above and to the rear of the apparatus to permit relatively unobtrusive observation of the subjects, and a piece of window screen was placed over the plexiglass lid of the chamber to make the experimenter's presence more difficult for the animals to detect. In addition, all observations took place in a darkened room. All stimulus deliveries and data collection were accomplished by means of Coulbourn Instruments Inc. programmable modules situated in an adjoining room.

Procedure.

Subjects were randomly assigned to one of five groups (N = 10). Several rats died of pneumonia during the course of the study decreasing the N of some groups to 9. Consequently, one subject was randomly deleted from each of the remaining groups to leave nine subjects in each group (total N = 45). The animals were further divided into two groups (N = 24 and 21, respectively) which were each run by one of two different experimenters. Of the five experimental groups, the first group (3.5 - P) was given

a single 4 second, white noise CS of moderate intesity paired with a 4 second 3.5 mA scrambled shock US at a forward inter-stimulus interval of 4 seconds. The second group (1.05 - P) received the same CS parameters but the US was a 1.05 mA shock. The third group (CS-A) was exposed to the tone CS only. The remaining two groups were explicitly unpaired groups (3.5 - U and 1.05 - U) which differed only in the intensity of the US (3.5 mA and 1.05 mA, respectively). Half of each unpaired control group received a single forward ordering of the CS and US separated by a 20 minute interval. The other half received a single backward ordering of the CS and US also separated by 20 minutes. The large temporal separation between stimuli was intended to minimize associative effects that have been observed in single trial conditioning sessions employing briefer (i.e. 2 minute) ISI's (Nicholaichuk, 1977).

The treatment day consisted of a 30 minute session in the conditioning apparatus for all groups. Fifteen minutes from the onset of the session, the paired and CS alone groups received the appropriate stimulation. For the unpaired groups, the initial stimulus (either the CS or the US) was delivered 5 minutes after placement in the chamber and the following stimulus (either the US or the CS respectively) 20 minutes later.

On the following day, each subject was placed in the test chamber and allowed to habituate to the new environment. A

measure of general activity was taken in six consecutive 5 minute intervals. The level of general activity was determined by counting the number of squares which both front paws entered. The number of times the rat crossed over to a different side of the chamber was also recorded. The rest of the experiment consisted of ten daily sessions. In each of these sessions the rat was placed in the test chamber and after 5 minutes given a single 4 second CS alone presentation in the side of the chamber occupied by the subject. The side of the chamber in which the animal was initially placed was alternated daily. The session was terminated when the rat left or escaped the portion of the chamber in which it received the CS.

During the test phase, a number of behaviors were recorded. Before the CS presentation an index of activity and number of cross overs were obtained. After CS onset, the duration of freezing, latency to escape and an index of activity were measured. Freezing was defined as a complete absence of movement characterized by abruptness of onset, wide open eyes, and muscular rigidity. Latency to escape was defined as the time between CS onset and the moment the subject placed both front paws through the door separating the two sides of the test chamber. Bolles (1971) and Walsh and Cummins (1976) have reported that decreased activity levels, particularly freezing, have been widely employed to

measure fear levels. As fear dissipates, freezing behavior dissipates; the response repetoire broadens and activity increases (Walsh & Cummins, 1976). Accordingly, in order to accurately follow behavioral changes during testing, latency to escape the side of the chamber in which the CS is presented, the length of the freezing response, and level of activity were taken as indicies of acquired fear.

Subjects were returned to their home cages 2 minutes after the occurence of the escape response, and the apparatus was washed out with a disinfectant solution before the next trial began. Unlike the procedure employed by Rohrbaugh and Riccio (1970), there was no ceiling imposed on the length of each test trial.

The design of the experiment maximized the probability of observing the incubation of fear phenomenon by: (1) employing an intertrial interval in the test phase which exceeds the temporal range of the time-dependent changes in fear (e.g. Kamin, 1957); (2) monitoring several of the subjects' innate responses to anxiety-provoking stimuli (Bolles, 1971) rather than relying on random response selection; (3) employing a medium (1.05 mA) and intense (3.5 mA) shock intensity to evaluate the hypothesized (Eysenck, 1968, 1976) subtractive relation between incubation of fear and extinction; and (4) using unpaired and CS alone controls to

separate the associative from the non-associative effects of intensity of the shock US.

Results

The results of this investigation were organized and analysed in the following manner. First, t-tests were applied to experimenter and control group differences. Second,analyses of variance (ANOVA) were applied to measures of cross overs and activity taken during the habituation period. Third, measures of cross overs and activity recorded before the introduction of the CS on each of the 10 trials were subjected to analyses of variance. In addition, changes in these response measures over trials were assessed by means of a Multivariate analysis of variance (MANOVA) using duration of freezing, latency to escape and activity after CS presentation as the dependent variables for the analysis. Finally, univariate analyses of variance with orthogonal components for trend were employed to assess the changes in freezing, escape and activity, across trials and groups as well as the effect of CS - US pairing and shock intensity.

In each of the above univariate analyses the level of significance was set at $\alpha = .01$ in order to reduce the experiment-wise error rate. For the Multivariate comparisons the level of significance was set at $\alpha = .05$. Although somewhat higher than the level of significance selected for the univariate tests, this latter alpha is generally regarded as conservative when Multivariate comparisons are employed.

Experimenter and control group comparisons.

Table 1 presents the mean scores on each of the post-CS dependent variables (i.e. freezing, escape, and post-CS activity)

Table 1

Experimenter differences on each of the post-CS dependent variables : mean scores and t-statistics.

Response Measure	Experimenter $\bar{X}_1$	Experimenter $\bar{X}_2$	Degrees of Freedom	t	Probability (two-tailed)
Freezing	232.66	348.07	43	-1.083	$p > 0.05$
Escape	389.05	583.95	43	-1.213	$p > 0.05$
Activity	6.33	7.17	43	-0.470	$p > 0.05$

for the subjects emplyed by the different experimenters. While the mean scores on all dependent measures tended to be slightly higher for experimenter 2, the differences did not approach significance when examined with t-tests. In table 2, the mean scores on each of the dependent variables for the forward and backward orderings of the unpaired control groups are presented. Although the backward ordering of the CS and US produced higher mean scores on all dependent measures, t-tests applied to the differences in scores between the orderings were not statistically reliable. Since the liberal t-statistic yielded no significant effects in the two sets of analyses, the factors of Experimenter Differences and Forward-Backward Ordering were deleted from all subsequent analyses.

Habituation period: cross overs and activity.

Measures of cross overs and activity taken during habituation and before the introduction of the CS in the test phase were subjected to analysis of variance in order to determine the role of apparatus cues in the conditioning process. The mean number of cross overs during the habituation period for Groups 3.5 - P, 3.5 - U, 1.05 - P, 1.05 - U, and CS-A were 12.1, 21.1, 20.1, 22.2, and 26.9 respectively. Analysis of variance performed on the cross over measurement revealed that group differences were not significant ($F < 1.0$).

The mean activity scores during the habituation session for Groups 3.5 - P, 3.5 - U, 1.05- P, 1.05 - U, and CS-A were 18.4, 22.6, 28.7, 25.7, and 37.5 respectively. A two-way repeated measures analysis of variance (c.f. Table 3) indicated that

Table 2

Forward vs backward orderings of unpaired control groups on each of the post-CS dependent variables : mean scores and t-statistics.

Response Measure	Ordering Backward	Ordering Forward	Degrees of Freedom	t	Probability (two-tailed)
Freezing	227.00	101.80	16	0.848	$p > 0.05$
Escape	432.57	242.82	16	0.958	$p > 0.05$
Activity	8.70	5.30	16	1.103	$p > 0.05$

the differences between groups only approached significance [$F(4,40)=2.65, p < 0.47$]. Post-hoc orthogonal contrasts between groups inidcated that the marginal group main effect resulted from the fact that the groups that received shock (i.e. Groups 3.0-P, 3.0-U, 1.05-P, 1.05-U) had lower activity scores that the unshocked (Group CS-A) control group [$F(1,40)=7.63, p < .01$].

Figure 1 presents the mean activity over the 5 minute intervals of the habituation session and shows that the amount of activity had a negatively decelerated decrease over the habituation session. The ANOVA confirmed the graphical interpretation by revealing a significant effect of 5 minute intervals [$F(5,200)=113.42, p < 0.001$] which contained a significant linear [$F(1,40)=219.59, p < 0.001$], quadratic [$F(1,40)=91.70, p < 0.001$], and cubic [$F(1,40)=19.21, p < 0.001$] trend components. There were no interactions between groups and five minute intervals.

Test phase: cross overs and pre-CS activity.

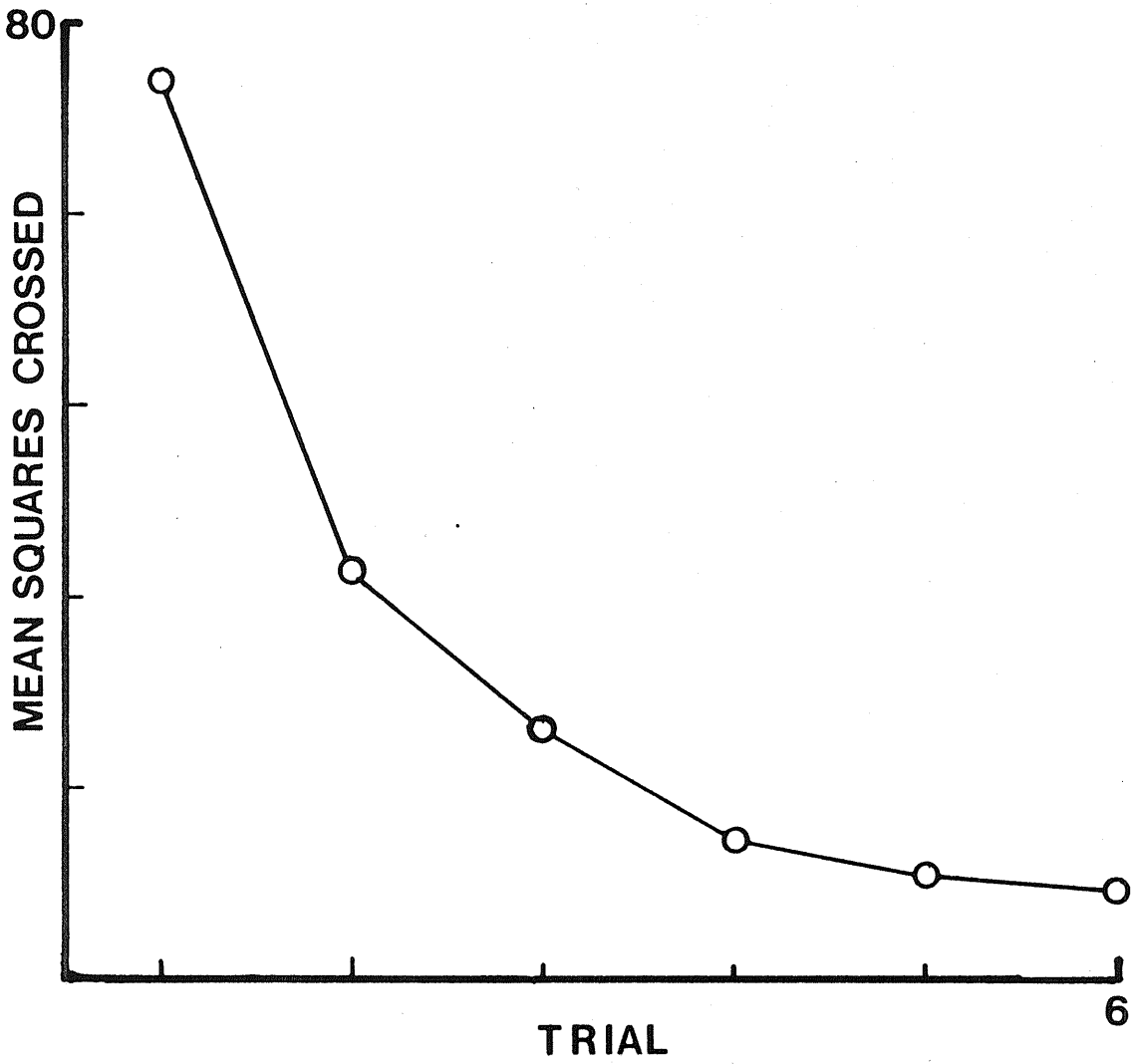
The mean number of cross overs during the pre-CS period over the test phase for Groups 3.5-P, 3.5-U, 1.05-P, 1.05-U and CS-A were 4.0, 6.4, 7.0, 9.5, and 8.6 respectively. A two-way repeated measures ANOVA (c.f. Table 4) indicated that the difference in cross overs between groups approached significance [$F(4,40)=3.54, p < 0.015$]. Post-hoc orthogonal contrasts between groups showed that subjects that received intense shock (i.e.

Table 3

ANOVA and trend analysis for activity during consecutive 5 minute intervals of the habituation session.

Source	df	MS	F	p less than
Groups	4	2808.89	2.65	0.047
Error: Between	40	1060.08		
5 min. Intervals (Int)	5	30076.00	113.42	0.000
Linear	1	116613.94	219.59	0.000
Quadratic	1	29528.66	91.70	0.000
Cubic	1	3391.06	19.21	0.000
Quartic	1	616.00	4.00	0.052
Quintic	1	230.57	1.62	0.210
Int. X Groups	20	303.19	1.14	0.308
Linear	4	572.67	1.08	0.380
Quadratic	4	396.50	1.23	0.313
Cubic	4	173.64	0.98	0.428
Quartic	4	83.53	0.54	0.706
Quintic	4	289.63	2.04	0.107
Error: Within	200	265.17		
Linear	40	531.05		
Quadratic	40	322.02		
Cubic	40	176.57		
Quartic	40	154.08		
Quintic	40	142.11		

Figure 1. Mean activity during habituation session: main effect of 5 minute intervals.


80
MEAN SQUARES CROSSED
6
TRIAL

Groups 3.5-P and 3.5-U) had fewer cross overs [$F(1,40)=9.52$, $p < 0.01$] than groups which received less intense shock (Groups 1.05-P and 1.05-U) or no shock at all (Groups CS-A).

Figure 2 depicts the mean number of cross overs over the 10 days of the test phase and shows that the number of cross overs slowly decreased until day 7 and subsequently increased. This U-shaped function was confirmed by the significant quadratic trend component [$F(1,40)=12.77, p < 0.001$] to the significant trials effect [$F(9,630)=2.95, p < 0.002$] in the ANOVA. The ANOVA contained no statistically significant interactions.

The mean pre-CS activity scores during the test phase for Groups 3.0-P, 3.0-U, 1.05-P, 1.05-U, and CS-A were 31.0, 47.9, 54.1, 64.4 and 59.3 respectively. A two-way repeated measures ANOVA (c.f. Table 5) indicated that the groups factor approached significance [$F(4,40)=3.05, p < 0.028$]. Post-hoc orthogonal contrasts showed that subjects which received intense shock (i.e. Groups 3.5-P and 3.5-U) were less active [$F(1,40)=8.63$, $p < 0.01$] than groups which received less intense shock (Groups 1.05-P and 1.05-U) or no shock at all (Group CS-A).

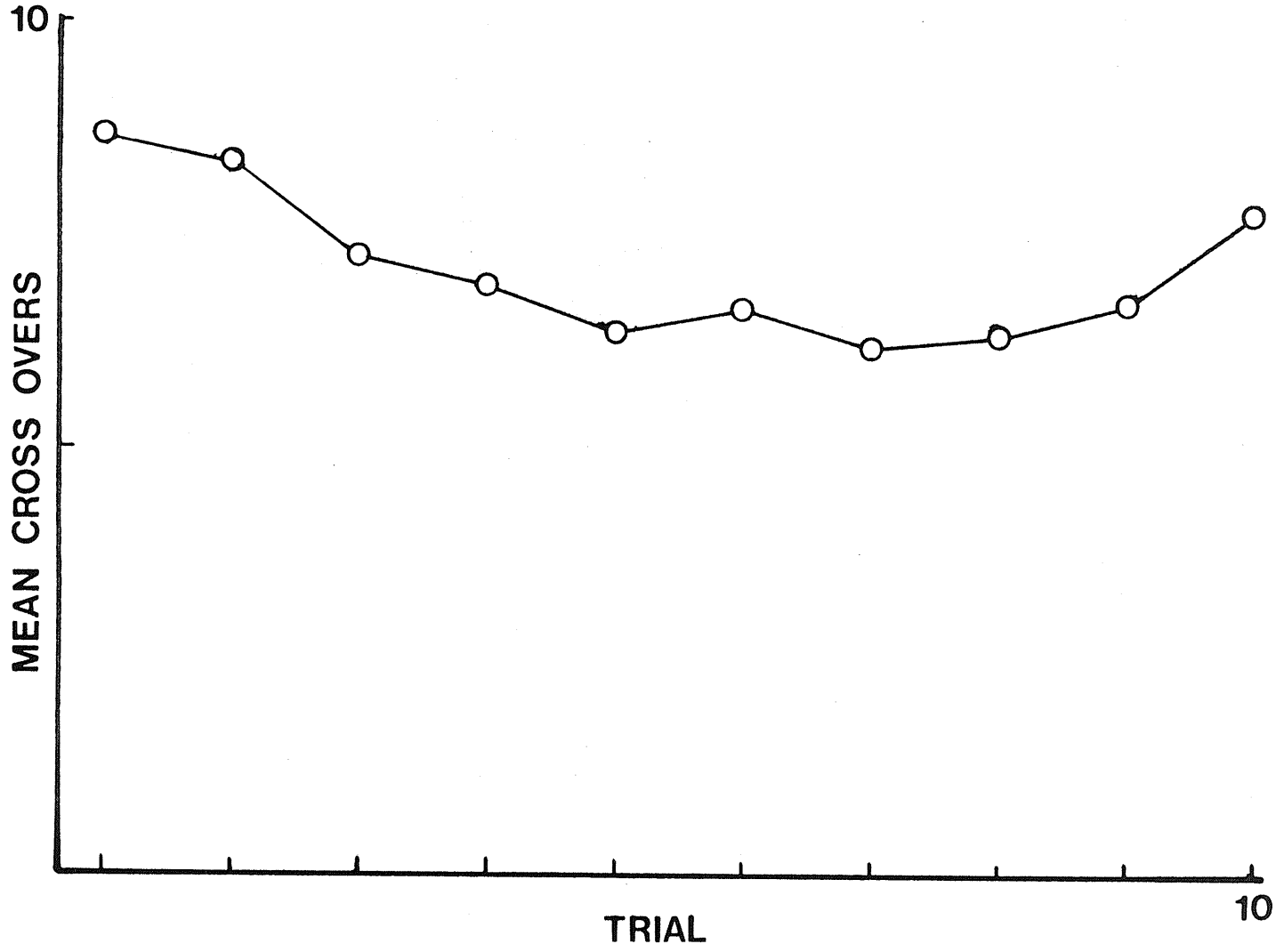
Figure 3 presents the mean pre-CS activity observed on each of the ten trials of the test phase. The figure suggest that, like the number of cross overs, the mean amount of activity slowly

Table 4

ANOVA and trend analysis for cross overs during test phase.

Source	df	MS	F	p less than
Groups	4	407.96	3.54	0.015
Error: Between	40	115.28		
Trials	9	35.29	2.95	0.002
Linear	1	100.06	2.96	0.093
Quadratic	1	192.74	12.77	0.001
Cubic	1	6.78	0.54	0.466
Quartic	1	0.66	0.08	0.780
Qunitic	1	8.81	1.44	0.283
Sextic	1	2.95	0.35	0.556
Septic	1	0.01	0.00	0.982
Octic	1	0.05	0.01	0.924
Novic	1	5.55	0.62	0.436
Trials X Groups	36	9.09	0.76	0.842
Linear	4	28.53	0.84	0.506
Quadratic	4	10.48	0.69	0.600
Cubic	4	5.91	0.47	0.757
Quartic	4	2.39	0.29	0.886
Quintic	4	6.84	1.12	0.362
Sextic	4	8.12	0.97	0.435
Septic	4	9.19	1.05	0.392
Octic	4	0.95	0.17	0.954
Novic	4	9.46	1.05	0.392
Error: Within	360	11.98		
Linear	40	33.84		
Quadratic	40	15.09		
Cubic	40	12.54		
Quartic	40	8.39		
Quintic	40	6.13		
Sextic	40	8.38		
Septic	40	8.72		
Octic	40	4.71		
Novic	40	8.98		

Figure 2. Mean number of cross overs in the test phase.


10
MEAN CROSS OVERS
TRIAL
10

decreased until day 7, when there was a subsequent slight increase in activity. The ANOVA confirmed the graphical interpretation by revealing a significant trials effect [$F(9,630)=7.81, p < 0.001$] which contained significant linear [$F(1,40)=9.48, p < 0.001$] , quadratic [$F(1,40)=27.57, p < 0.001$] , and quartic [$F(1,40)=8.85, p < 0.005$] trend components. (The quartic component reflects the fact that the amount of activity on day 3 was slightly less than day 4. This results in three points of inflection for the function, one at day 3, one at day 4, and one at day 7). The ANOVA also revealed a marginally significant Group X Trials interaction [$F(4,40)=1.62, p < 0.016$] which contained a highly significant quadratic component [$F(4,40)=5.10, p < 0.002$] . Figure 4 illustrates the Group X Trials interaction. From the figure, it can be seen that Groups 3.5-P, 3.5-U, and 1.05-P have the characteristic U-shaped function of the main effect. In contrast, the amount of activity for Group 1.05-U was an inverted U-shaped function of trials and Group CS-A appeared to have no consistent quadratic component over trials. These differences in the quadratic function for the groups yielded the Group X Trials interaction. Taken together, the absence of major between groups differences on cross overs and activity in habituation and before the introduction of the CS in the test phase suggest that apparatus cues played a minimal role in the conditioning process.

Multivariate between groups comparisons.

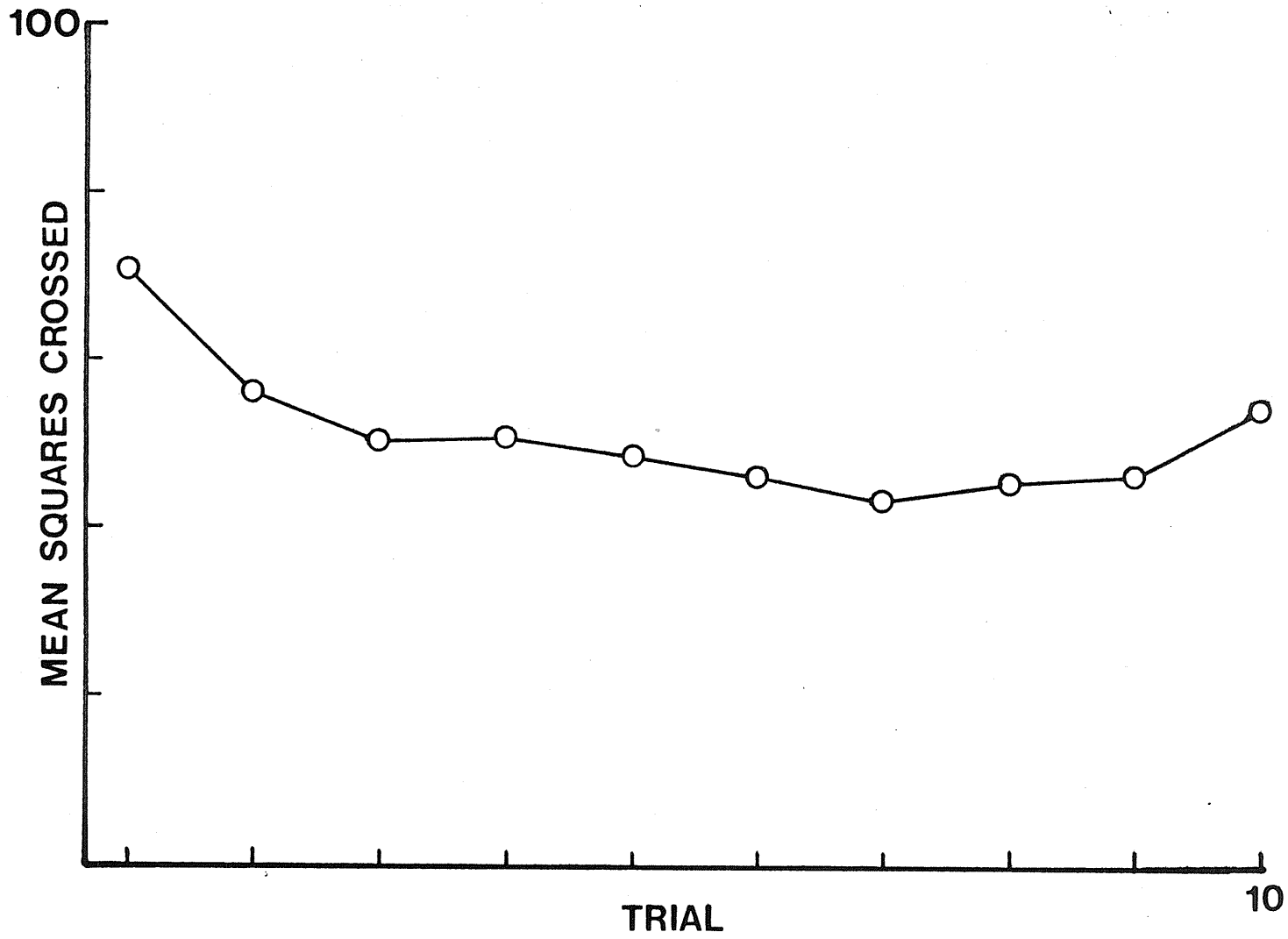
Hypotheses concerning specific differences between groups were investigated by means of a Multivariate Analysis of Variance.

Table 5

ANOVA and trend analysis for pre-CS activity during the test phase.

Source	df	MS	F	p less than
Groups	4	14989.66	3.05	0.028
Error: Between	40	4911.62		
Trials	9	2764.63	7.81	0.000
Linear	1	9606.67	9.48	0.004
Quadratic	1	13096.18	27.57	0.000
Cubic	1	25.40	0.09	0.764
Quartic	1	1758.05	8.85	0.005
Quintic	1	96.66	0.47	0.499
Sextic	1	0.66	0.00	0.987
Septic	1	244.98	1.16	0.288
Octic	1	4.79	0.02	0.882
Novic	1	49.27	0.15	0.702
Trials X Groups	36	572.03	1.62	0.016
Linear	4	599.97	0.59	0.670
Quadratic	4	2424.90	5.10	0.002
Cubic	4	720.87	2.58	0.052
Quartic	4	53.78	0.27	0.895
Quintic	4	29.23	0.14	0.966
Sextic	4	561.89	2.23	0.083
Septic	4	159.90	0.76	0.559
Octic	4	111.33	0.52	0.724
Novic	4	486.62	1.47	0.231
Error: Within	360	353.82		
Linear	40	1013.47		
Quadratic	40	475.06		
Cubic	40	279.04		
Quartic	40	198.59		
Quintic	40	207.50		
Sextic	40	252.01		
Septic	40	210.98		
Octic	40	215.71		
Novic	40	331.99		

Figure 3. Mean CS activity for each of the 10 days in the test phase.


100
MEAN SQUARES CROSSED
TRIAL
10

The computer program used for all Multivariate analyses was "MULTIVARIANCE, Univariate and Multivariate Analysis of Variance, Covariance and Regression. Version $\overline{V}$, Release 3." (Finn, 1976). Response measures employed in these analyses were duration of freezing, latency to escape and level of activity following CS onset. The means of each experimental group, collapsed across trials for freezing, escape, and activity are presented in Figures 5, 6, and 7 respectively. Duration of freezing and latency to escape were longest for Group 3.5-P with Groups 3.5-U, 1.05-P, 1.05-U, and CS-A following in approximate order of decreasing values. On the other hand, Group means on the activity variable appeared to follow no systematic pattern. Pearson's product-moment correlations among the dependent variables indicated that freezing and escape were highly interdependent ($\underline{r} = 0.90$) while activity and freezing ($\underline{r} = 0.09$) and activity and escape latency were not ($\underline{r} = 0.20$).

Multivariate contrasts were performed in order to determine where specific group differences lay. Group 3.5-P was found to differ from Group 1.05-P [$\underline{F}(3,38)=8.631, \underline{p} < 0.001$], Group 3.5-U [$\underline{F}(3,38)=4.474, \underline{p} < 0.009$] and group CS-A [$\underline{F}(3,38)=3.382, \underline{p} < 0.028$]. Group 1.05-P differed from 1.05-U [$\underline{F}(3,38)=3.598, \underline{p} < 0.022$] and Group CS-A [$\underline{F}(3,38)=3.404, \underline{p} < 0.027$].

Univariate F statistics for each of the comparisons are

Figure 4. Mean pre-CS activity for Groups 3.0-P, 3.0-U, 1.05-P, 1.05-U and CS-A as a function of trials in the test phase.

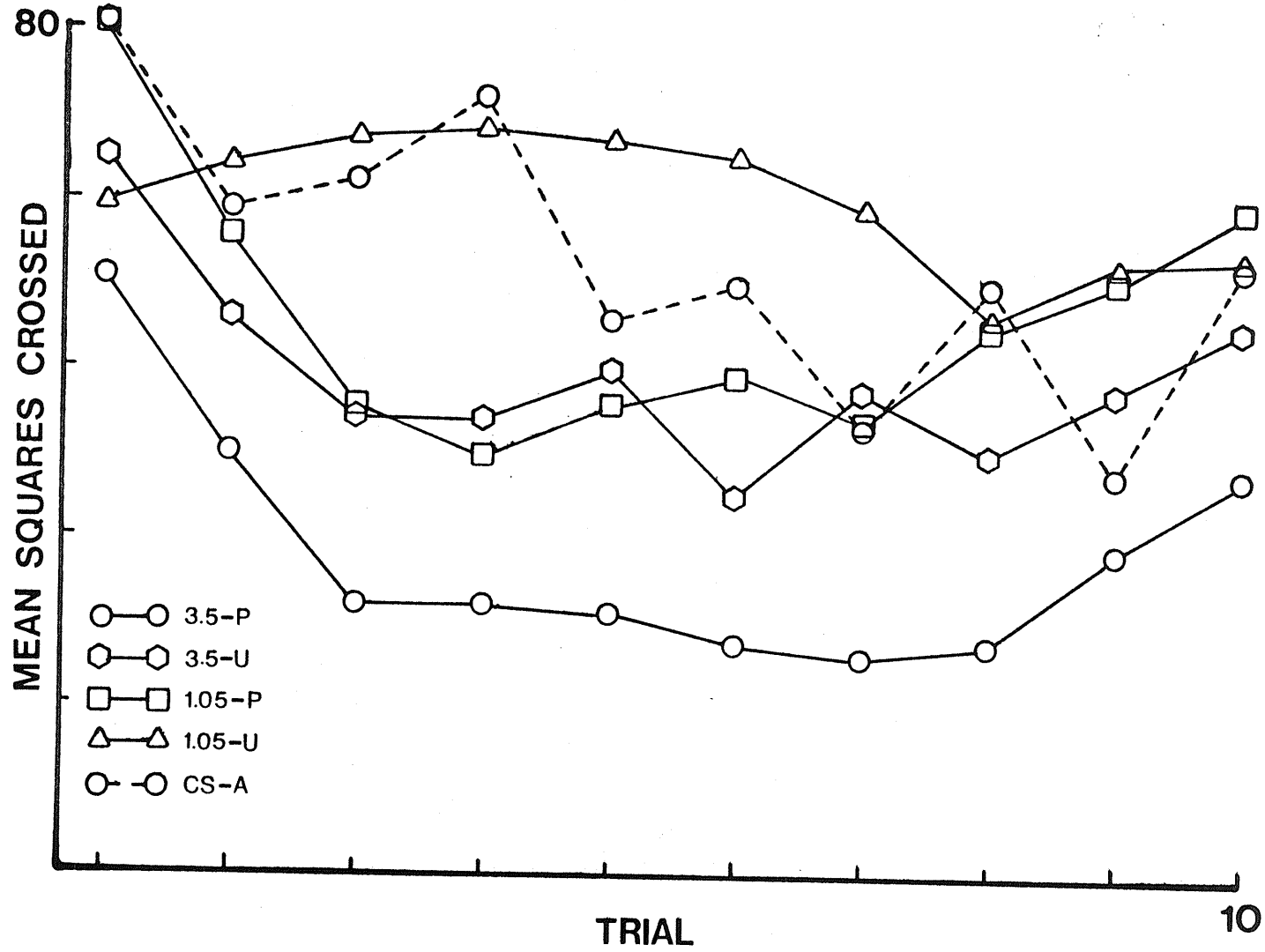

80
MEAN SQUARES CROSSED
3.5-P
3.5-U
1.05-P
1.05-U
CS-A
TRIAL
10

Figure 5. Mean duration of freezing in Groups 3.0-P, 3.0-U, 1.05-P, 1.05-U and CS-A in the test phase.

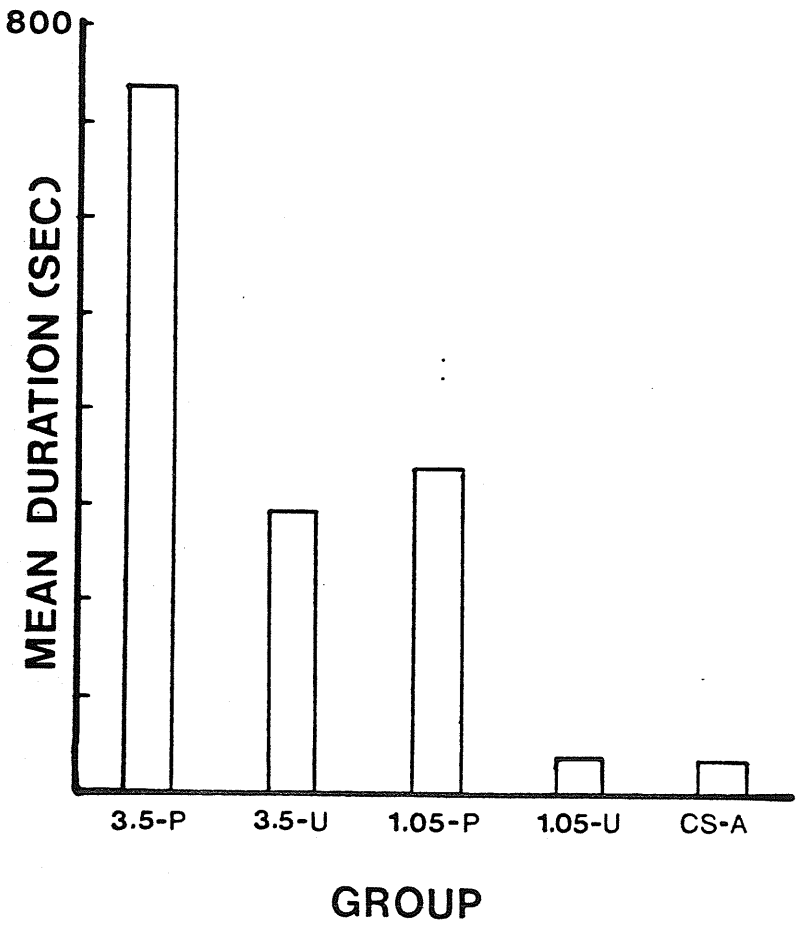

800
MEAN DURATION (SEC)
3.5-P
3.5-U
1.05-P
1.05-U
CS-A
GROUP

Figure 6. Mean latency to escape for Groups 3.0-P, 3.0-U, 1.05-P, 1.05-U and CS-A in the test phase.

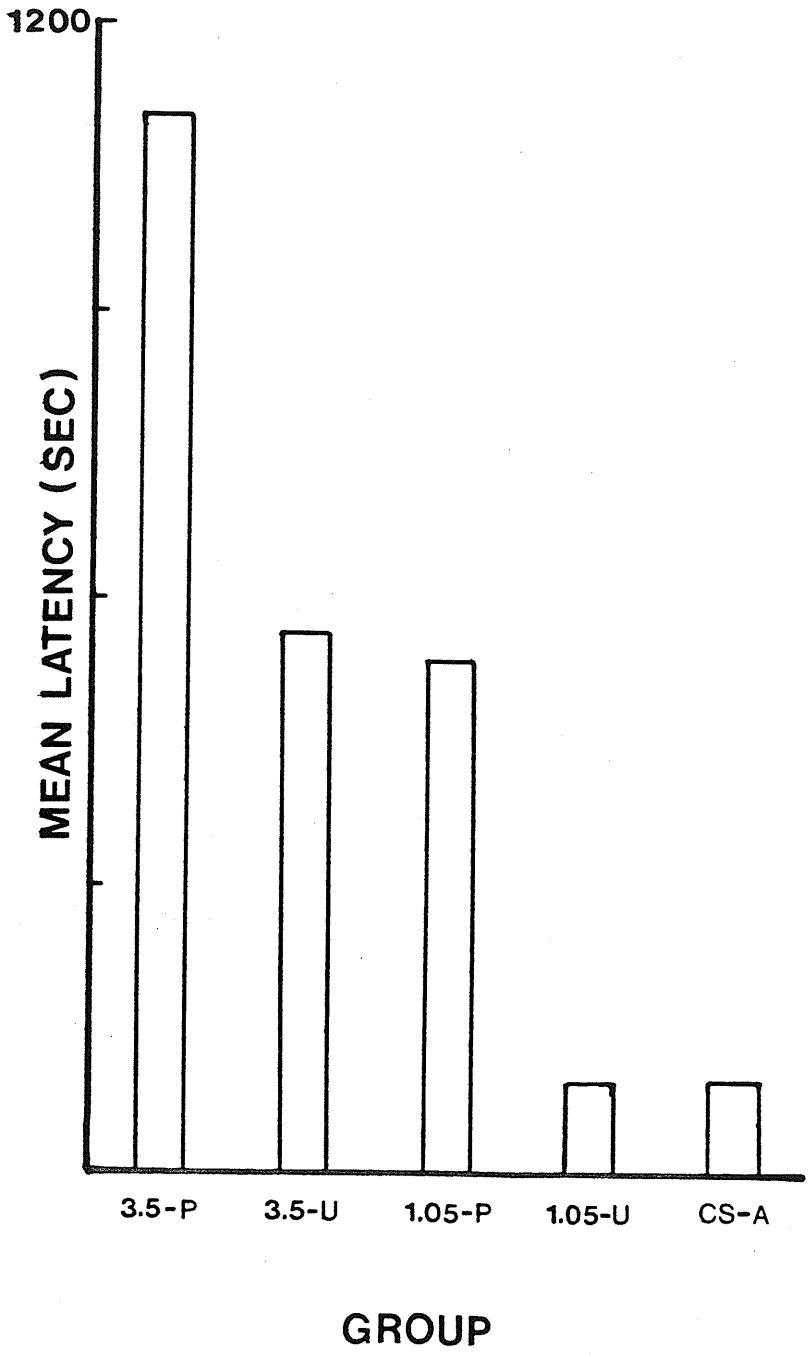
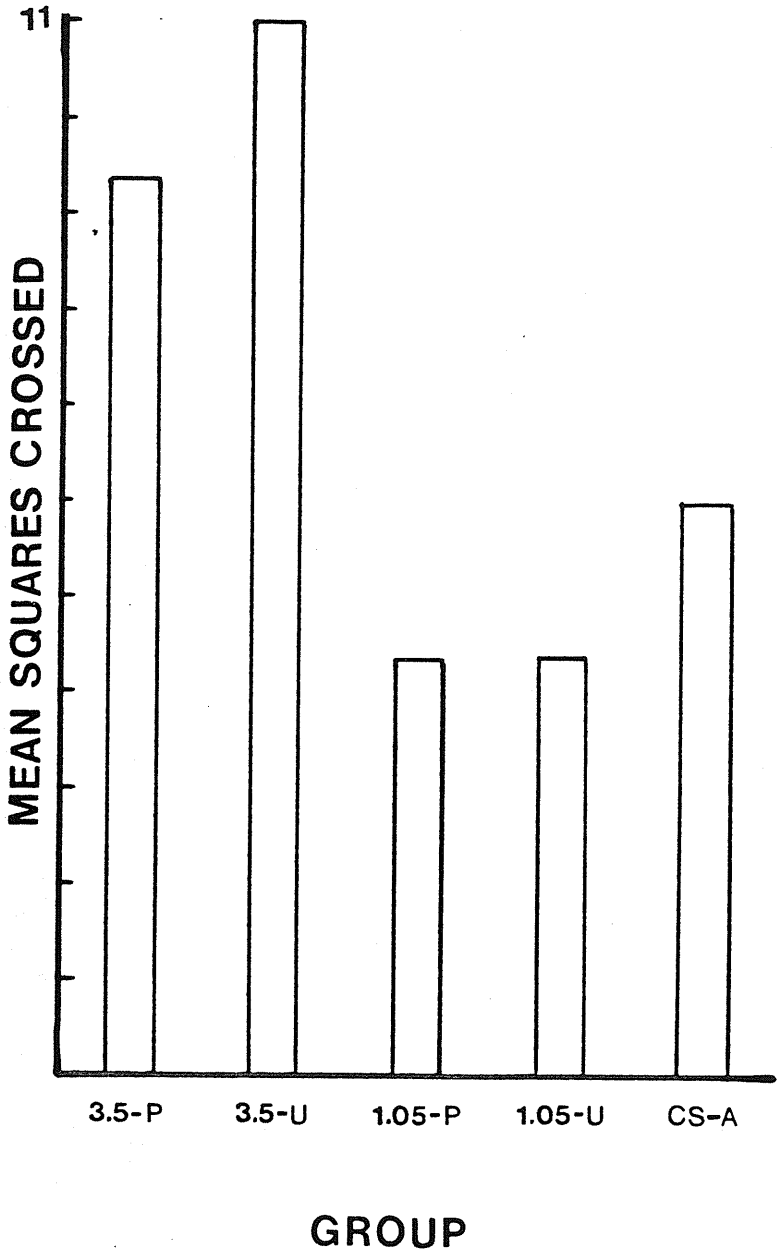

1200
MEAN LATENCY (SEC)
3.5-P
3.5-U
1.05-P
1.05-U
CS-A
GROUP

Figure 7. Mean amount of activity for Groups 3.0-P, 3.0-U, 1.05-P, 1.05-U and CS-A in the test phase.


11
MEAN SQUARES CROSSED
3.5-P
3.5-U
1.05-P
1.05-U
CS-A
GROUP

presented in Table 6. It should be noted from Table 6 that the largest univariable F's were obtained with the freezing dependent measure, that the freezing escape dependent measures yield significant values for all contrasts, and that the activity dependent measure only approached significance on one of the contrasts (3.05-P vs 1.05-P).

Although not of predictive concern, a discriminant analysis was performed on each between groups contrast to determine which variables contributed most strongly to group differences. Raw and standardized discriminant function coefficients are reported in Table 7. Table 7 indicates that for each comparison, the standardized discriminant function coefficient for freezing was two to three times the value for the next highest coefficient; that the standardized discriminant function coefficient for latency to escape was substantially greater than the standardized discriminant coefficient for amount of activity for three of the contrasts (i.e. 3.5-P vs 3.5-U; 3.5-P vs CS-A; 1.05-P vs CS-A); and that the standardized discriminant function coefficients for escape latency and amount of activity were about the same for two contrasts (3.5-P vs 1.05-P; 1.05-P vs 1.05-U).

Taken together, both the univariable F statistics and the discriminant function loadings indicated that freezing was the most heavily weighted variable with escape and activity in order of decreasing importance. The different durations of freezing be-

Table 6

Summary of univariate F statistics associated with the significant multivariate contrasts.

Contrast	Variable	df	MS	F	p less than
3.5-P vs 1.05-P	Freezing	1	154248350.34	22.88	0.000
	Escape	1	380409760.09	22.83	0.000
	Activity	1	29788.50	6.04	0.019
3.5-P vs 3.5-U	Freezing	1	86585342.83	12.85	0.001
	Escape	1	133466211.20	8.01	0.007
	Activity	1	1283.56	0.26	0.613
3.5-P vs CS-A	Freezing	1	68831342.00	10.21	0.003
	Escape	1	162866201.62	9.78	0.003
	Activity	1	3422.81	0.69	0.409
1.05-P vs 1.05-U	Freezing	1	69745771.60	10.35	0.003
	Escape	1	164804973.47	9.89	0.003
	Activity	1	7761.80	1.57	0.217
1.05-P vs CS-A	Freezing	1	70966784.96	10.53	0.002
	Escape	1	163641577.09	9.82	0.003
	Activity		1185.80	0.24	0.630

Table 7

Discriminant function coefficients for between group contrasts.

Contrast	Variable	Raw Coefficient	Standardized Coefficient
3.5-P vs 1.05-P	Freezing	-0.000259	-0.6735
	Escape	-0.000052	-0.2320
	Activity	-0.005159	-0.3623
3.5-P vs 3.5-U	Freezing	0.000546	1.1417
	Escape	-0.000122	-0.4962
	Activity	0.002325	-0.1633
3.5-P vs CS-A	Freezing	-0.000269	-0.6989
	Escape	-0.000078	-0.3192
	Activity	-0.000298	-0.0209
1.05-P vs 1.05-U	Freezing	-0.000286	-0.7427
	Escape	-0.000051	-0.2075
	Activity	-0.003737	-0.2624
1.05-P vs CS-A	Freezing	-0.000269	-0.6989
	Escape	-0.000078	-0.3192
	Activity	-0.00298	-0.0209

havior then, probably most parsimoniously explain the observed group differences in the MANOVA.

In summary, inspection of the Multivariate contrasts and Figures 5, 6, and 7 indicated that paired group performance (Groups 3.5-P and 1.05-P) significantly differed from their respective unpaired control contrasts (Groups 3.5-U and 1.05-U) and the unshocked control (Group CS-A). This, conditioned fear was obtained. In addition, the indices for conditioned fear were greater for the high intensity paired group (3.5-P) relative to the low intensity paired group (1.05-P). Thus a shock intensity effect was obtained.

Univariate analyses of Trials and Trials by Groups interactions for the post-CS dependent measures.

Univariate, two-way, repeated measures ANOVA's with orthogonal components for trend were employed to examine the effect of trials and trials by group interactions for freezing, escape, and activity, respectively.

Freezing.

Figure 8 presents the main effect of trials for each of the three dependent variables. The figure suggest that the mean amount of freezing increased over the first three days, and then decreased to a level (97.4 sec.) that was substantially lower than the initial value (289.5 sec.). The graphic interpretation

was confirmed by an ANOVA (c.f. Table 8) which contained a significant trials effect [$F(9,360)=4.72, p < .001$] which resulted from a highly significant linear trend component [$F(1,40)=13.5, p < 0.001$] and marginal nonsignificant quadratic trend component [$F(1,40)=4.27, p < 0.045$].

The ANOVA also yielded a marginal but nonsignificant Group X Trials interaction [$F(3,360)=1.63, p < 0.015$] which was composed of a highly significant linear trend component [$F(4,40)=418, p < 0.006$], but not a quadratic trend component [$F(4,40)=1.53, p > 0.20$]. The Group by Trials interaction is depicted in Figure 9. An examination of Figure 9 showed that Groups 1.05-U and CS-A had very low levels of freezing that did not change appreciably over trials; that the duration of freezing for Groups 3.5-P, 3.5-U and 1.05-P was much higher than for Groups 1.05-U and CS-A but after an initial increase over the first few trials, declined to the levels of Groups 1.05-U and CS-A and that Group 3.5-P had the highest level of freezing and the sharpest decrease. Thus the linear component to the Group X Trial interaction resulted from the flat functions for Groups 1.05-U and CS-A, the moderate declines for Groups 3.5-U and 1.05-P, and the rapid decline for Group 3.5-P. It should also be noted that since the Group X Trial interaction did not contain a quadratic trend component, the marginal quadratic trend component to the

Figure 8. The main effect of trials for the duration of freezing, latency to escape and amount of activity dependent measures. The abscissa for the freezing and escape measures is on the left side of the figure, while the abscissa for the activity measure is on the right side of the figure.

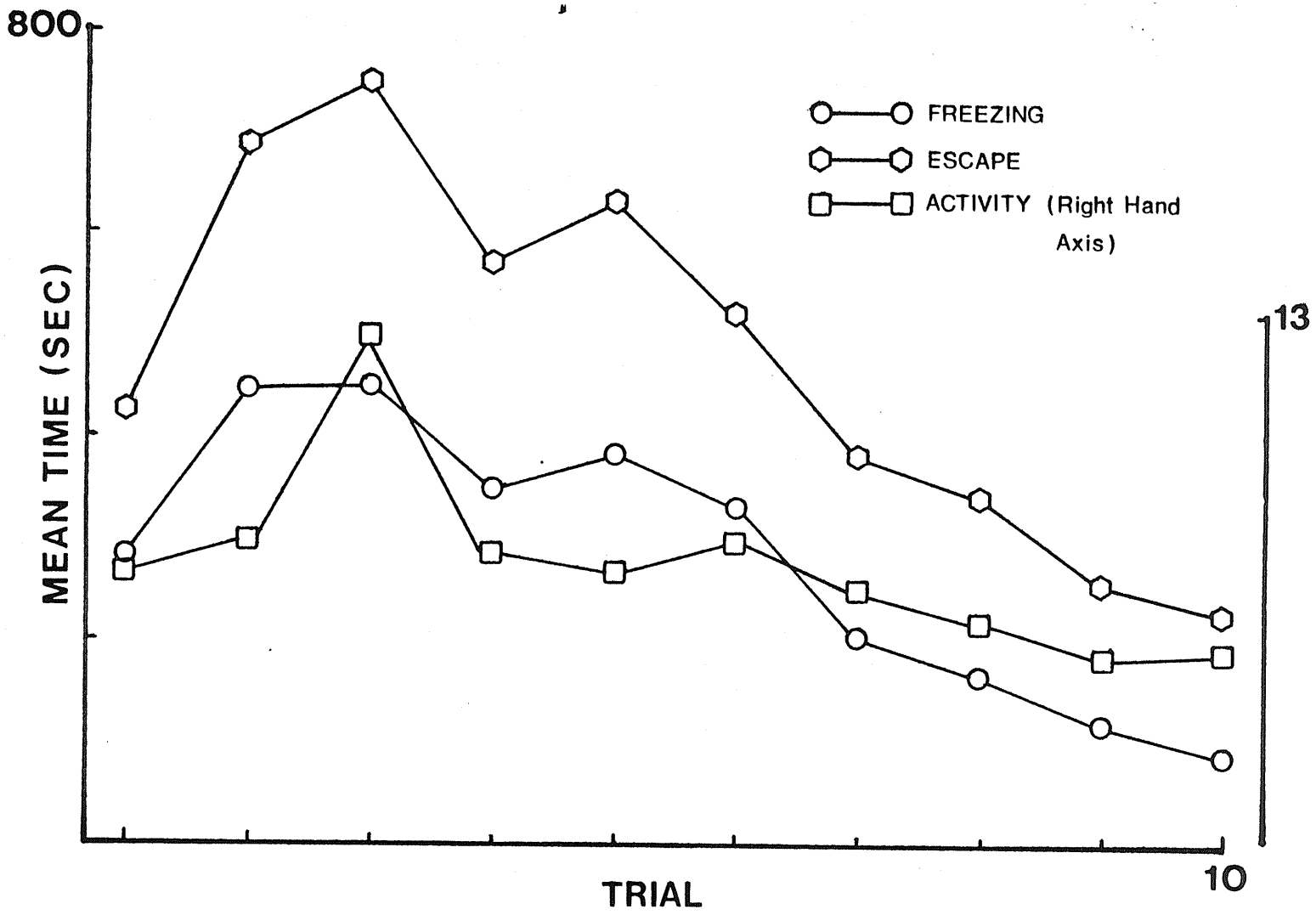
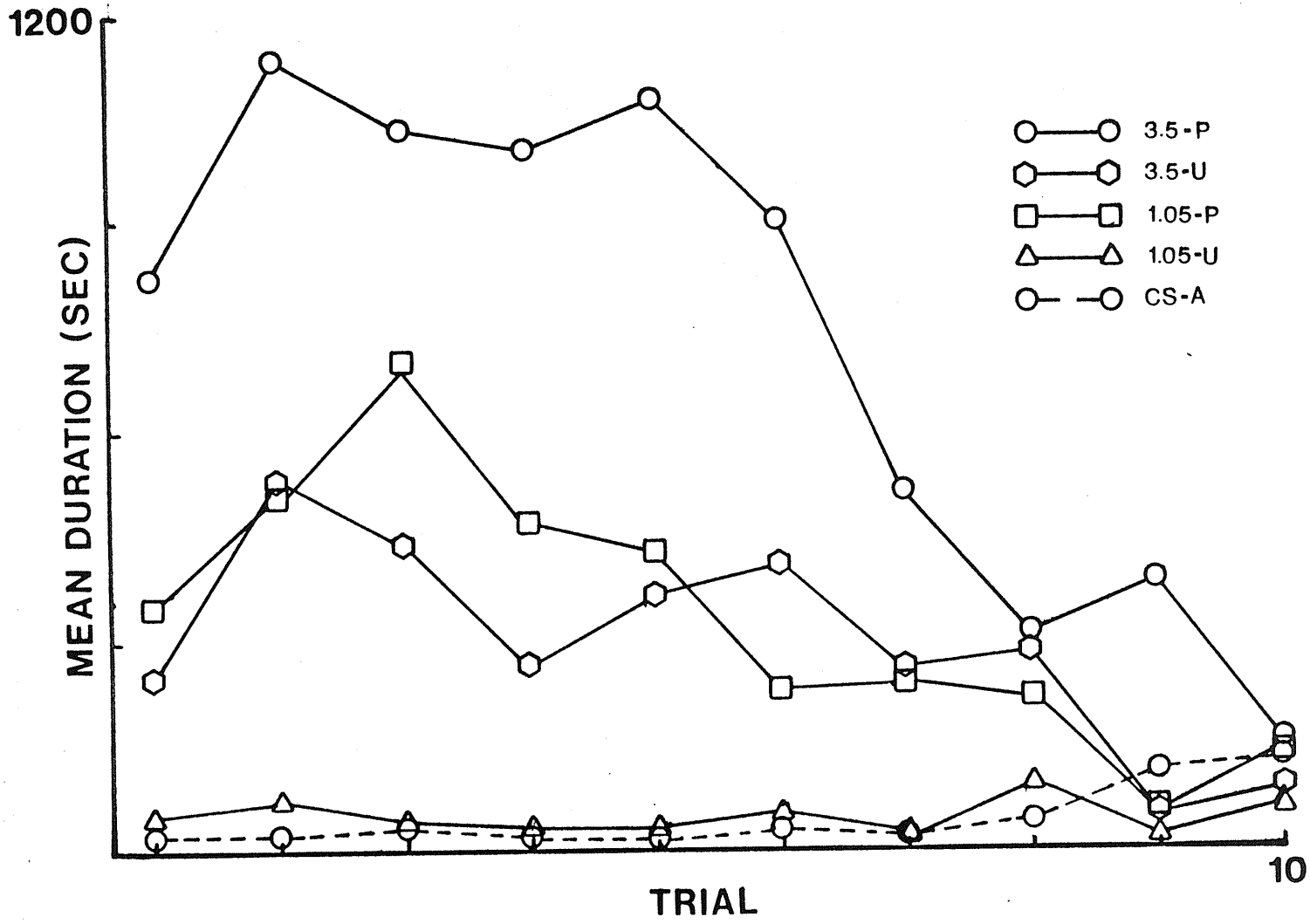

800
MEAN TIME (SEC)
FREEZING
ESCAPE
ACTIVITY (Right Hand Axis)
13
TRIAL
10

Table 8

ANOVA and trend analysis for the post-CS freezing duration in the test phase.

Source	df	MS	F	p less than
Groups	4	7341508.00	10.89	0.000
Errors: Between	40	674028.00		
Trials	9	748421.31	4.72	0.000
Linear	1	4709531.00	13.50	0.001
Quadratic	1	931682.00	4.27	0.045
Cubic	1	677445.00	3.84	0.057
Quartic	1	80018.00	0.66	0.421
Qunitic	1	70406.00	0.63	0.432
Sextic	1	78878.00	0.51	0.480
Septic	1	351.00	0.00	0.951
Octic	1	181790.00	2.13	0.152
Novic	1	5754.00	0.05	0.827
Trials X Groups	36	257553.31	1.63	0.015
Linear	4	1459834.00	4.18	0.006
Quadratic	4	334834.25	1.53	0.211
Cubic	4	154049.75	0.87	0.489
Quartic	4	17608.25	0.15	0.964
Quintic	4	86724.50	0.78	0.547
Sextic	4	215709.25	1.39	0.254
Septic	4	4204.00	0.05	0.996
Octic	4	18048.50	0.21	0.930
Novic	4	27022.25	0.23	0.922
Error: Within	360	158455.06		
Linear	40	348834.13		
Quadratic	40	218172.19		
Cubic	40	176585.75		
Quartic	40	121101.13		
Quintic	40	111631.75		
Sextic	40	155029.31		
Septic	40	90219.19		
Octic	40	85226.63		
Novic	40	119296.88		

Figure 9. The mean duration of freezing as a function of trials for Groups 3.5-P, 3.5-U, 1.05-P, 1.05-U and CS-A.


1200
MEAN DURATION (SEC)
3.5-P
3.5-U
1.05-P
1.05-U
CS-A
10
TRIAL

Trials main effect reflected the initial increase in the functions for Groups 3.5-P, 3.5-U and 1.05-P.

Escape.

An examination of the Trials main effect for the latency to escape measure depicted in Figure 8 reveals a function that parallels the Trials main effect for the freezing dependent measure. The figure indicates that after an initial rapid increase, the mean latency to escape decreased to a value (223.2 sec.) that was much lower than the initial value (432.2 sec.). Again the graphical interpretation was confirmed by the ANOVA (c.f. Table 9) which contained a significant Trials main effect [$F(9,360)=5.58, p < 0.001$] composed of a highly significant linear [$F(1,40)=14.98, p < 0.001$] and marginal nonsignificant quadratic [$F(1,40)=6.56, p < 0.014$] and cubic [$F(1,40)=5.65, p < 0.022$] trend components.

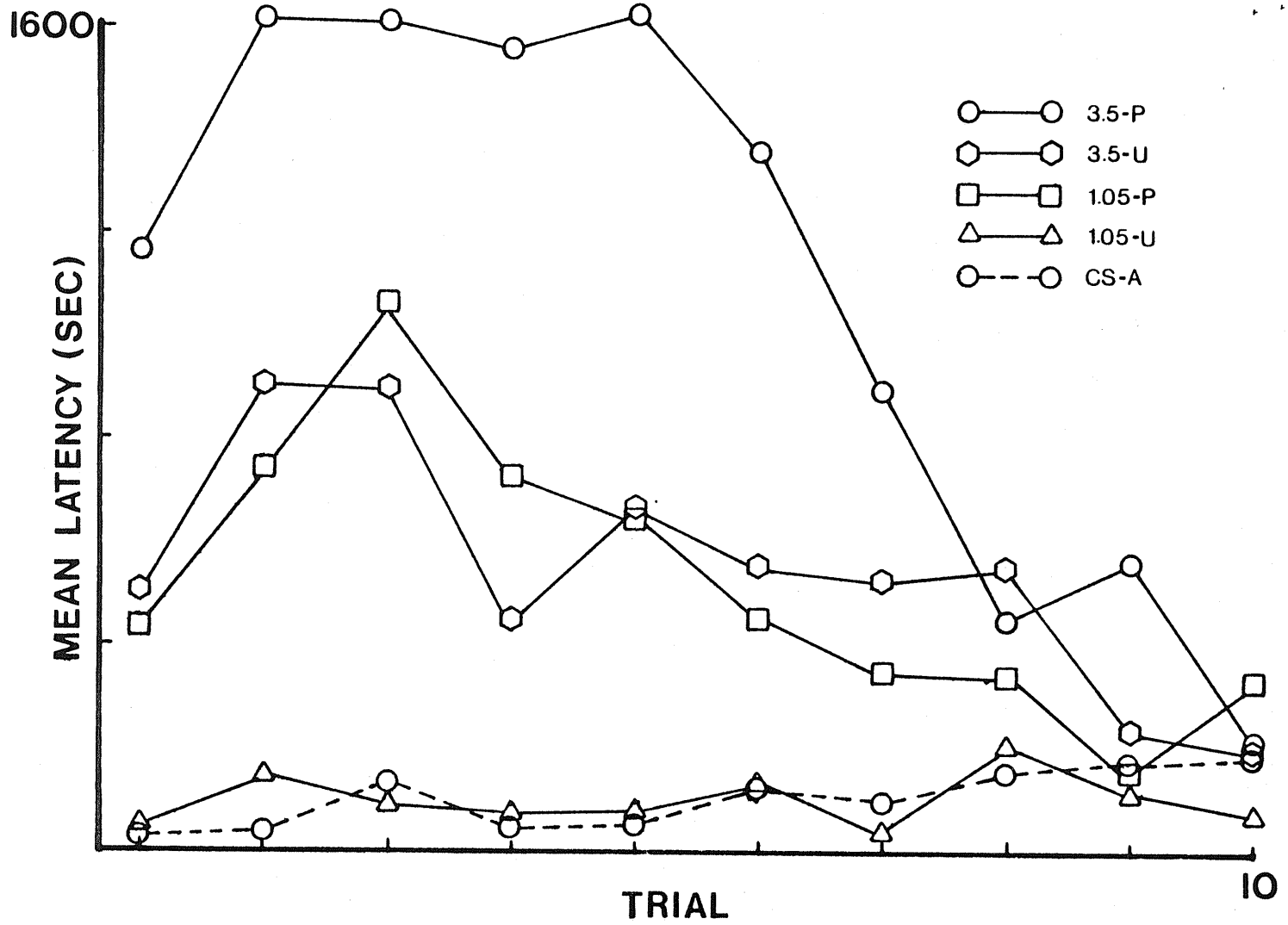
The ANOVA also indicated that the Trials X Group interaction was significant [$F(36,360)=2.10, p < 0.001$] and resulted from a significant linear [$F(4,40)=5.63, p < 0.001$] trend component. The significant Trials X Group interaction is depicted in Figure 10. The figure shows that the mean escape latencies for Groups 1.05-U and CS-A were short and did not change over trials. In contrast, the mean escape latencies for groups 3.5-P, 3.5-U, and 1.05-P were much longer, with the 3.5-P Group having the longest latencies, and after an initial increase, decreased to about the levels of Groups 1.05-U and CS-A. The flat functions over trials for

Table 9

ANOVA and trend analysis for the post-CS escape latencies in the test phase.

Source	df	MS	F	p less than
Groups	4	15672328.00	9.41	0.000
Error: Between	40	1665903.00		
Trials	9	1519568.00	5.58	0.000
Linear	1	8120416.00	14.98	0.000
Quadratic	1	2614952.00	6.56	0.014
Cubic	1	1904960.00	5.65	0.022
Quartic	1	230900.00	1.05	0.311
Quintic	1	181155.00	1.02	0.319
Sextic	1	43439.00	0.19	0.668
Septic	1	11252.00	0.06	0.810
Octic	1	514863.00	3.29	0.077
Novic	1	54237.00	0.28	0.602
Trials X Groups	36	572908.00	2.10	0.000
Linear	4	3054320.00	5.63	0.001
Quadratic	4	931886.00	2.34	0.072
Cubic	4	506458.75	1.50	0.220
Quartic	4	103906.75	0.47	0.755
Quintic	4	115215.50	0.65	0.631
Sextic	4	301517.00	1.30	0.288
Septic	4	23466.00	0.12	0.974
Octic	4	65367.75	0.42	0.795
Novic	4	54093.25	0.28	0.892
Error: Within	360	272503.31		
Linear	40	542095.56		
Quadratic	40	398776.56		
Cubic	40	337409.06		
Quartic	40	219498.88		
Quintic	40	177701.19		
Sextic	40	232706.13		
Septic	40	192192.63		
Octic	40	156458.19		
Novic	40	195755.75		

Figure 10. The mean latency to escape as a function of trials for Groups 3.5-P, 3.5-U, 1.05-P, 1.05-U, and CS-A.


1600
MEAN LATENCY (SEC)
3.5-P
3.5-U
1.05-P
1.05-U
CS-A
TRIAL
10

Groups 1.05-U and CS-A, the gradual decline in escape latencies for Groups 3.5-U and 1.05-P and the large rate of decrease for Group 3.5-P produced the linear interaction component. Again it must be noted that the Trial X Group interaction did not contain a quadratic component, and therefore, the quadratic component to the main effect must be due to the initial increase in escape latencies for Groups 3.5-P, 3.5-U, and 1.05-P.

Activity.

The trials main effect for the activity measure is also shown in Figure 8. Again an inverted U-shaped functions was observed with the mean amount of activity showing a small rise wich was followed by a very gradual and irregular decrease over trials. The change in mean amount of activity over trials did not produce a Trials main effect [$F(9,360)=1.47, p > 0.15$] in an ANOVA (c.f. Table 10) applied to the activity dependent variable. However, the trend analysis yielded marginally nonsignificant linear [$F(1,40)=5.44$, $p < 0.025$] and quartic [$F(1,40)=4.37, p < 0.043$] components which indicated that slightly reliable changes in activity were occurring over trials.

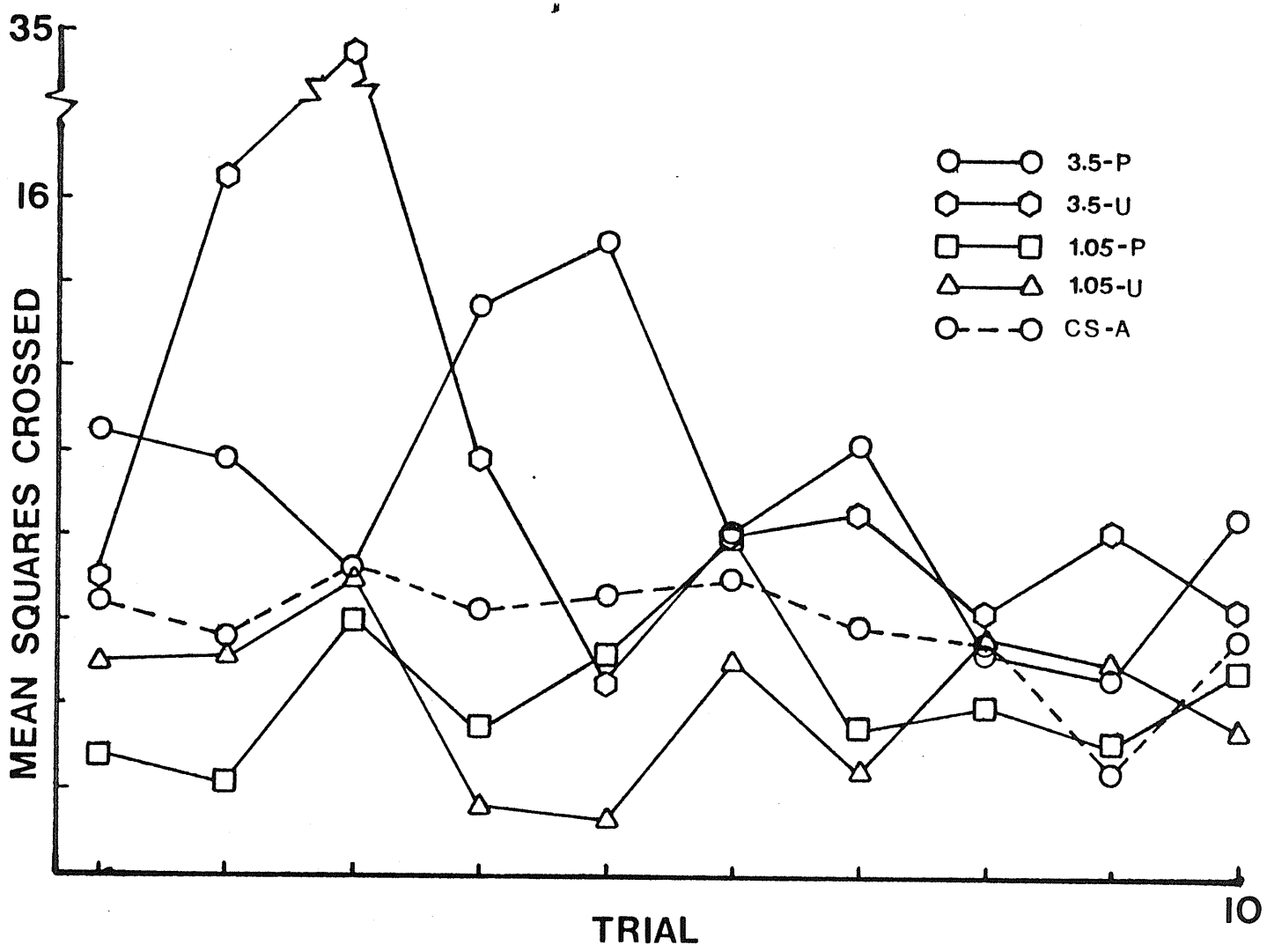
Figure 11 illustrates the nonsignificant Trials X Groups interaction. The figure indicates that the mean amount of activity for each group was not a smooth function of trials. The activity level functions were irregular, with the greatest

Table 10

ANOVA and trend analysis for the mean amount of post-CS activity in the test phase.

Source	df	MS	F	p less than
Groups	4	815.73	1.65	0.180
Error: Between	40	493.08		
Trials	9	197.38	1.47	0.157
Linear	1	559.23	5.74	0.025
Quadratic	1	72.67	1.41	0.243
Cubic	1	437.64	4.37	0.043
Quartic	1	90.29	0.53	0.470
Qunitic	1	29.81	0.71	0.406
Sextic	1	105.84	0.44	0.510
Septic	1	320.35	0.97	0.330
Octic	1	154.63	1.26	0.269
Novic	1	5.91	0.12	0.732
Trials X Groups	36	166.11	1.24	0.170
Linear	4	206.27	2.01	0.112
Quadratic	4	23.22	0.45	0.772
Cubic	4	210.96	2.11	0.098
Quartic	4	483.65	2.86	0.036
Quintic	4	20.19	0.48	0.752
Sextic	4	92.74	0.39	0.816
Septic	4	329.40	1.00	0.420
Octic	4	96.97	0.79	0.540
Novic	4	31.59	0.64	0.639
Error: Within	360	134.25		
Linear	40	102.85		
Quadratic	40	51.71		
Cubic	40	100.04		
Quartic	40	169.35		
Quintic	40	42.23		
Sextic	40	239.48		
Septic	40	329.89		
Octic	40	123.14		
Novic	40	49.57		

Figure 11. Mean amount of post-CS activity in the test phase for Groups 3.5-P, 3.5-U, 1.05-P, 1.05-U, and CS-A.


35
16
MEAN SQUARES CROSSED
3.5-P
3.5-U
1.05-P
1.05-U
CS-A
TRIAL
10

fluctuations occurring over the first five days. The absence of significant effects for the activity dependent variable (c.f. Table 10) would appear to corroborate the low, standardized discriminant function coefficients assigned to this variable (c.f. Table 7).

Discussion

The principle results of the experiment were as follows: (1) during the habituation session there was a slight general suppressive effect of shock on activity levels that did not substantially affect the decreases in activity over the session; (2) during the pre-CS period in the test phase, high shock intensity produced lower levels of crossovers and activity relative to low and no shock groups; (3) during the pre-CS period in the test phase, the main effect of trials showed first a decrease, and then an increase for both the number of crossovers and the amount of activity; (4) during the post-CS period in the test phase, the MANOVA revealed that paired group performance was significantly higher than the control groups, and that the high shock intensity paired group (3.5-P) had significantly higher measures than the low shock intensity paired group (1.05-P); (5) the discrimenant analysis indicated that differences between groups were primarily due to changes on the freezing and escape dependent measures; (6) during the post-CS period in the test phase, the main effect for trials for the freezing, escape, and activity measures, showed small initial increases in levels and subsequent large declines; and finally (7) for the freezing and escape measures, Group 3.5-P exhibited a large decline, Groups 3.5-U and 1.05-P showed moderate declines, whereas, Groups 1.05-U and CS-A displayed constant, low-level performance, over trials.

These observations will be expanded upon in the following paragraphs.

The absence of highly significant between group main effect differences on measures of crossovers and activity taken during the habituation session suggests that apparatus cues played a minimal role in the subsequent test phase of the experiment. Although post-hoc orthogonal contrasts revealed that animals which received shock during conditioning displayed reliably lower levels of activity than the nonshock control group, there were no differences between the shocked groups. Accordingly, the suppressive effect cannot be attributed to a direct associative effect, and more likely was consequence of experiencing shock. Bolles (1971) who also observed a shock produced decrement in activity levels, suggested that the suppression was due to a general increase in "emotionality" following shock presentation rather than to any associative effects of conditioning.

Similarly, analysis of the number of crossovers and levels of pre-CS activity observed during the test phase were marginally non-significant. Post-hoc orthogonal contrasts revealed that Groups 3.5-P and 3.5-U had fewer crossovers than Groups 1.05-P, 1.05-U and CS-A, indicating a suppressive effect of increasing shock intensity. The same phenomenon was observed in relation to the pre-CS activity measure, with observed levels of activity being slightly lower for Groups 3.5-P and 3.5-U than Groups 1.05-P,

1.05-U and CS-A. Again, these results point to a suppressive effect of increasing shock intensity upon behavior. Perhaps the most parsimonious account of the observation that high intensity shock has a more disruptive effect on behavior than low intensity shock is the "emotionality" mechanism proposed by Bolles (1971) and McAllister (1971). If "emotionality" results from experiencing shock and increases in "emotionality" lead to disruption of activity levels, then it follows that more intense shock should produce greater "emotionality", and therefore greater disruption of activity measures. While the data is consistent with the "emotionality" hypothesis, the most striking feature of the pre-CS data in the test phase is that between group differences are not very pronounced.

However once the CS was presented, dramatic differences between groups were observed. The MANOVA indicated that the post-CS performance for paired groups was significantly greater than for the unpaired and CS-alone controls. Since the primary weighting in the MANOVA were the freezing and escape measures, the greater performance by the paired groups reflected longer periods of freezing and longer escape latencies. Since freezing and escaping are indices of fear (c.f. Walsh and Cummins, 1976), the CS produced differences in freezing and escaping between the paired and control groups indicated that conditioned fear was

established in the experiment. In addition, since Group 3.5-P had significantly higher performance indicators than Group 1.05-P, the experiment demonstrated that the magnitude of conditioned fear was directly related to the intensity of the paired US. This result is consistent with the monotonic function between shock intensity and amount of conditioned fear reported by McAllister and McAllister (1971), Moyer and Korn (1969) Theios et. al. (1966), and Zammit-Montebello et. al. (1969).

The assumption that fear was conditioned mainly to the tone CS is given further support by the finding that major, between groups differences, particularly on the freezing and escape variables, appeared only after the introduction of the CS during testing. This further suggests that the effects of apparatus cues on behavior were minimized and that the associative effects to the CS primarily accounted for the observed results.

The finding that shock (particularly intense shock) evokes a freezing response corroborates reports by Blanchard and Blanchard (1968a,1968b, 1969), Bolles (1971) and Myer (1971). In the present study, the subjects' typical reaction to CS offset was immediate immobility, especially in Group 3.5-P. Some animals from this group remained immobile for up to 59.3 minutes; a result which would have been overlooked had a ceiling been imposed on trial length as in the Reynierse (1966) and Rohrbaugh and Riccio

(1970) experiments.

Although some animals did escape before the termination of the CS, these instances were almost exclusively restricted to rats from the CS-A and 1.05-P groups. In so far as the present experiment is concerned, latency to escape was necessarily a function of the length of the freezing response; short latencies to escape being incompatible with longer durations of freezing as escape ended each trial. This is consistent with the standardized discriminant function coefficients assigned these variables (c.f. Table 7).

The discriminant function coefficients indicated that the least effective dependent measure was the post-CS activity variable. On the basis of Walsh and Cummins (1976) review it was predicted that the amount of activity would increase, as conditioned fear extinguished. However, only slight changes in activity over trials and no between group differences were noted. Close examination of the procedure employed here suggests that the absence of differences in levels of activity between groups might be an artifact of the experimental design. Assuming that activity did increase as fear decreased, then, the probability of the animals' stepping through the door separating the two sides of the chamber (a valid form of activity as defined here) would presumably increase. Therefore, on each trial, a rise in

levels of activity could lead to the termination of the test before the presumed elevations in activity could be fully recorded. Accordingly, had a criterion other than escape be used to end each trial, the activity measure might have more accurately reflected levels of conditioned fear, as expected.

In the test phase of the experiment, Eysenck's (1968, 1976) incubation of fear hypothesis would predict increasing durations of freezing, increasing latencies to escape and decreasing levels of activity with repeated CS alone presentations. This prediction was not supported. The data reported here strongly suggest, instead, that fear reactions to the CS, as indexed by the response measures employed here, decreased to levels indistinguishable from subjects which received no initial fear conditioning. It is apparent that the conditioned fear extinguished rather than grew more severe with repeated CS alone presentations.

While the initial trials of the experiment might be taken as support for Eysenck's hypothesis, the increase in durations of freezing and latency to escape (c.f. Figures 9 and 10) cannot be attributed to the associative effects of conditioning since Group 3.5-U exhibited a similar increase in responding. Since the incubation of fear hypothesis invokes an associative mechanism, the parallel functions for associative and nonassociative groups precludes an incubation of fear interpretation. The observation

of initial increases in performance indices early in extinction is not unique to the present study. Similar increases in responding early in extinction have been observed in studies employing an avoidance contingency (Bolles, 1971) and in classical conditioning preparations (Tait, Wall and Gormezano, 1972). Bolles (1971) attributed the effect to "emotionality" which he presumed to be something other than fear itself. The hypothesized effect of "emotionality" was assumed to dissipate in a few hours, although no precise time course was specified. However, if the time course of "emotionality" increases with the intensity of the US, then it might be expected that a very severe shock, such as used in the present study, might produce a long lasting emotional reaction.

The fact that those animals which received 3.5mA shock displayed the longest durations of freezing and latency to escape on Trial 2 gives the "emotionality" argument further support.

Since the incubation of fear construct is an associative hypothesis, Eysenck (1968, 1976) would predict no increase in duration of freezing or latency to escape or a reduction in activity with repeated CS alone presentations for groups which did not receive CS-US pairings (i.e. Groups 3.5-U, 1.05-U and CS-A). The performance of Groups 1.05-U and CS-A are consonant with this position in that they demonstrated low initial levels of duration of freezing and latency to escape which further declined with repeated trials.

Eysenck's (1968) theory also accounts for the finding that conditioned fear extinguished with repeated CS alone presentations in Group 1.05-P. With the moderate intensity (1.05 mA) of the US experienced by subjects in this group Eysenck would predict that the decremental effects of CS presentation outweighed any incrementing tendencies which may be present in the situation. It is assumed, in this case, that the CR to the US was not of sufficient magnitude to effectively reinforce the CS and further increment the CR.

However, it is the behavior of Group 3.5-P which presents some difficulty for Eysenck's (1968) hypothesis. According to Eysenck, incubation of fear should result from the use of a high intensity US (Eysenck, 1976), a CS of short duration (c.f. Rohrbaugh and Riccio, 1970; Rohrbaugh et. al., 1972) and repeated CS alone presentations (c.f. Reynierse, 1966; Silvestri et. al., 1970). In the present study, each of these conditions were met and yet the conditioned fear as indexed by increasing durations of freezing and latencies to escape extinguished with repeated CS alone presentations.

Perhaps the most parsimonious interpretation would be to attribute the absence of incubation of fear in Group 3.5-P to the effects of extinction and assume that Eysenck's (1968,1976) theory is invalid. Alternatively, it could be argued, that the absence of incubation was due to the insufficient intensity of

the US, and that this experiment was not an adequate test of the theory. It should be noted, however, that the shock employed here was of considerably greater intensity than levels of shock typically employed in Pavlovian conditioning preparations (approximately 1.0 mA or less). The theoretical interpretation would be that incubation did not result because the CR produced by the US was not of sufficient magnitude to overcome the decrementing tendencies of the extinction process. Taken together, however, results reported here lead to the conclusion, also supported by McAllister and McAllister (1967), that incubation of conditioned fear has yet to be reliably demonstrated.

In conclusion, the absence of incubation of fear calls into doubt Eysenck;s (1968, 1976) interpretation of data reviewed previously (eg. Campbell et al, 1964; Napalkov, 1963). It may well be that an incubation-like phenomenon is the product of situational and procedural variables as yet unknown. In the final analysis, however, Eysenck's (1968) theory of incubation/neurosis can only be properly assessed in the light of repeated and varied investigations which attempt to ascertain the boundary conditions necessary for the reliable production of the phenomenon. The present study does suggest, however, that the multivariate approach will be an invaluable tool in future experimentation.

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