SLEEPY AND HOSTILE: THE EFFECTS OF REM DEPRIVATION ON SHOCK-ELICITED AGGRESSION

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SLEEPY AND HOSTILE:
THE EFFECTS OF REM DEPRIVATION
ON SHOCK-ELICITED AGGRESSION

An Abstract

Presented to

the Graduate Council of

Austin Peay State University

In Partial Fulfillment

of the Requirements for the Degree

Master of Arts

in Psychology

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by

Mary Nell Travis Mollenhour

June 1976

ABSTRACT

A study was designed to investigate the effects of rapid eye movement (REM) sleep deprivation on shock-elicited aggression in rats. Subjects were randomly assigned to 4 equal groups and deprived of REM sleep for 0, 24, 48, or 72 hours, respectively. Experimental animals were deprived of REM sleep by maintaining them on small platforms surrounded by water. Control animals (0 REM deprivation) were maintained under similar conditions with the exception that the platforms were sufficiently large to allow these subjects to obtain REM sleep. Subsequent to the deprivation procedures, all subjects were restrained and tested individually for shock-elicited aggression.

Results of statistical analyses suggested that the amount of shock-elicited aggression shown by rats is an increasing linear function of REM deprivation up to the 72 hours limit employed. This relationship was found for both time spent making aggressive contacts as well as total number of aggressive displays. In an attempt to explain these results, attention was given to the biochemical and physiological processes presumed to underlie REM sleep deprivation as it is affected by shock-elicited aggression. Specifically, the effects of stress and catecholamine level were considered.

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June 1976

To the Graduate Council:

I am submitting herewith a Thesis written by Mary Nell Travis Mollenhour entitled "Sleepy and Hostile: The Effects of Rem Deprivation on Shock Elicited Aggression". I recommend that it be accepted in partial fulfillment of the requirements for the degree of Master of Arts, with a major in Psychology.

Stephen F. Davis
Major Professor

We have read this thesis and recommend its acceptance:

Thomas L. Jackson
Second Committee Member

Garland E. Haas
Third Committee Member

Accepted for the Council:

Wayne C. Stacy
Dean of the Graduate School

ACKNOWLEDGEMENTS

I would like to express my sincere appreciation to my major professor, Dr. Stephen F. Davis, whose patience amazed me, to my husband, Mike, whose affectionate chiding finally jolted me into action, and to my dog, Chopper, whose friendly lick was there in spite of it all.

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CHAPTER I

INTRODUCTION

The phenomenon of aggression has piqued man's curiosity for centuries. The Biblical account of the slaying of Abel by his brother Cain clearly demonstrates that concern over aggression is not contemporary in origin. Recently, Moyer (1968) has described aggression as "behavior which leads to, or appears to an observer to lead to, damage or destruction of some goal entity". Although this evaluation appears to emphasize its malevolent aspects, Lorenz's (1966) commentary on intraspecific aggression suggests that such behaviors should be included in the repertoire of life-preserving instincts of the organism. However, he also points out that, like any other adaptive system, intraspecific aggression may accidentally serve a deleterious, destructive function.

One particular type of intraspecific aggression that has received attention during recent years is pain-elicited aggression. Subsequent to the publication of findings by Ulrich and Azrin (1962) concerning the stereotyped fighting behavior of rats in response to electric shock (i.e., when exposed to foot shock, paired rats typically assume an upright posture, bare their teeth, and strike vigorously at each other with their forepaws), pain-elicited aggression has become a topic of considerable scientific interest. Attempting to elucidate a phenomenon previously described by O'Kelly and Steckle (1939), Ulrich and Azrin (1962) determined that shock-induced fighting in rats was a function of both enclosed floor area and shock intensity. Manipulating sex of the subjects, strain, previous familiarity with other subjects, and number of subjects present during shock did not

alter this pattern of stereotyped fighting. Optimal conditions for inducing fighting were defined as two rats confined in an experimental chamber exposed to a 2 mA foot shock.

Following Ulrigh and Azrin's (1962) initial statement concerning the variables affecting pain-aggression in rats, other researchers have sought to further describe the factors that are operative in modulating this behavior. Within this context several investigators have attempted to demonstrate shock-induced aggression in various mammalian species. For example, Ulrich and Azrin (1962) showed that paired hamsters displayed fighting reactions similar to those of the rat in response to shock whereas guinea pigs did not. Additionally, Ulrich and Azrin (1963), Ulrich, Wolff, and Azrin (1964), and Dunstone, Cannon, Chickson, and Burns (1972) reported that shock successfully elicited intraspecific aggressive responding between paired squirrel monkeys, cats, and gerbils, respectively. Curiously, the gerbils appeared to exhibit attacks that were more persistent and vigorous than those produced by other rodent species.

A similar line of research has involved manipulating modality of the pain-evoking stimulus. Ulrich and Azrin (1962) also reported that fighting between paired rats was elicited by electrode shock to the back of one animal as well as with intense heat. However, intense noise and moderate cold failed to produce the stereotyped fighting response. It appeared, however, that competing responses which developed during the presentation of intense heat rendered this pain modality somewhat undesirable for pain-aggression studies. Azrin, Hake, and Hutchinson (1965) found that squirrel

monkeys exposed to brief tail pinches also exhibited aggression as a direct function of the force of the tail pinch.

Azrin, Rubin and Hutchinson (1968) identified a major problem found in most shock-elicited aggression studies, namely the reliance upon subjective evaluation of movements and postures of the subject pairs that were considered to be aggressive. The ideal situation would be that of observing aggression in a single subject, thus eliminating the need for evaluation of specific movements and postures. Unfortunately, Ulrich and Azrin (1962) found that a lone rat typically does not aggress toward an inanimate object in response to foot shock. Desiring to develop a technique whereby automatic recordings could be obtained for aggressive responses, Azrin et al. (1968) described a method by which individual rats could be made to bite an inanimate target object. In this procedure restrained rats received unavoidable tail shocks of 5 mA intensity with a 200-msec duration every 10 sec. for 20 min. Results of this investigation clearly revealed that biting attacks toward inanimate targets could be elicited by applying tail shock to restrained rat subjects. Consequently, the pain-aggression reaction in rats was more readily accessible to objective measurement and study. Azrin, Hutchinson, and Sallery (1964) had previously demonstrated that aggression toward inanimate objects could be elicited in squirrel monkeys by applying foot shock. Therefore, these investigators had suggested that domesticated rats were perhaps inherently less aggressive than squirrel monkeys. Obviously, this distinction was not valid.

Although Ulrich and Azrin (1962) reported that shock-induced

fighting in rats was independent of sex, some investigators have found that sex and age of the subject are related to the frequency of reflexive aggression. (Despite the fact that some argument has been made for including shock-elicited aggression within the instrumental learning paradigm (Powell & Creer, 1969; Dreyer, Russell, & Church, 1970), most investigators seemed to lean more toward a reflex interpretation of these behaviors). Milligan, Powell, and Borasio (1973) found that sex significantly affected rate of fighting in Long Evans rats (i.e., males fought significantly more than females). Hutzell and Knutson (1972) reported that shock-elicited fighting and shock-elicited biting were differentially affected by sex of hooded rats obtained from the University of Iowa colony. Specifically, males displayed significantly more intraspecific fighting than females, but frequency of shock-elicited biting (number of attacks toward an inanimate target) was found to be independent of the sex of the subject. On the other hand, Powell, Silverman, Francis, and Schneiderman (1970) suggested that intraspecific fighting between paired Sprague-Dawley rats exposed to foot shock was unaffected by sex of the subject. Clearly, inconsistencies regarding the effects of sex on shock-elicited aggression are apparent in the literature.

Age of the subject has also been a topic of investigation. Powell and Creer (1969) stated that maturation interacted with prior shock and fighting experience in determining amount of shock-induced aggression in Sprague-Dawley rats. Hutchinson, Ulrich, and Azrin (1965) found that reflexive fighting in rats increased as a direct function of the subject's age and that castration produced lowered fighting probability in adult subjects, regardless of whether the

subject was castrated before or after puberty.

Control of previously administered shock and prior experience with fighting also appeared to be salient features in determining amount of subsequent shock-elicited aggression. Maier, Anderson, and Lierberman (1972) reported that rats exposed to a series of inescapable tail shocks displayed less aggressiveness on subsequent shock-elicited aggression tests than rats given previous exposure to the same number of escapable shocks. Similarly, Powell et al. (1970) determined that experiences with shock and fighting resulted in increased fighting frequencies when rats were placed in a shock-aggression situation. Furthermore, they showed that rat subjects receiving trials spaced over several sessions fought more frequently than subjects receiving the same number of trials during a single session.

Attempting to amplify the relationship described by Ulrich and Azrin (1962) between shock intensity and shock-induced aggression, several investigators have considered the specific effects of manipulating shock duration, shock intensity, and shock source on reflexive fighting. Azrin, Ulrich, Hutchinson, and Norman (1964) found that shock-induced fighting between pairs of rats varied directly with both shock intensity and duration. However, it was noted that continued shock presentations tended to partially reverse this function. Similarly, Creer and Powell (1971) reported that extended training tended to wash out the effects of shock intensity. Dreyer and Church (1968) attempted to quantitatively specify the functional relationship between shock intensity and duration on probability of shock-elicited fighting. Essentially, these inves-

tigators found that fighting probability was a linear function of the logarithm of both shock intensity and duration. Anecdotal evidence revealed that subjects tended to be more sensitive to increases in shock intensity than duration, as subjects were more prone to vocalize with increasing intensity than with increase in duration. Statistical data also revealed that the slope of the intensity function was approximately twice that of the slope of the duration function. Additionally, Follick and Knutson (1974) found that at low stimulus intensities, dc shock resulted in greater fighting frequencies in paired rat subjects than either ac or ac rectified shocks. Consequently, shock source appears to be at least a peripheral factor in shock-induced aggression studies, particularly at lower stimulus intensity values.

Several studies have sought to determine the effect of housing conditions on the shock-elicited aggression paradigm. Creer (1974) concluded that housing rats 6 to a cage for 30 days prior to testing apparently influenced reflexive fighting in response to foot shock. Creer argued that communal housing served to produce greater inconsistency in fighting over sessions. However, due to serious methodological errors, specifically lack of appropriate control groups, these conclusions can only be regarded as tentative. Creer (1975) extended his earlier study to specifically investigate the effects of housing rats in single or communal cages for varying periods of time on shock-induced fighting. Again, great variability in fighting frequencies was reported. Housing rats in communal cages for 21 or 28 days prior to aggression testing produced a particularly deleterious effect on frequency of aggressive contacts. On the other

hand, Hutchinson et al (1965) found that rats housed in groups demonstrated higher fighting frequencies than isolates. However, stabilization of the aggression parameter occurred more rapidly for isolates than for communally housed subjects. Obviously, the effects of housing still remain somewhat unclear.

The effects of specific deprivation states and related drive states on shock-induced aggression have received somewhat scanty attention in the literature. Cahoon, Crosby, Dunn, Hill, and McGinnis (1971) determined that the effect of food deprivation on reflexive fighting was simply to increase, to a point, exhibited levels of aggression. Similarly, Hamby and Cahoon (1971) and Devine (1971) reported that frequency of shock-induced fighting was a direct function of level of water deprivation, the function seemed to be best described as a curvilinear one. In a related study, Bisbee and Cahoon (1973) found that nausea induced by injection of lithium chloride produced high levels of fighting in rat subjects at small dosage levels, while larger doses served to inhibit display of shock-elicited aggression.

Within the context of deprivation and resultant drive enhancement, it would appear that some functional relationship might be obtained between deprivation of rapid eye movement sleep (REM) and reflexive fighting in rats. A phenomenon first described by Aserinsky and Kleitman (1953), REM sleep has frequently been described as the state of sleep during which dreaming occurs (Dement & Kleitman, 1957). As reported by Aserinsky and Kleitman (1953), rapid eye movements were typically accompanied by changes in respiration and heart rate, and changes in EEG pattern from the usual large,

slow, regular wave pattern of deep sleep to a low-voltage, desynchronized one similar to that of an alert waking state. Although the discovery of REM sleep went virtually unnoticed for the next several years, research dealings with this phenomenon proliferated during the 1960's and has persisted into the 1970's.

Of special interest to many investigators have been the behavioral and biochemical effects of deprivation of REM sleep. Human research dealing with the specific effects of REM deprivation has produced the following general results: (a) a reliable tendency existed to "makeup" or compensate for lost REM time, and (b) reports of psychological distress were frequent concomitants of the deprivation procedure (Hokanson, 1969). Sampson (1966) also found that some human subjects developed intense hunger and cravings for certain food substances. More importantly, this investigator observed a statistically significant increase in aggressive content in REM dream fragments with increasing levels of REM deprivation. Despite the fact that some controversy surrounds research dealing with specific clinical effects of REM deprivation, the aforementioned research would seem to place behavioral and psychological effects resulting from REM deprivation within the realm of motivationally determined behaviors.

Although relatively little is known regarding the behavioral effects of REM deprivation in animals (Stern, 1969), research has demonstrated that certain behaviors are sensitive to REM deprivation manipulations. For example, Pearlman (1971) reported that REM sleep deprivation impaired latent learning in rats. In a corollary publication, Pearlman (1973) also determined that REM sleep deprivation

had a deleterious effect upon latent extinction in rat subjects. In a related series of studies, Stern (1971) found that acquisition of three tasks - passive avoidance, active avoidance, and an appetitive alternation discrimination - was markedly impaired by 5 days of REM deprivation.

Other investigators, however, have found that certain behaviors were apparently unaffected or enhanced as a function of REM deprivation. Specifically, Holdstock and Verschoor (1973) reported that retention of food motivated position habits in a T-maze was unaffected by REM deprivation. The possibility of an interaction between REM deprivation and type of learning task was suggested as an explanation for discrepancies in the literature relating REM deprivation and learning. Hicks and Paulus (1973) ascertained that 0, 24, 48, or 72 hours of REM deprivation produced a significant inverse effect on latency of T-maze performance. A significant effect of REM deprivation was not observed, however, for accuracy of performance in this situation. These results were interpreted as supporting the contention that REM deprivation tends to increase generalized drive, as was previously suggested by Dement, Henry, Cohen, and Ferguson (1967). Similarly, in a series of 4 experiments, Albert, Cicala, and Siegel (1970) found that both shuttle avoidance and runway avoidance were unaffected by 3, 6, or 9 days of REM deprivation. Furthermore, increasing REM deprivation resulted in an enhancement of activity. These investigators also adopted a motivational-effects model to explain these results. They specifically posited that REM deprivation increased sensitivity of the REM deprived subjects to environmental

stimulation.

Regarding the biochemical effects associated with REM sleep deprivation, the literature is far from definitive. Hartmann (1973) has suggested that desynchronized sleep serves a homeostatic function in maintaining brain catecholamines (neurotransmitter substances) or catecholamine-containing systems. The specific mechanism by which REM sleep accomplishes this, however, was unclear. According to Stern and Morgane (1974), a logical consequence of REM deprivation (assuming one adopted the catecholamine-maintenance hypothesis) would be induced impairment of catecholamine functioning and a concomitant reduction in brain catecholamine levels. These authors pointed out that research has not typically borne out this assumption (Bliss, 1967; Pujol, Mouret, Jouvet, & Glowinski, 1968). A study conducted by Hartmann and Stern (1972), however, revealed that learning deficits produced by 4 days of REM deprivation could be reversed by administration of L-dopa, the chemical precursor of dopamine, which is a catecholaminergic substance. In light of such discrepancies, Stern and Morgane (1974) and Stern and Hartmann (1972) suggested the possibility of stress confounding, particularly when the so-called water tank island technique is employed. Thierry, Fekete, and Glowinski (1968) had previously demonstrated that metabolism of brain catecholamines is profoundly increased by stress. Consequently, it was suggested that stress may, in effect, overide any tendency toward lowered catecholamine synthesis during REM deprivation. Despite the fact that no clear cut relationship between REM sleep deprivation and catecholamine functioning has been established, consideration of this topic

was indicated in view of the fact that Welch and Welch (1971) proposed an involvement of these neurotransmitters in modulating intraspecific fighting.

In light of the aforementioned research, a logical direction for REM studies to follow seemed to be in the area of shock-elicited aggression. Stern (1969) reported that after 5 days of REM deprivation using the water tank island method, the rat subjects subjected to REM deprivation manifested significantly lower aggression thresholds than either stress controls (those subjects partially immersed in cold water for 20 minutes per day over the course of aggression testing) or typical control subjects (those subjects maintained on large islands in the water tank apparatus). In a previous two-experiment series, Morden, Conner, Dement, and Levine (1968) deprived male Long Evans rats of REM sleep for 7 days. Shock intensity was also systematically varied across 3 equal groups of the subjects. In Experiment I, all of the subjects were tested for aggression on alternate days for 7 days. Experiment II essentially replicated Experiment I with the exception that the subjects were tested after Day 7 only. Results of these manipulations indicated that the subjects deprived of REM sleep exhibited higher fighting frequencies than controls, particularly at low shock intensities. However, the fact that level of REM deprivation was a within subjects variable introduced the possibility of confounding across successive days of aggression testing.

The purpose of the present study was to systematically investigate the effects of level of REM deprivation on shock-elicited aggression in rats. Unlike the Stern (1969) and Morden et al. (1968)

studies, the present study sought to determine the effects of 4 specific levels of REM deprivation on two measures of shock-induced aggression in retrained rats: total number of aggressive responses and time spent making aggressive contacts. Additionally, REM deprivation was made a between subjects factor so that each rat was tested only once. This procedure eliminated the possibility of confounding across successive days of aggression testing. Also unlike Morden et al. (1968), measures of shock-elicited aggression were obtained for each of the specified levels of REM deprivation as opposed to testing on alternate days of REM deprivation. In view of previous research concerned with the effects of REM deprivation on drive (i.e., REM deprivation is presumed to enhance generalized drive (Hicks & Paulus, 1973; Dement et al., 1967), it was specifically hypothesized that a linear function would be obtained between level of REM deprivation and shock-elicited aggression in rat subjects. In addition it was hoped that some inferences could be made concerning the neurochemical and physiological processes responsible for mediating shock-elicited aggression as it is affected by REM sleep deprivation.

CHAPTER II

METHOD

Subjects

The subjects were 36 male, albino rats purchased from the Holtzman Co., Madison, Wisconsin. The subjects were approximately 150 days old at the experiment's outset. Although experimentally naive with respect to shock-elicited aggression and REM sleep deprivation procedures, all rats had previously served as subjects in a contrafreeloading experiment. A three month period separated the conclusion of the contrafreeloading experiment and the inception of the present study. During this period all animals were housed in individual cages with water and food available on an ad lib basis.

Apparatus

REM Deprivation Apparatus. The REM deprivation apparatus used in the present investigation was similar to that described by Hicks and Paulus (1973). More specifically, during REM deprivation periods all subjects were maintained in 5-gallon metal pails, the tops of which were covered with one-half in. hardware cloth. A water bottle and a metal food container were affixed to the hardware cloth such that each subject had free access to both food and water while confined to the pail. Inverted flower pots with bases measuring 7.4 cm and 11.2 cm in diameter served as islands for the subjects in the REM and control groups, respectively. Each pail was filled with water to within approximately 1.14 cm of the top of the inverted flower pot. Use of this technique precludes REM sleep since animals

typically lose muscle tonus at the onset of REM. Consequently, the subject either awakens or falls into the water. Amount of non-REM sleep, however, is not significantly altered since muscle tonus is maintained during non-REM periods (Jouvet, 1963). Plastic maintaining tanks with appropriately sized platforms were utilized to house the animals while water from the deprivation tanks was being changed. To reduce odors and the possibility of disease, the water in each tank was changed daily.

Shock-elicited aggression apparatus. A rat restraining device similar to that described by Azrin et al. (1968) served as the apparatus in shock-elicited aggression testing. This apparatus consisted of an opaque plastic tube, measuring 21.5 centimeters in length and 7.5 centimeters in diameter, mounted on a plexiglas sheet. The plexiglas sheet was, in turn, stabilized on a wooden platform. However, the plexiglas sheet was easily removed from the wooden platform to facilitate placement of the subject into the tube and to permit easy removal of fecal material and urine that accumulated in the tube during testing. A 1.5 cm hole at the enclosed end of the tube allowed the subject's tail to be extended from the apparatus and secured to a wooden restraining rod by means of adhesive tape. The other end of the tube was open. Two pieces of No. 14 copper wire were permanently attached to the rod 7 cm apart and served as tail electrodes. Thus, when the rod was secured in place it served as both a restraining device to prohibit unauthorized escape from the apparatus and as an electrode carrier. A 1.5 mA ac rectified current was supplied by a modified Lafayette (Model 85204) shock generator. A Jackson (Model 665-J-2) mA meter was used

to monitor shock intensity across each subject. The aggression target consisted of an omnidirectional lever (Model 80111) purchased from the Lafyette Instrument Co., Lafayette, Indiana. This lever was mounted on the wooden platform, perpendicular to the open end of the restraining tube and parallel to the wooden platform on which the tube was mounted. When the tube was in place on the platform, the lever extended across the open mid-portion of the end of the tube. The lever was 1.5 cm from the tube and required a movement of 1 cm to activate the attached microswitch. Closure of the microswitch, in turn, activated: (1) a Standard Electric Timer, and (2) a Lafayette (Model 5707PS) impulse counter.

Procedure

At the beginning of the experiment, the subjects were randomly assigned to one of four equal groups: Group C (control, no REM deprivation), Group 24-R (24 hr. REM deprivation), Group 48-R (48 hr. REM deprivation), and Group 72-R (72 hr. REM deprivation). Subjects in Group C were, in turn, randomly assigned to one of three equal subgroups (24 hr., 48 hr., and 72 hr.) to provide appropriate time-in-tank controls.

On day 1, subjects were placed on the inverted flower pots at 30 min. intervals in order to insure an individual testing period for each subject following the confinement-in-the-tank period. Subjects in Group C were placed on the large (11.2 cm) pots while subjects in Groups 24-R, 48-R, and 72-R were placed on the small (7.4 cm) pots. The order for placing subjects into the deprivation tanks, and hence the order for running subjects in the subsequent shock-elicited aggression task, was random.

Upon completion of the specified confinement period for each subject, the subject was removed form the deprivation tank and secured in the restraining tube. Prior to taping the restraining rod to the animal's tail, electrode paste was applied to the electrodes. The subject was positioned in the tube such that its nose was approximately 1 cm from the target rod. Each subject experienced a 5 min. habituation period in the restraining tube prior to the administration of shock. A 15 min. period of shock administration immediately followed habituation. During this time, each subject was exposed to a series of 300 msec. duration 1.5 mA shocks administered at 3 sec. intervals. Thus, each subject experienced a total of 300 shocks. Both the total number of aggressive responses and the total time of aggression were recorded for each subject.

CHAPTER III

RESULTS

Both the time and response data were transformed into $\log_{10}(X_i+1)$ scores prior to analysis. These transformed scores were subjected to analysis of variance. Subsequent comparisons of significant effects were performed by the Newman-Keuls procedure. Group means for the time data are presented in Figure 1, while group means for the response data may be found in Figure 2.

Prior to overall analysis of either the time or response data, F max tests were performed on the data from Group C in order to insure the propriety of pooling the data of the subjects in this group. The results of these analyses yielded nonsignificance for both the time, $\underline{F}$ (2,2) = 5.74, $\underline{p} > .10$, and response, $\underline{F}$ (2,2) = 8.65, $\underline{p} > .10$, measures.

Analysis of the time data yielded a significant Groups effect, $\underline{F}$ (3,32) = 3.71, $\underline{p} < .05$. Subsequent comparisons indicated that Groups 48-R and 72-R were significantly ($\underline{p} < .05$) more aggressive than Groups C and 24-R. The analysis of variance for the time data is summarized in Table 1.

Analysis of the response data also yielded significance for the Groups factor, F (3,32) = 3.61, $\underline{p} < .05$. Subsequent comparisons indicated that Groups 48-R and 72-R were significantly ($\underline{p} < .05$) more aggressive than Group 24-R. The analysis of variance for the response data is summarized in Table 2.

In view of past research on shock-elicited aggression, the shape of the obtained function appeared to be of some interest. Hence, tests for trend were performed on both the time and response

data. The analysis of the response data yielded a significant linear trend, $\underline{F}$ (1,32) = 10.42, $\underline{p} < .01$, and a nonsignificant departure from linearity, $\underline{F}$ (2,32) = .15, $\underline{p} > .25$. Similarly, a significant, $\underline{F}$ (1,32) = 10.73, $\underline{p} < .01$, linear trend, and a nonsignificant, $\underline{F}$ (2,32) = .20, $\underline{p} > .25$, departure from linearity were found in the time measure analyses.

CHAPTER IV

DISCUSSION

The present study suggests that the amount of shock-induced aggression shown by rats is an increasing linear function of REM deprivation up to 72 hours. This relationship was found for both time spent in aggressing and number of aggressive responses made. These results are consistent with those of Stern (1969) and Morden et al. (1968), who found that REM deprived rats (5 and 7 days respectively) were more aggressive than controls.

Although not significant, a considerable amount of within-group variability was found in the present study. This observation is consistent with that of other investigators working in the general area of shock-elicited aggression (Azrin, Hutchinson, & Hake, 1963; Azrin, Hutchinson, & Hake, 1966; Cahoon et al., 1971).

However, the results of the present study do not appear to be compatible with those of previous studies investigating the effects of other deprivation states on shock-induced aggression. For example, Cahoon et al. (1971) and Hamby and Cahoon (1971) reported a curvilinear function between shock-induced fighting and level of food and water deprivation, respectively. Perhaps this discrepancy could be attributed to the fact that the uppermost level of REM deprivation employed in this study was not severe enough to produce the decrements observed under other deprivation states.

Simply adopting the model that REM deprivation serves to enhance generalized drive (Dement et al., 1967) seems somewhat inadequate

for fully explaining these results. Rather, it appears that consideration should be given to the biochemical and physiological processes underlying such an increase in motivation. In an attempt to more thoroughly explain the results of the present investigation, a catecholamine maintenance hypothesis similar to that described by Hartmann (1973) and Stern and Morgane (1974) is suggested. Hartmann (1973) proposed that REM sleep serves a homeostatic function in maintaining brain catecholamines or catecholamine-containing systems. More specifically, Stern and Morgane (1974) argued that a consequence of REM deprivation would be induced impairment of catecholamine functioning and an associated reduction in brain catecholamine levels. Regarding a possible relationship between brain catecholamines and aggression, Welch and Welch (1971) proposed an involvement of these neurotransmitters in determining level of activation and reactivity of the central nervous system. More importantly, these investigators suggested that increasing the level of available catecholamines facilitates many behaviors, including fighting. Conversely, when supply of these substances is diminished due to inhibition of their biosynthesis, animals typically become sedate, and many behavioral processes are deleteriously affected (Wise & Stein, 1969). Directly applying the catecholamine maintenance model to the present situation was somewhat problematic. Since shock-induced aggression was found to increase directly with increasing levels of REM deprivation, this model would, at least supperficially, appear to be inappropriate. Stern (1969), however, suggested that REM deprivation alters the physiological mechanism of shock-elicited aggression and pointed

out that the nature of these changes was undetermined. Perhaps the mechanism by which REM sleep deprivation modifies shock-induced aggression is stress. As pointed out by Stern and Morgane (1974), stress may operate as an inherent confounder in REM deprivation studies, particularly those employing the water tank island technique, which was used in the present study. It would seem feasible then to postulate that the effects of stress are simply to compensate for any detrimental effects of lowered catecholamine levels at low to moderate levels of REM deprivation. However, at extreme levels of REM deprivation, the compensatory effects of stress would perhaps be mitigated by severely lowered levels of available brain catecholamines and decrements with respect to shock-elicited aggression would be observed. This speculation, however, remains to be systematically investigated.

Clearly, additional research is needed to further amplify the factors operative in modulating shock-induced aggression in response to REM deprivation. Extending level of REM deprivation beyond three days and making systematic comparisons among the effects of each level of REM deprivation on shock-induced aggression would appear to be most appropriate. Further study regarding the biochemical processes involved in mediating shock-induced fighting is also indicated. Although the area of shock-elicited aggression and REM sleep deprivation is fraught with problems, it appears to be one that is fertile for future research efforts.

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APPENDIX A: FIGURES

Fig. 1 - Mean Number of Aggressive Responses.

MEAN LOG10 (Xi + 1) RESPONSES
2.25
2.00
1.75
1.50
C
24-R
48-R
72-R

Fig. 2 - Mean Time of Aggressive Behavior

MEAN LOG10 (Xi+1) TIME
2.25
2.00
1.75
1.50
C
24-R
48-R
72-R

APPENDIX B: TABLES

TABLE 1

SUMMARY OF TIME DATA ANALYSIS OF VARIANCE

Source	SS	df	MS	F
Between Groups	3.49	3	1.16	3.71*
Within Groups	10.04	32	.31	
Total	13.53	35		

*$p < .05$

TABLE 2

SUMMARY OF RESPONSE DATA ANALYSIS OF VARIANCE

Source	SS	df	MS	F
Between Groups	4.08	3	1.36	3.61*
Within Groups	12.07	32	.38	
Total	16.15	35		

*$p < .05$