THE EFFECT OF EXERCISE ON THE SEXUAL BEHAVIOR OF THE MALE RAT

by

Michael DeStefano

A Thesis Submitted to the Faculty of the

DEPARTMENT OF PSYCHOLOGY

In Partial Fulfillment of the Requirements
For the Degree of

MASTER OF ARTS

In the Graduate College

THE UNIVERSITY OF ARIZONA

1 9 6 9

STATEMENT BY AUTHOR

This thesis has been submitted in partial fulfillment of requirements for an advanced degree at The University of Arizona and is deposited in the University Library to be made available to borrowers under rules of the Library.

Brief quotations from this thesis are allowable without special permission, provided that accurate acknowledgment of source is made. Requests for permission for extended quotation from or reproduction of this manuscript in whole or in part may be granted by the head of the major department or the Dean of the Graduate College when in his judgment the proposed use of the material is in the interests of scholarship. In all other instances, however, permission must be obtained from the author.

SIGNED: Michael De Stefano

APPROVAL BY THESIS DIRECTOR

This thesis has been approved on the date shown below:

Sigmund Hsiao
SIGMUND HSIAO
Assistant Professor of Psychology

September 10, 1968
Date

ACKNOWLEDGMENTS

I would like to express my deepest thanks and appreciation to the following individuals:

Dr. Sigmund Hsiao, who as Chairman of my Committee has offered many timely suggestions in the course of our numerous theoretical discussions as well as stimulated and broadened my area of approach on the central theme of this study. His helpful instructions and demonstrations have been invaluable in the performance of the surgical procedure and injections of the female animals. I am deeply indebted to him for his outstanding role both during the research stage and the final preparation and completion of this thesis.

Dr. Jack Capehart, who in serving on my Committee confirmed the value of this study as well as provided the statistical advice.

Dr. Cecil Rogers, who originally inspired me with the encouragement to undertake this study and who also served on my Committee.

Dr. Howard A. Baldwin, who provided me with the use of his Sensory Systems Laboratory and instruments during the surgical procedures.

Janet Baker, who generously served as my assistant during the surgical procedure, the laboratory work, and the

initial observation sessions. Without her help and understanding this research would have been much more difficult than it was.

Mitchell Lewis, who served as personal confidant in numerous discussions that resulted in greater clarification and understanding of the various theoretical ramifications of this study. His generous and conscientious help during the observational part of this research was invaluable.

Janice Buchanan, whose conscientious assistance during the latter stages of the observational procedure greatly enhanced what would ordinarily have been a tedious, monotonous process by her vivacious personality, lively sense of humor, and by her gifted capacity for appreciating the unusual.

Dr. Abraham Segal, whose timely comments and advice further aided the progress of this thesis.

Without their help and consideration this thesis would not have been possible.

"I thought that working on sex was the easiest way to help mankind. If I could discover a way to improve the sexual life by even one per cent, then I could improve the whole species [Maslow, 1968, p. 40]."

TABLE OF CONTENTS

LIST OF TABLES

LIST OF ILLUSTRATIONS

ABSTRACT

The intention of this thesis was to determine if chronic exercise as a form of stress administered to male rats beginning at 40 days old for over a period of 100 days would have some augmentative effect on sexual performance. This hypothesis was based on the findings that (1) chronic exercise has resulted in significant morphological alterations in endrocrine structures necessary for normal sexual functioning, and (2) such stress during the pre-puberal stages of development might also positively enhance the responsiveness of neural and/or somatic tissue mediating sexual behavior.

Of several response measures chosen the results were non-significant except for one. There is some evidence that any augmentative effect of the exercise was counteracted by the inferior performance of that increased proportion of Ss which ordinarily would have been expected to be non-maters.

There still remains some question of the results had the Ss been subjected to this same procedure at an older age when sexual performance is naturally on the decline.

The most remarkable outcome of this study was the naturalistic observation after the experiment that the

exercised group was significantly more combative and aggressive, possibly reflecting some relationship between exercise, sex, and aggression.

INTRODUCTION

The general thesis of this paper is based on the following assumptions: (1) Sexual behavior is in part dependent on adequate endocrine functioning; (2) A significant alteration of optimal hormonal secretion results in some effect on sexual functioning; (3) There is a definite relationship between non-specific stress on the organism and endocrine secretion.

The aim of this thesis is to show that some relationship exists between stress administered in the form of exercise and its corresponding effect on sexual behavior of the male rat.

In studies of the sexual behavior of the human male, the variables influencing sexual functioning are many indeed. As of the present there has been no significant study correlating the extent of physical exercise as a form of stress with the sexual behavior of the human male. One obvious explanation for this lack of any significant finding is a result of the complex interaction and contamination of other more complicated factors such as genetic variation among individuals, extreme ranges of environmental stimulation, and various other cultural and learned determinants.

In the investigations of Kinsey, Pomeroy, and Martin (1948) it is clearly shown, however, that

semi-skilled laborers do have a greater frequency of sexual outlet compared to white collar workers. Granted that many variables are operating here to result in this difference, it is suspected that the general level of energy expenditure which also seems to differ with these two groups may be one of the many contributory variables in producing such a difference. It is well established that the effect of chronic physical exercise as a form of stress on the mammalian organism definitely results in many marked physical increments. According to Steinhaus (1963) a summary of the chronic effects of exercise, that is, exercise engaged in over a long period of time--long enough to constitute the state generally known to be characteristic of that of the trained athlete--includes the following characteristics:

1. An increase in muscle size, strength, and endurance.
2. An increase in size and weight of the heart.
3. An increase in cardiac output in volume per minute.
4. A low basal or resting pulse rate.
5. A reduction in systolic blood pressure.
6. An increase in the concentration of erythrocytes--red blood cells--and, consequently, a corresponding increase in hemoglobin content which results in an increased capacity for oxygen consumption.
7. A slight increase in lymphocytes.
8. An increase in thrombocytes--blood platelets.

9. An increased alkali reserve thus allowing the organism to tolerate higher concentrations of carbon dioxide and lactic acid, the by-products of muscular contraction.
10. An increase in vital lung capacity and a resultant economical exchange of gasses during strenuous activity.
11. A lower basal metabolism, which results in less energy expenditure in the performance of strenuous activity.
12. A decreased rate of cardiac acceleration in the face of strenuous activity.

In short, there is less of a deviation from the basal level of the organism under the influence of strenuous activity. Interestingly enough, the basal level of vegetative functioning is lowered somewhat as a result of chronic exercise.

Most characteristic of the trained state is a shortened recovery interval which allows the trained organism to return sooner to the basal level of functioning once deviation has occurred.

Physiological effects as dramatic as have just been listed cannot occur in isolation of the endocrine system, as a whole, which serves as a focal point for metabolic activity. One strong indication of alterations in

endocrine functioning comes from various studies demonstrating evidence of morphological or structural alterations in the size and weight of various endocrine glands and other body organs.

Donaldson (1931) has shown that rats that have lived their entire life in a revolving drum cage in which exercise was taken voluntarily show significant increases when compared with controls in weight of heart, kidneys, submammillary glands, gonads, and adrenal glands when sacrificed at 220 days old. Interestingly enough, he also reports an increase of brain weight of three per cent over the control animals.

Hatai (1915) working with the albino and wild Norway rats 30 days of age and placing them in a revolving cage--involuntary exercise--for a period of three to six months, found significant increases in the weight of the heart, kidneys, liver, ovaries, testes, and hypophysis. There was a marked increase in weight of the adrenal glands in the female but no significant deviation for the males for this gland. When compared with the control group, there is a decrease in weight of the thyroid gland and spleen, depending on the age of the animal when sacrificed. In confirmation of Donaldson's findings, Hatai has found a four per cent increase in brain weight for these experimental animals.

In each case here the Ss underwent chronic exercise during their development and, surprisingly enough, there resulted a slight alteration in the nervous system--an increase in brain weight. In view of this finding, one might hypothesize that exercise could have a more subtle effect on other neural tissue throughout the body. Perhaps the threshold of neural tissue in response to hormones could be altered through some interaction of the effects of chronic exercise during a critical developmental period. The reader should keep in mind at this point that a good deal of the sexual functioning in the mammal is a result of some interaction between hormones and neural tissue.

The method of exercise also seems to have some effect on organ differences. Steinhaus, Hoyt, and Rice (1932) have shown that for growing dogs the adrenal gland, for example, is increased in size by swimming exercise and decreased slightly by running exercise. Such a finding, however, may reflect more the differences of the two modes of exercise at different stages of the reaction to such a stress.

According to Selye (cited by Constantinides and Carey, 1949), stress is defined as "a non-specific deviation from the normal resting state; it is caused by function or damage and it stimulates repair." Such extremes as cold, fasting, emotional excitement, injuries, electric shock, muscular exertion, and the invasion of foreign

organisms into the body--any one of these--will result in the same non-specific deviation from the normal resting state of the organism. This general reaction of the organism in the face of stress is what Selye calls the "General Adaptation Syndrone." It is divided into three stages: (1) the Alarm Reaction, during which full adaptation has not been acquired; (3) the Stage of Resistance, during which adaptation to the stressor is optimal; and (3) the Stage of Exhaustion, during which acquired adaptation is lost. During the first stage, the Alarm Reaction, the characteristic findings in response to stress are: (1) an enlargement of the adrenal glands and a change in color from yellow to brown if the stress is severe, (2) an involution of the thymus gland, and (3) ulcerations of the stomach. The most dramatic change during the Alarm Reaction occurs with the enlargement of the adrenal glands. Anderson (1935) has shown that short term exercise of seventy-five 90-day-old male rats subjected to a revolving cage for eight 1/2-hour exercise intervals with a half hour rest between intervals, and sacrificed sixteen hours later, showed a marked hypertrophy of the adrenal glands and an atrophy of the thymus.

Specifically, the adrenal cortex produces hormones necessary for life and stores them in fat droplets or lipids which give the cortex its yellowish color. All these fat soluble hormones are called steroids--20 in

all--which have the same basic chemical structures as the sex hormones. One of these steroid hormones in both the male and female is androgen--generally called the male hormone, the principal source of which is the testes primarily and the adrenal cortex secondarily. During the first hours of the Alarm Reaction these hormones in their lipid vehicles are rapidly discharged from the adrenal cortex into the blood stream and race to the tissues of the body where they exercise homeostatic effects on the tissues in reaction to stress (Constantinides and Carey, 1949).

Of course, the adrenal gland itself does not act autonomously. Like most endocrine glands, it is under the direct control of the anterior lobe of the pituitary gland in reactions to stress releases andrenocorticotrophic hormone (ACTH), which stimulates mainly the adrenal cortex.

From a long series of experiments, Selye (1956) outlines the AR mechanism. Stress results, through some unknown pathway, in stimulating the anterior pituitary to release ACTH, which mobilizes the adrenals to discharge their lipids. As a result of these steroids released from the cortex of the adrenals, they tend to atrophy somewhat. The steroids have their main effect in helping the organism adapt to the stressor. Should the stress continue at an intensity below the lethal level, the adrenals refill their empty stores with lipids and revert to normal size and the thymus tends to regain some of its mass. In general,

functioning tends to return to normal in spite of the stress; this is known as the Stage of Resistance. It is interesting to note that the adrenals may be fluctuating in size depending on the demands required from the stressor by changes in intensity or duration. An autopsy performed on a S at this stage might very well show adrenal glands a bit smaller than normal, as in the finding of Steinhaus et al. (1932) with the dogs subjected to running exercise, but not to swimming. If another stress is introduced at this stage, or the original stress becomes too great, the organism enters the final stage--the Stage of Exhaustion--during which the organization of the organism, its life, tends to decompensate in face of this added stress. In this stage the adrenals are larger than in stage II.

The work of Kimeldorf and Baum (1954) in which male rats were swum for a period of fifteen to thirty minutes with weights clipped to their chests five times per week for varying periods up to thirty days shows that of all the changes the most dramatic is the increase in adrenal size. Though there was a decrease in body weight for the exercised group, the absolute size of the testes was not altered by exhaustive exercise. The remaining structural and functional adaptations found by Kimeldorf and Baum agree essentially with the findings of Hatai (1915) and Donaldson (1931).

Thus far it has been established that stress in the form of physical exercise does have some effect at least on the morphology of the adrenal cortex (e.g., Selye, 1956). One of the steroids produced by the adrenal cortex is androgen, the male sex hormone, which, in interaction with neural tissue or other hormones has some effect on the organization and activation of those neural tissues mediating sexual behavior in the male rat. The exact relationship of androgen to this function will be discussed in more detail shortly.

Before concluding this section on the relationship of exercise and endocrine activity, I would like to mention a preliminary report of some work by Herbert Vogel (cited by Steinhaus, 1963) of the Deutsche Hochschule fur Leibesubungen in Berlin. Vogel approaches the problem from the opposite direction of this thesis in presenting the effects of nocturnal emissions on the athletic proficiency of the male. Using students in track and field as Ss, he kept precise records on the seasonal performance of three events based on a nine point scale. For each of the three events, all Ss showed periodic and cyclic fluctuations in performance in which high points rhythmically occurred at regular three week intervals and low points in performance at equal three week intervals. The most remarkable observation in relation to this cyclic fluctuation in performance is that "nocturnal emissions regularly come

when the performance curve is on its way down and about three or four days before it hits bottom [Steinhaus, 1963, p. 145]." Social influences were controlled to a certain extent in that the Ss were not even allowed the sexual stimulation afforded by a movie. An interesting physiological finding of this observation is when one of the athletes just past his "high point"--peak performance phase--was given a bottle of beer which was otherwise forbidden in training, a nocturnal emission occurred that night at least four days before it was expected, and the S's athletic record would hit bottom the next day. The subsequent rise, then, in athletic performance came earlier than scheduled; this procedure was repeated twice for two athletes; the other one required a hypnotic drug as a sufficient stimulant for inducing nocturnal emissions. The point of this study according to Vogel is his claim that contestants can be prepared for specific contests so that they will be in a period of maximum performance ability by controlling the time at which ejaculation occurs. Though the performance measures are objectively determined, the remainder of the design leaves some room for doubt regarding the extent of control over human Ss. If a direct physiological is involved, then it certainly is not invalid on the basis of a small sample. Steinhaus (1963) maintains that this finding was a preliminary report based on one season's observations of five Ss--which only hints at a

possible relationship between sexual behavior and athletic performance. On the animal level Slonaker of Stanford in his study of activity in relation to the gonads of the domestic rat found that in both the male and female spontaneous running activity is considerably decreased after gonadectomy (Steinhaus, 1963). This reduction of running activity was not the finding in the case of non-domestic rats. Richter and Uhlenhirth (cited by Young, 1961) found that castrated wild Norway rats showed only a very slight decrease in running activity. As an explanation for this difference between domestic and wild rats the authors call attention to the further finding that the adrenal glands in wild rats are significantly larger and thus provide a sufficient quantity of the necessary steroids. Castrated domestic rats with smaller adrenals increase their voluntary running activity when injected with additional adrenal steroids.

Androgens and Mating Behavior

One of the general findings regarding sexual behavior in the human male is the strong relationship between androgen and sexual behavior. Androgen, commonly considered the "male" sex hormone, is secreted primarily by the Leydig cells of the testes in the male, and secondarily by the cortex of the adrenal gland in both male and female mammals. Clinical studies of hypogonadal males

confirm that androgen is necessary to induce morphological maturity and to maintain a well functioning eroticism. Such hypogonadal and eunuchoid males do not mature sexually as normally expected. But such individuals do respond to androgen administration with extensive dilation of the vasculature and growth in size of the penis. Maintenance of an erection by complete engorgement of the organ with blood is facilitated by androgen. Although tumescence of the penis can occur in the absence of this hormone, the erection is generally not complete and long lasting. The other primary sex structures, seminal vesicles and prostate gland, remain immature until stimulated into growth by androgen; then the fluids from these glands which provide the media for the sperm are secreted only as long as there is sufficient androgen in the system. When hypogonadal individuals for various reasons neglect or discontinue their androgen injections, the most sensitive indicator is the ejaculate, which results in a decrease in volume until no fluid is emitted. Along with this decrease such individuals also report fewer erections, a lessened sex drive, and a definite decrease in the frequency of erotic dreams (Money, 1961).

On the animal level investigators have shown that androgen administered to intact male rats and rabbits increased the amount of sex behavior. When a quantity of testosterone propionate (androgen) greater than the amount

necessary to restore normal sexual functioning in castrated animals was administered, the Ss also showed an increase in the strength of sex behavior beyond the normal level--that level demonstrated before castration. On the other hand, castrated rats receiving less than the required amount exhibited sex behavior inferior to their pre-castration performance (Beach and Holz-Tucker, 1949).

Investigations reported during the past 30 years show that when the primary source of androgen is removed, as in castration, there is a gradual and significant decrease in sexual behavior for such lab animals as the rat, guinea pig, rabbit, hampster, and cat (Young, 1961).

The same situation holds for human castrates who experience a decrease in sex drive. In regard to mammals, then, there is some obvious relation between the quantity of androgen and the strength of sexual behavior. In consideration of this relationship, it is hypothesized that the secretion of additional androgens from the adrenal cortex as a result of chronic stress (as the findings of Selye and others indicate) in the form of exercise over a long period of time, should have some augmentative effect on sexual functioning in the male.

Further consideration of this relationship between androgen and the strength of sex behavior shows that it is not a simple one to one relationship. Although sexual activity is greatly diminished in the male rat after

castration, it is not totally lost. The decrease at first is certainly significant, but thereafter it proceeds gradually; for male rats have been known to demonstrate conspicuous sexual activity as long as one year after castration. Guinea pigs in the castrated state 30 days are unable to copulate but can be utilized as testors for estrus females (Young, 1961).

One proposed explanation for the persistence of sexual behavior, though considerably diminished following castration experiments, is the fact that the adrenal cortex also supplies androgen that may compensate for the loss of testicular androgens. In man the relationship between pathologies of the adrenal cortex and virilism is well recognized. Also according to Money (1961) Price and Howard attributed marked and extensive development of the prostate gland and seminal vesicles in castrated mice to adrenal androgens. Money furthermore presents the finding that rats injected with testosterone propionate definitely produce greater than normal weights of the seminal vesicles, prostate gland, and coagulating gland; also the level of fructose and citric acid in the prostate and seminal vesicles of these Ss was found to be greater when compared with normal animals. On the other hand, castrated animals deprived also of adrenal glands produce a complete involution of the ventral prostate. This same organ fails to

develop at all in the case of prepuberal rats that have been adrenalectomized and castrated.

Gersh and Grollman (cited by Price and Williams-Ashman, 1961) maintained in 1939 that the seminal vesicle and prostate impairment in castrated and adrenalectomized mice was a result of poor physical condition rather than a loss of adrenal androgens. It is possible, however, that Gersh and Grollman may have overlooked the relationship between good physical condition and androgen level. At any rate, the majority of evidence tends to show some relation between androgen from this secondary source, the adrenal cortex, and its effect on primary sexual structures.

The evidence in support of androgen from the adrenal cortex of castrated animals being sufficient to account for the persistence of sex behavior is not as certain, for Warren and Aronson (cited by Young, 1961) in 1957 have shown no difference in sexual activity between male hampsters that were castrated compared with those that were castrated and adrenalectomized.

In an attempt to explain this diminished but persistent sexual behavior pattern following the removal of testicular androgens, Beach (cited by Young, 1961) presents his translation of what Steinach has to say:

> Since testicular hormones are probably dissipated within a few days after castration the more prolonged survival of sexual responsiveness is best explained on the basis of a relatively

> enduring change in the nervous system. It may be suggested that once the Central Excitatory Mechanism has been sensitized by androgen, thus neural mechanism remains in an excitable state for some time after the responsible hormones are withdrawn. In the absence of testicular androgens the essential central elements gradually lose their responsiveness (p. 1183).

In contrast to Steinach's central theory, Nissen (cited by Young, 1961) proposes a peripheral theory to explain this sex behavior pattern persistence after hormones have been deprived. He maintains that androgenic hormones exert their main influence (action) through peripheral nervous structures rather than central nervous structures. Consequently, the gradual decrease in sex behavior pattern in the male is a result of the gradual loss in capacity of the penis for tumescence; and this loss in responsiveness results in a loss, absence, or diminution of sensory impulses that initiate and result in sexual behavior.

In view of these two theories it would seem that central neural tissue or peripheral tissue--neural or somatic--could have a range of variable sensitivity rendering it responsive to varying quantities of androgen; then the increment in hormone supplied by some stimulation of the adrenal cortex could have some influence on the sex behavior pattern.

Along with consideration of the primary sex structures some attention should be given to the possible

influence of sperm formation as a peripheral variable in affecting sex behavior. Studies of the mammalian testes conclude that androgen is responsible for the maturation of the spermatid; however, the formation of androgen is in part dependent on the anteriorpituitary releasing the gonadotrophic hormone which stimulates androgen in the Leydig cells of the testes, the site of sperm production. Apparently there is a biological limit to the rate at which sperm can develop (Albert, 1961). Certainly the healthy and more efficient organism will be closer to this optimal functioning than the slightly less efficient. The pressure resulting from the formation and storage of sperm volume could very well result in peripheral messages governing sexual arousal.

Hormones and Neural Tissue

Evidence thus far clearly demonstrates the interactional relationship between the male sex hormone and neural tissue. Grunt and Young (cited by Young, 1961) maintain that the role of the male hormone is twofold; during early embryonic and fetal periods the action of the hormone is organizational in its action on neural centers that later become involved in the display of mating behavior. After maturity of the neural tissue the male hormone is activational; it acts on neural and muscle tissues participating in the display of mating behavior.

The organizational function of androgen has been demonstrated by experimental embryologists who have shown that genetically male rabbits and mice castrated *in utero* prior to genital tract differentiation follow the feminine pattern of morphology regardless of the genetic sex--thus demonstrating rather clearly the possibility of prenatal gonadal hormones playing a role in the differentiation of neural tissue mediating sexual behavior. When fetal testes were transplanted into female fetuses, inhibition of the female portion of the genital tracts and persistence of the male portions occurred in the vicinity of the graft. From these studies it was concluded that differentiation and organization of male genital tract and accessories occurs only in the presence of testicular hormones; whereas, differentiation of genital structures is independent of ovarian hormones. Female guinea pigs treated prenatally with testosterone propionate possess as adults an intensified capacity to display masculine behavior (Grady, Young, and Phoenix, 1965).

The primary elements of female sexual behavior not ordinarily exhibited by the male rat are displayed by the adult male rat that is castrated neonatally. In fact, castration after birth--prior to the first 10 days of age in male rats--produces *Ss* deficient in display of masculine behavior, normal penile growth, and neural differentiation. One essential point here is that previously it was believed

that inadequate or incomplete male behavior was due solely to peripheral factors such as inadequate penile development. But the responses of very early castrates, one to five days after birth, as contrasted with pre-puberal castrates after about 15 days of age, suggests the strong importance of central neural factors--which may be equal to peripheral morphology or less dependent on peripheral morphology than was commonly believed. In such Ss the absence of a vagina did not prevent the display of female behavior closely resembling the normal female (Grady et al., 1965).

Early Experience and Neural Tissue

Since it has been shown that there is a qualitative relationship between early social experience and mating behavior in the monkey, other investigators working with animals lower on the phylogenetic scale have found some interesting quantitative relationships. For example, Valenstein, Riss, and Young (1955) have shown that guinea pigs raised in an environment of social interaction demonstrated superior mating performance when compared with guinea pigs raised in isolation alone. Both groups were then castrated and allowed to reach a basal level in sexual performance. Replacement therapy of testosterone propionate resulted in the two groups reaching a level of performance that characterized their difference prior to

castration--thus presenting evidence that responsiveness of tissues mediating mating behavior to androgenic stimulation had been altered by an experiential factor. Apparently the amount of hormone produced to activate the neural tissue at maturity after the Ss had undergone these two conditions is not as critical as the earlier organization and alteration of the neural tissue itself. Consequently, by the time of full sexual maturity the behavior pattern displayed in response to the activation of androgenic stimulation is a result of the coaction of genetics and experiential factors.

An interesting species difference between the rat and the guinea pig should be mentioned at this point. While the strength of the sex drive in the male rat is considered proportional to the amount of androgen, data from male and female guinea pigs support the alternative hypothesis that the neural tissue which mediates sexual behavior is limited in responsiveness or threshold. In other words, a minimum amount of androgen is required to activate the neural threshold that mediates sexual behavior; an excess of androgen has no further effect (Young, 1961). In the male rat this threshold of responsiveness is less dichotomous and more of a continuous range that is responsive to differential quantities of androgen (Beach and Holz-Tucker, 1949).

Obviously, then, the somatic or constitutional factor which is genetically determined--but somewhat susceptible to experiential organization--nevertheless, tends to limit the action of the hormone and account for differences within species as well as between them. Grunt and Young (cited by Young, 1961) visualize the situation with the following analogy: the somatic substrate is compared to the exposed but undeveloped film; the hormone is equivalent to the developer substance; and the pattern of behavior brought about by the hormone is the picture.

Neural Tissues and Mating Behavior

Identification of neural tissue correlated with sex behavior so far has been somewhat of a problem. It has long been held that neural mediators of sexual behavior differ for the two sexes. In the male the neo-cortex is more heavily involved in mating performance than in the female. Hormonal removal, as in the case of castration studies, results in a gradual reduction and eventual disappearance of sex behavior in the male rat; in replacement therapy restoration of functioning is also gradual. In the female the disappearance of sex behavior after hormonal removal is abrupt; restoration likewise is abrupt for the female (Young, 1961).

As a result of chemical investigations of sex behavior centers in the brain, Kawakami and Sawyer (cited by Young, 1961) in 1959 expressed the problem thusly:

> The areas in the brain which are affected by steroids are so extensive as to suggest that the whole nervous system is influenced primarily and localized systems of integrated behavior secondarily. Sex steroids affect simultaneously . . . the midbrain reticular system, which probably includes a mammillary body mating behavior "center" and the rhinencephalic-hypothalmic system which includes the basal tuberal gonadotrophic "center" (p. 1207).

As far back as 1956 Fisher found that testosterone sulfate injected into the lateral preoptic area of male and female rats induced mating behavior; within minutes after the time of injection the rat would engage in mounting behavior on a partner. If the same injections were moved a little to the medial area of the hypothalamus, both male and female animals displayed coordinated elements of mating behavior (Fisher, 1956). Further investigations by Harris, Michael, and Scott (cited by Pfaff, 1965) in 1958 and Lisk (cited by Pfaff, 1965) in 1962 tended to confirm the hypothalamus as a center for mating behavior that could be activated hormonally by an androgenic compound. An interesting conclusion of this work was the finding that effective sites for inducing mating behavior could not be distinguished from ineffective sites on the basis of the two biophysical parameters: (1) the rate of hormonal

diffusion, and (2) the degree or distance of spread of the hormone.

In ablation studies of the cortex of the male rabbit and rat it was found that there is a decrease in the proportion of males that continue to copulate when lesions involve 20 per cent of the neopallium. As lesion size increases the proportion of male animals that copulate on a given test decreases. When the total extent of the lesion exceeds 75 per cent of the cerebral cortex, sexual reactions are eliminated. The significant point here is that this behavioral change does not appear to depend on the locus of the lesion within the cortex--a finding which obviously tends to argue against any specific "cortical sex center." In other words, the greater the extent of cerebral damage in any given animal, the lower the probability of that animal engaging in mating behavior on a given test. Such Ss would show fewer positive tests of mating behavior; but once mating was initiated on any one of the few positive tests the number of copulations during this test would not differ from that of an intact control S. The full sex behavior pattern was not affected--only the percentage of positive mating sessions. Apparently cortical lesions have no effect on the copulatory behavior pattern but do affect the ease of sexual arousal or the percentage of times the animal will manifest the incentive to mate (Beach, Zitrin, and Jaynes, 1956).

In considering such an apparent dichotomy in the total sexual behavior pattern and also the lack of a direct quantitative relationship between androgen and sex behavior, Beach has proposed that the total sex behavior pattern is a result of two different mechanisms--each with its own threshold or level of responsiveness to hormones--an arousal mechanism (AM) and a consummatory mechanism (CM)--involving copulation and ejaculation (Beach and Jordan, 1956; Young, 1961).

The findings of other investigators lend support to Beach's proposal. Maclean's (1965) stimulation techniques with the brain of the squirrel monkey resulted in his uncovering strongly positive points of penile erection in the cingulate gyrus just rostral to the knee of the corpus callosum. In the male the positive loci for erection that were found in the forebrain and diencephalon were distributed along three corticosubcortical divisions of the limbic system: (1) the hyppocampal projections to the system, anterior thalamus and hypothalamus; (2) various areas of the Papez circuit comprising the mammillary bodies, mammillothalamic tract, the anterior thalamic nuclei and anterior cingulate gyrus; and finally (3) parts of the medial orbital gyrus, dorsal medial nucleus of the thalamus, and regions of other known connections. In regard to this last finding, Maclean calls attention to the prefrontal lobotomy which severs connections between the

medial dorsal nucleus and the orbitofrontal and prefrontal cortex and which sometimes results in bizarre, uninhibited sexual behavior.

In the stimulation of these cortical areas mentioned above, ejaculation does not occur. By the mapping of various areas of the brain, Maclean and others have found a number of points lower in the thalamus and along the course of the spinothalamic pathway which elicits sexual discharge with motile sperm. In fact, seminal discharge was even achieved independent of penile erection; more specifically it anticipated the appearance of penile erection. Such findings recall once again the evidence that erection is a parasympathetic phenomenon and ejaculation a result of sympathetic activation (Maclean, 1965).

Each of these findings coincide with the two physiological mechanisms proposed by Beach and Jordan (1956). The Arousal Mechanism (AM) includes motor activities associated with the appetitive aspect; it is innervated essentially by the voluntary central nervous system and has a high degree of cortical representation. Androgen is said to raise the excitability of the cortical areas involved in this AM and (1) increase the male's susceptibility to arousal and (2) lower the threshold in neural circuits mediating male copulatory behavior. The Consummatory Mechanism (CM) involves such behavior as mounting, copulation, and ejaculation; it is more

represented in the subcortical regions of the brain and the spinal thalamic tract. Penile erection belongs essentially to the AM and ejaculation, to the CM. Yet both functions are innervated through sacral antonomic nerves and may be stimulated to function independent of the motor part of the pattern (Young, 1961).

The CM of primates, both male and female, does embrace centers and systems extending into the neocortex; this is true also for male carnivores, but not true for animals lower on the phylogenetic scale including male and female rodents. In the male rat, and probably the female, the highest centers of the CM lie in the diencephalon. Such an anatomical placement would explain how the coital act for these animals can be performed without practice (Young, 1961).

For male rodents and male carnivores and both male and female primates the AM includes cortical elements; this is evidenced by the observation that castrates can copulate once the acquired degree of arousal is attained; male rats with a considerable proportion of the cortical area removed (most of the AM removed) can still copulate well on any one test once they are aroused, but not on very many tests since arousal is difficult to achieve (Beach et al., 1956). The essential point here is that it is very likely the thresholds of these neural mechanisms, if somewhat

distinct, may differ; thus the whole sexual behavior pattern may be responsive to variable quantities of androgen.

The main implication of these findings is that the total sexual behavior pattern can justifiably be considered as consisting of an integrated function (action) of two separate neural mechanisms--an AM and a CM--each with its own structural and functional components--each with its own threshold to hormonal stimulation; the threshold of each may be variable and the range of each may overlap and interact. If so, then it would seem reasonable to expect the total sexual behavior pattern that emerges to be influenced by the quantity of circulating hormone--androgen--which can interact with the neural tissue. Furthermore, it is possible that varying amounts of this hormone may have some organizational effect on the neural tissue. In a study by Green, Clement, and deGroot (cited by Young, 1961) in 1957, it was demonstrated that prepuberal cats displayed masculine behavior when testosterone was administered and feminine behavior when stilbestrol tablets were given intramuscularly. This effect before puberty was reversible; but after puberty, only masculine behavior could be elicited by both hormones; now the effect was irreversible. The authors' explanation for the reaction to steroids before and after puberty is speculative but worth considering.

> It's unlikely that education in any ordinary sense is involved after the first coitus . . . the type of first sexual experience may determine the subsequent behavioral reaction to hormones that have the same effect on the secondary sex characteristics and reproductive tract both before and after puberty. . . . Perhaps the simplest explanation is that before sexual experience the steroids set the stage for later education and determine the animal's receptivity to another kind of experience; that is, they determine the mood of the prepuberal animal. Without full experience, then, the mood is reversible; but once experience has been acquired the pattern of behavior is set and is not changed by administrations of the steroids of the opposite sex. The steroids in the adult would thus be assumed to increase drive in a non-specific way (p. 1221).

Of course, there are difficulties in this hypothesis if it is to be extended to a consideration of the female. But here again, as in the situation of guinea pigs raised in an environment of social interaction, is evidence that the responsiveness of tissues mediating sex behavior to androgenic stimulation has been determined apparently by an experiential factor.

One common variable or element composing the experiential factor could be the element of stress. Stress is obviously an experiential factor; and it is the task of this thesis to determine if chronic stress in the form of physical exercise during this prepuberal period might not set the stage for augmenting post puberal mating behavior by its interactive effect on neural tissue and early hormonal stimulation. It is already known that such stress does seem to augment androgen and other steroid

secretions from the adrenal cortex. Consequently, it is hypothesized that the increased metabolic stress placed on the nervous system due to such chronic exercise will tend to sensitize the neural tissue to the added amount of androgen and other steroids in the system which are produced from alterations of the cortex as a result of stress. It is also known that androgens are specific in bringing about adult growth of the genital tract in the male (and estrogen in the female) and that androgen does have an effect on increasing nitrogen metabolism and thus general muscular size and strength (Money, 1961). A similar effect but more difficult to discern could occur with certain neural tissue thus affecting its range of reactivity or threshold.

METHOD: SUBJECTS, APPARATUS, AND PROCEDURE

Selection and Maintenance of Subjects

The Ss consisted of 35 male hooded rats of Long-Evans strain. At the start of the experiment, all rats were about 40 days old. After randomly dividing them into experimental and control groups of 17 and 18 Ss respectively, all Ss of each group were housed together in the same cage separated into two distinct compartments, both equally large enough so that over-crowding was not a problem. Both groups were fed Purina lab chow and water ad libitum and maintained in the laboratory with a regulated light-dark cycle. During the earlier days of the experimental procedure one S from each group died from causes unknown. Midway through the experiment one experimental animal drowned leaving a final number of 17 control and 15 experimental Ss.

In addition to the males primarily used in the experiment were 18 females of the same age and genetic strain. All the females were kept together in a large cage under similar laboratory conditions. All were ovariectomized when approximately 80 days old so that estrus could be manipulated experimentally by exogenous injections of estrogen and progesterone.

Two Ss died postoperatively leaving 16 for testing purposes.

Apparatus

The swimming apparatus consisted of 15 five-gallon buckets. The dimensions of each container were 11.5 inches in diameter and 14 inches high. The inside of each container was uniformly white in color. At the initial stage of the experiment water 10 inches deep was sufficient to prevent the rats from reaching either the top or bottom while swimming.

After the first 50 days of swimming exercise aluminum siding riveted together forming a circle coinciding with the circumference of the five-gallon buckets was fitted around the top of each. This extended the containers four more inches in height and allowed at the same time an increase in water depth to approximately 13 inches. With this added construction some of the larger rats were unable to let themselves sink under water to the bottom and spring up high enough above the surface to reach the edge of the bucket and escape.

The testing apparatus for mating behavior consisted of a special observation cage. Compared to the semi-circular cages used by Beach and Jordan (1956) and Hsiao (1965), this observation cage, constructed from cardboard, was 23 inches high. The front side of each box contained a

wire screen window nine inches high from the bottom and 20.5 inches wide. The rear half of the top was hinged to allow introduction and removal of the Ss by hand.

Procedure

Independent Variable

At approximately the same time each day during the light phase of the 24-hour cycle, Ss from the experimental group were placed in one of the two cages used as "waiting stations" prior to the exercise procedure. One at a time they were taken by hand from the "waiting station." Lead weights fastened snugly by wire to a small long-nosed alligator clip were fastened to the fur of the S's back side. These weights were from 10 to 15 per cent of the S's total body weight. It was necessary to introduce the weights after the first 10 days of swimming exercise to prevent the Ss from floating and thus maintain the rat at a relatively constant swimming pace.

Each was then gently placed in an individual swimming bucket with a water temperature ranging from 82 to 85° F.

The Ss of the experimental group were exercised for 20 to 25 minutes five days a week for a period lasting more than 100 days. Periodically the weights were gradually increased to correspond in percentage with the animal's increase in body weight.

At the end of a swimming session each S was individually removed from the bucket in the same order as the session began to control for equality of exercise time.

To accustom the animals to the initial stress of swimming, the first 10 sessions were without weights. The first exercise session was for five minutes and every session thereafter was increased one minute. The very first session with weights was for 16 minutes. The daily time increase of one minute per session was continued until a period of over 20 minutes of exercise was attained. Thereafter, for the remainder of the 100 day exercise period, the time per session remained constant.

Every time the experimental group was exercised, the control group was handled and transported in the same way as previously described. Each S in the control group was gently submerged in a bucket of water and held there in the experimenter's hand with its head above water for three or four seconds. The purpose here was to control for such variables as handling, exposure to water, and grooming behavior. Both groups were weighed at approximately eight-day intervals throughout the experiment.

Dependent Variable

Incentive females previously ovariectomized were brought into heat by injections of .10 mg. of estrodiol benzoate 60 hours prior to testing and 1.0 mg. of progestin

10 hours prior (Hsiao, 1965). Only fully receptive females were used in each test. If a female was found to be non-receptive, which was rarely the case, it was replaced by another. No attempt was made to pair each male with the same female in different tests.

In order to facilitate the mating process, it was considered a slight improvement to cover the cardboard floor with newspaper shavings instead of the conventionally used sawdust. Three observation cages were placed on a firm table in such a position that they could all be watched simultaneously by one observer seated at a desk approximately four feet away. Each of the three observers was equipped with a large-sized tablet for recording and a stop watch so that he could record the start and finish of each mating sequence. Later, when the records were analyzed, the frequency and timing of response could be determined.

When the Ss were about 90 days old, a random selection of eight from each group was selected to test for mating behavior. This test was arranged in time to follow a natural two-day rest sequence from the exercise. On the day of mating, however, the animals received no swimming exercise.

Before the start of a test the male was placed in the mating cage and allowed at least five minutes for cage adaptation.

At the end of this period the receptive female was introduced. Because of the small percentage of mating behavior in both groups at this time, it was decided to begin testing when the Ss were about 110 days old to insure that all Ss were sexually mature. This was called the preliminary test.

Following two days of rest from the exercise the preliminary test at this age consisted of recording only the mount latency (the time required for the male to engage in mounting sexual behavior once the receptive female had been first introduced into the cage). Both male and female were removed as soon as mounting behavior occurred. Males that failed to mount within thirty minutes of the test were removed from the cage and scored as negative.

The 100 days of swimming exercise were terminated when the Ss were about 150 days old. Following two days of rest the Ss began a formal test for mating behavior. The formal test consisted of two mating sessions separated by a three-day rest interval. Each mating session was limited to a two-hour period. It was concluded from the normative data of Beach and Jordan (1956) that two hours was sufficient for sexual exhaustion to occur for most male rats. Also a three day recovery interval before the second session seemed an ideal time interval for incomplete recovery from the first session. Because of the large number of Ss involved, the animals were tested on different

days, and in order to control for age between the two groups, the Ss were rank ordered and selected according to corresponding body weights. Because all Ss could not be tested on the same day, the termination dates for exercise were spread out over a period of approximately 10 days, always leaving a two-day rest between termination of exercise and the mating test. During each of the two-hour mating sessions, each of the three observers recorded the following response measures: (1) the mount latency, (2) the number of intromissions prior to the first and second ejaculation, (3) the ejaculatory latency (the amount of time required for the S to achieve ejaculation once mounting behavior had been initiated) for both the first and second ejaculations, (4) the relative refractory period (the rest interval between the first ejaculation and the next mounting response), and (5) the total number of ejaculations of each S during the two-hour interval. All testing was done during the dark phase of the 24-hour cycle.

RESULTS

The overall effect of the independent variable, exercise, on body weight can be seen from Figure 1 where the mean weights of the two groups of Ss are presented at approximate eight-day intervals throughout the entire exercise period. The figure clearly shows that the exercised Ss are consistently lighter in average weight. The mean difference between the two groups has not been found to be significant at any one age; however, no attempt was made to control for the variance between the two groups by pairing the Ss according to initial weight. Though the slope of the curve gradually decreases, the mean weight for both groups is still increasing at 150 days of age--the termination of exercise.

The first observation of sexual behavior, the preliminary test, was done when the Ss were about 110 days old. The formal test consisting of two sessions was conducted when the Ss were about 150 days old. The results of the preliminary test and the two sessions of the formal test of response measures of sexual behavior are presented in Table 1. In the preliminary test only the mount latency was recorded and found by a t test to be non-significant at the five per cent level. In this test it should be noted that only 70 per cent of the non-exercised Ss--12 out of

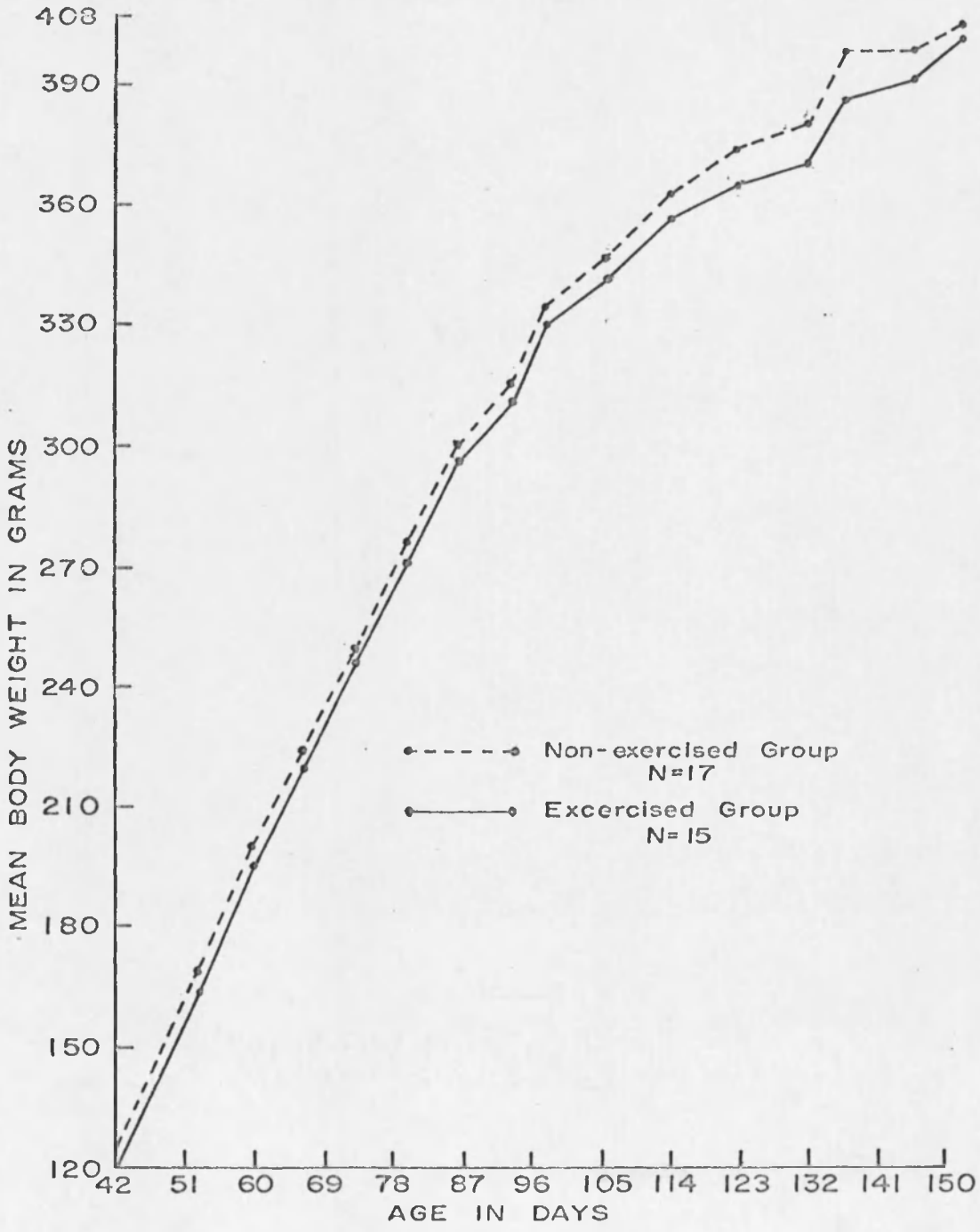

Fig. 1. Mean body weight in grams of exercised vs. non-exercised group measured at various ages during the 100 day exercise period.

Table 1

Mating Test Results

	Preliminary Test (Non-Exercised) (age 110)		Preliminary Test (Exercised) (age 110)	
Mount Latency	697.4 ± 790.2	N = 17 S = 12	502.9 ± 757.4	N = 15 S = 12

	Formal Test (3 Days' Rest) Session 1 (age 150)				Session 2 (age 150)			
	(Non-Exercised)	S	(Exercised)	S	(Non-Exercised)	S	(Exercised)	S
Mount Latency	562 ± 927	16	83 ± 113	14	194 ± 275	15	277 ± 400	14
Intromissions (1st Ejac.)	16 ± 10	13	18 ± 8	14	18 ± 11	14	20 ± 7	14
Ejac. Latency (1st Ejac.)	804 ± 1013	13	762 ± 759	14	920 ± 1456	14	890 ± 524	13
Refractory Period	410 ± 253	13	562 ± 358	14	614 ± 184	14	622 ± 129	12
Intromissions (2nd Ejac.)	12 ± 5	13	10 ± 4	14	13 ± 7	14	13 ± 6	13
Ejac. Latency* (2nd Ejac.)	641 ± 475	12	347 ± 177	14	433 ± 275	14	383 ± 177	13
Ejaculations	4 ± 2.78	17	5 ± 1.98	15	3.4 ± 1.6	17	3.3 ± 1.5	15

N = Total number in sample.

S = Number in sample that responded.

*Significant at the 5% level.

17--engaged in mounting the female within the 30 minute criterion. Of the exercised group, 80 per cent or 12 out of 15 engaged in mounting responses; consequently, an arbitrary value of 1800 seconds was assigned to the non-responders (this value coincides with the standard used by Beach and Jordan in the Normative study in 1956). A X^2 test between the two groups comparing maters with non-maters did not yield a significant difference.

The formal test consisting of two mating sessions included the following response measures for each session: (1) mount latency, (2) first ejaculatory latency, (3) intromissions for first ejaculation, (4) the relative refractory period, (5) second ejaculatory latency, (6) intromissions for second ejaculation, and (7) the total number of ejaculations per two hour session. A series of *t* tests were run to determine if the difference between the means of these response measures were significant. Both groups were compared within sessions and between sessions and found to be non-significant at the five per cent level; however, one important response measure, the ejaculatory latency preceding the second ejaculation--after the males were initially accustomed to the females, were still fresh enough, and well on their way in the mating behavior pattern--was found to be significant at the five per cent level for the first mating session only. The mean number of ejaculations for each group also includes those *S*s that

did not respond to the female and were consequently assigned zero values, which were included in the calculation of the group mean. For the remainder of the response measures, except also for the preliminary test of mount latency, the Ss that failed to participate or mate were not included in the data as the N in the table indicates. In each group only one S failed to respond consecutively on both of the formal sessions. In the non-exercised group, however, an additional four more Ss failed to perform during one of the two sessions. In the exercised group, only one other S did not perform on the second session. Again, a X^2 test showed no significance for such a small difference. In only two cases were response measures considered invalid and eliminated from the data due to recording error in spite of the high degree of reliability of the three observers.

In addition to sexual behavior a noticeable difference in social behavior was especially prominent about seven days after termination of the last group of maters. Four experimenters working in the laboratory at various times during this period claimed the Ss had been periodically fighting the past several days; but being unfamiliar with this experiment, they were unable to designate for certain whether it was the control or experimental group that was doing the fighting. The following day it was observed by the experimenter that just about all of the

experimental animals were raised up on their hind legs, paired off, and facing each other stone still prepared to fight. This pose lasted anywhere from about 10 to 20 seconds until one rat in the group initiated an aggressive response to its paired mate resulting in an instantaneous chain reaction of fighting behavior between each pair of the previously motionless animals. Only a small number of non-competitors seemed to be crowded in one of the corners of the cage--about three or four in number. By the following day, after a series of skirmishes, one of the laboratory assistants kindly removed two of the experimental animals that had rather severe and bloody wounds on the face and genital areas--placing them in an individual cage. To prevent any further destruction all the Ss of both groups were placed in individual cages later that day. A head count was then made to determine the number of Ss in both groups that had obviously noticeable cuts on their faces. None out of 17 in the control group showed any visible signs of facial or body wounds; but in the experimental group 9 out of 15 were found to have cut and swollen faces due to bloody wounds. A X^2 test comparing the findings of these two groups was significant at the .01 level suggesting that exercise may have some relationship to fighting or aggressive behavior.

DISCUSSION

On the basis of the evidence presented thus far it has been rather difficult to discern the precise relationship between stress in the form of physical exercise and sexual behavior. It has been established by Hatai (1915), Steinhaus et al. (1932), Anderson (1935), and Selye (1956) that physical exercise can result in endocrinological and morphological changes of the organism. It has also been established by Valenstein et al. (1955) that the interactive effect of hormones such as androgens on neural tissue mediating sexual behavior had been altered by an experiential factor--the stress of early social interaction. In fact the stress of the first coital experience in the cat tends to set the stage for the irreversible expression of masculine behavior in the male (Young, 1961). The positive relationship between the quantity of circulating androgen and the sex drive in the male rat is well established (Beach and Holz-Tucker, 1949).

Though the mean trend of the data in Table 1 is slightly in favor of the exercised Ss, significance has been difficult to obtain due to the overlap in variance of the two groups. There is some indication that the Arousal Mechanism as reflected by the mount latency in particular may be slightly affected by the exercise variable. The

response measure which was significant at the .05 level, the ejaculatory latency for the second ejaculation, may reflect an interactive combination of both the CM and AM. The actual time, the latency, is more reflective of the AM while the copulatory behavior itself, the intromissions leading up to the second ejaculation, are essentially consummatory responses. In essence it is rather difficult to clearly separate the two concepts of Arousal and Consummation, which may overlap considerably.

Methodologically it would be very difficult to control for the extreme overlap of variance of these two groups of Ss. A design that pairs the Ss according to prior mating performance would be ideal; however, such a design would be impossible for prepuberal Ss. The closest criteria that could be used would be to match the Ss according to litter, which would insure a greater degree of genetic control.

It is possible that the stress variable of exercise did have a more significant effect on sexual behavior than the results reflect--especially if it was counteracted or masked by the performance of those marginal Ss that ordinarily would not have mated but were influenced enough by the independent variable to perform, and do so rather poorly; such a result would account for the considerable overlap in variance between the two groups as well as the greater number of Ss in the control group that did not

participate and, consequently, were not included in the data. Since prepuberal Ss cannot be paired according to mating performance, perhaps a statistical control could have been employed to account for this "marginal factor" by automatically eliminating the worst five performances of each group including non-maters. Such a technique would eliminate marginal Ss from the experimental group and their corresponding equals in the control group.

Another complicating factor in this design is the fact that endocrine functioning at adolescence as far as male sexual functioning is concerned is at optimal level. Kinsey's et al. (1948) studies show that the sex drive for the male is optimal in the late teens. Likewise, it is certainly likely that the endocrine functioning of the Ss of both groups was asymptotic; one could then conclude that early physiological development is much more heavily influenced by genetic determination than any experiential factor from the environment introduced after the very early critical period of neural organization had already occurred.

In addition it must also be recognized that both groups were subjected to a considerable degree of stress; both were housed in group cages which afforded plenty of social interaction; both were subjected to the same amount of handling; and both were exposed to water. It is quite possible that in spite of the exercise administered, the

overall stress of both groups was not much different; each group may have represented points too near each other on the same continuum. Perhaps such is the price that must be paid if the stressful effects of social interaction between groups are to be controlled. It is quite possible, then, that a minimal amount of stress, as is a minimal amount of androgen in the guinea pig, is sufficient stimulus to reach the threshold, which, once elicited, leads to optimal behavioral functioning. It is quite possible then that an experimental design which included a third group of Ss raised under isolated conditions would have yielded a significant difference in mating performance when compared with the two groups used in this design.

In view of the overall asymptotic sexual performance of adolescent mammals resulting primarily from the strong influence of genetic and developmental factors, it would certainly be worth considering the effects of such experiential factors on mating behavior during the stage of general decline in the human and other mammals. It is a generally accepted biological principle that many physiological and neurological behavior patterns, as well as some psychological patterns, and sexual activity steadily decline after 30 years of age on the average. Observations on male rats, rabbits, and guinea pigs indicate that copulation frequency is lower in older animals. Some of the more careful observations indicate that ejaculations

per hour by male rats increase up to one year of age and then decrease steadily after 20 months. Intromissions, which are greatest at puberty, decline slowly but steadily thereafter. The duration of each series of copulations is prolonged as animals age (Young, 1961).

In view of this finding an experimental design that pairs Ss into two groups based on a preliminary mating test conducted at one year of age and then introduces one of the groups to an exercise routine for several weeks or one or more months and then exposes the Ss to a final mating test may result in some highly significant findings.

Given enough time, assistants, and money, several groups could be matched according to a preliminary test and then be subjected to varying ranges of the independent variable in order to determine the precise relationship between quantity of exercise and improved mating performance.

Compared to the design of this study, the reasons for the findings, however, will be somewhat different; for, after all, the neural tissue has developed beyond any organizational influences. The effect of exercise in this design would be restorative; a more efficient biological organism--as a result of superior health--would account for improved performance in many other behavioral areas as well. The gradual decrease in gonadal activity which occurs in the later decades of life of the male is due not

to a failure of gonadotrophic stimulation (which if it were, exercise would possibly correct) but more probably due to a gradual diminishing blood flow through the gonads; this condition may in turn be related to arteriosclerosis and other cardiovascular and pulmonary changes that occur with advancing age (Steinhaus, 1963).

Before proceeding any further some mention should be made regarding the nature of the independent variable itself. Exercise is one specific form of stress. In this design one cannot overlook the possibility that exercise could have been contaminated or confounded with some other variable which is also stress provoking; for when the Ss were removed from their respective swimming containers at the termination of any one exercise session, approximately half of the buckets contained water which was considerably contaminated by the urine and fecies of the Ss. The extent of contamination did not appear to be significantly different when the Ss were undergoing the terminal stage of the design--the last several days--than it was the first few days at the beginning. Perhaps one inference that can be drawn from this observation is that the swimming experience may very well have been a rather frightening undertaking for these animals; the same response, instantaneous elimination, occurs when the laboratory rat is subjected to a pain inducing stimulus, such as a strong electric shock. It was anticipated at the beginning of this

experiment that swimming might be a rather fearful or negative experience for the Ss involved; however, the general expectation was that such a response would eventually extinguish in the face of well controlled cues from session to session in which the Ss were unharmed and always rescued from the water after the same amount of time.

The most interesting finding of this design and the most significant result obtained was the difference in outcome of the social behavior between the two groups. As the findings clearly indicate, the combative behavior of the exercised group resulted in obvious facial disfigurement from severely noticeable lacerations. No such outcome was the result of the non-exercised Ss. Though several graduate students conducting other experiments in the laboratory maintained that both groups were fighting at various times, cross-examination of the various observers seemed to strongly indicate a greater frequency of this combative or aggressive behavior occurring in the exercised group. Such a naturalistic observation was certainly the finding of the experimenter; and on the basis of this observation it would seem that whatever combative behavior the non-exercised group expressed was much lower in both frequency and severity. Since only quarter inch wire design separated the two cage compartments, it is quite possible that whatever combative or aggressive behavior the

non-exercised group displayed was initiated by the proximal influence of the exercised group.

In view of these results there seems to be some significant relation between exercise and combative or aggressive behavior. In the sociobiologic studies of Harlow much emphasis has been placed on the well established naturalistic observation that males are more aggressive by nature than are females--that it was the males that consistently made more aggressive and threatening gestures to each other while the females engaged in more grooming behavior (Harlow, 1958). Further studies conducted on such widely different infraprimates as fish, reptiles, birds, and mammals have shown that high scores on tests of dominance and aggressiveness are highly correlated with the amount of androgen concentration in the blood. Furthermore administration of androgen often produces an increase in aggressiveness of normal adults, both male and female, and castrates, as well as result in precocity in immature individuals of either sex. Male rats injected with androgen became irritable and pugnacious. Consequently, the general conclusion is that an increased concentration of androgen enhances dominance and aggressiveness of both male and female animals (while estrogens either reduce aggressiveness or are without effect). In considering this convincing correlation between androgen and aggressiveness, Ginsburg and Allee (cited by Guhl,

1961) also call attention to the experimental findings that psychological factors can also be instrumental in the attainment of a state that would have formerly been thought to be solely the consequence of hormonal achievement.

In the particular case of this experiment there may be several explanations that can possibly account for the extreme combativeness (aggressiveness) of the exercised group.

1. In the two week interval following the termination of 100 days of chronic exercise, full metabolic recovery--both somatic and neural--could have finally resulted, leaving an organism with a high nervous energy level and low threshold for irritability--thus creating a need for vigorous activity to reduce the built-up tension state.
2. The organism of the exercised Ss could have been physiologically and experientially conditioned to the scheduled requirements of daily energy expenditure. Without this reinforcing release in tension--this decrease in energy level--the Ss left to their own methods engaged in more vigorous play behavior which became too rough and culminated in combative, aggressive behavior.
3. A definite increase in the concentration of adrenal androgens as a result of full recovery and/or glandular conditioning to a scheduled period of

stress could have resulted in increased irritability and a lower threshold for aggressiveness.

4. Some other little understood or as yet undiscovered phenomenon could have produced the observed combativeness.
5. The aggressive combative behavior may have resulted from some interaction of the above mentioned explanations.

Whatever the mediating or intervening variables in the display of aggressive behavior, it cannot be denied that stress in the form of exercise was the only variable that significantly and decidedly differed between the two groups of Ss. Only further investigation and more carefully planned designs will be able to show a more quantitative relationship between exercise and aggressiveness.

Before closing this discussion some mention should be made of the relationship between sexuality and aggression. It was Freud who first called attention to the close relationship and interaction of these two behavioral manifestations in his theoretical formulations regarding the human being. Maclean (1965) has further substantiated the relation between sexuality and aggression on a physiological level. Especially in lower animals fighting or combative behavior is intimately related to both feeding and mating. In terms of neurological locations, if one

proceeds caudally from the level of the anterior commisure in the brain of the squirrel monkey, one can follow neural structures involved in fearful, angry, or combative behavior lying extremely near to those concerned with feeding and sexual responses. Within the small space of one millimeter electrical stimulation can result in erection in conjunction with an angry or fearful type of vocalization and bearing of fangs. A very slight lowering of the electrode results in a fearful or angry response with erection resulting as a rebound phenomenon upon termination or withdrawal of the stimulation.

The intimate relation between sexuality and aggression as a form of behavior apparently has its basis in a close neurological tie. In carrying this point further Maclean (1965) maintains that naturalistic observations on the squirrel monkey have well established the fact that the display of the erect penis in the face of another male monkey is a direct indication of hierarchical dominance--a response measure better than any competition for food, etc. This same displaying posture in relation to the female is part of the sex behavior pattern.

One can only speculate with some regret, however, what the outcome of the design would have been in terms of sexual behavior had the Ss been exposed to females during the time of this combative, aggressive behavior.

Perhaps then the relationship between the stimulation of enhanced sexuality and exercise may have been more clearly demonstrated. Along with such a demonstration would hang the conclusion that such an increase resulted from an experiential alteration of the endocrine and/or neural activation over a period of chronic stress in the form of physical exercise.

As the findings currently stand, it is very possible that the exercise enhanced the androgen circulation in the system after a period of full recovery; and like Harlow's monkeys the added increment of this hormone contributed to the rough and tumble play that is strictly reserved for the boys--in this particular case the boys with the greater quantity, and neural responsiveness to, androgen. At any rate such a finding does tend to support the relationship proposed by Money (1961) which correlates androgen with aggressiveness in his formulation of the vague and poorly defined concept of masculinity.

SUMMARY

To test the hypothesis that exercise has a positive effect on the sexual behavior of the male rat, 34 male hooded rats of Long-Evans strain were randomly divided into two groups at 40 days old. The experimental group, N = 16, was subjected to swimming exercise 25 minutes a day, five days per week, for a period of 100 days. When the Ss were 110 days old, a preliminary test to determine the difference in mount latency between the two groups was not found to be significant. After termination of the 100 day exercise period, a formal test consisting of two 2-hour mating sessions separated by a three day rest interval was conducted. The response measures included during these sessions were the mount latency, the number of intromissions, ejaculatory latency, post-ejaculatory refractory period, and the total number of ejaculations. All response measures were non-significant statistically except for the ejaculatory latency preceding the second ejaculation which was significantly faster for the exercised Ss for the first session only. Such a result may be interpreted to reflect some influence of exercise as a form of stress on the Consummatory Mechanism and possibly the Arousal Mechanism also of the sex behavior pattern.

An interesting naturalistic observation about two weeks after termination of the experiment revealed that the exercised group displayed a higher degree of combative, aggressive behavior.

REFERENCES

Albert, A. The mammalian testes. In W. C. Young (Ed.), Sex and internal secretions. Vol. I (3rd ed.) Baltimore: Williams and Williams, 1961. Pp. 305-365.

Anderson, D. The effect of food and exhaustion on the pituitary, thyroid, adrenal, and thymus glands of the rat. Journal of Physiology, 1935, 85(2), 162-167.

Beach, F. A. and Holz-Tucker. Effects of different concentrations of androgen upon sexual behavior in castrated male rats. Journal of Comparative and Physiological Psychology, 1949, 42(6), 433-453.

Beach, F. A. and Jordan, L. Sexual exhaustion and recovery in the male rat. Quarterly Journal of Experimental Psychology, 1956, 8, 121-133.

Beach, F. A., Zitrin, A., and Jaynes, J. Neural mediation of mating in male cats. Effects of unilateral and bilateral removal of the neo-cortex. Journal of Comparative and Physiological Psychology, 1956, 49, 321-327.

Constantinides, P. C. and Carey, N. The alarm reaction. A Scientific American reprint. San Francisco: W. H. Freeman, March 1949.

Donaldson, H. H. The effect of exercise continued for several generations on the weights of the organs of the albino rat. Science, 1931, 74, 573.

Fisher, A. E. Maternal and sexual behavior induced by intracranial and chemical stimulation. Science, 1956, 124, 228-229.

Grady, K. L., Young, W., and Phoenix, H. Role of the developing rat testes in differentiation of the neural tissues mediating mating behavior. Journal of Comparative and Physiological Psychology, 1965, 59(2), 176-182.

Guhl, A. M. Gonadal hormones and social behavior in infrahuman vertebrates. In W. C. Young (Ed.), Sex and internal secretions. Vol. II (3rd ed.) Baltimore: Williams and Williams, 1961. Pp. 1240-1267.

Harlow, H. F. The nature of love. American Psychologist, 1958, 13, 673-685.

Hatai, S. On the influence of exercise on the growth of organs in the albino rat. Anatomical Record, 1915, 9, 647-665.

Hsiao, S. Effect of female variation on sexual satiation in the male rat. Journal of Comparative and Physiological Psychology, 1965, 60(3), 467-469.

Kimeldorf, D. J. and Baum, S. J. Alterations in organ and body growth of rats following daily exhaustive exercise, x-irradiation, and post-irradiation exercise. Growth, 1954, 18, 79-96.

Kinsey, A. C., Pomeroy, W. B., and Martin, C. E. Sexual behavior in the human male. Philadelphia: W. B. Saunders, 1948.

Maclean, P. D. New findings relevant to the evolution of psychosexual functions of the brain. In J. Money (Ed.), Sex research: New developments. Chicago: Holt, Rinehart and Winston, 1965. Pp. 197-218.

Maslow, A. H. Reprint: A theory of metamotivation. Psychology Today, 1968, 2(2), 40.

Money, J. Sex hormones and other variables in human eroticism. In W. C. Young (Ed.), Sex and internal secretions. Vol. II (3rd ed.) Baltimore: Williams and Williams, 1961. Pp. 1383-1400.

Pfaff, D. W. Cerebral implantation and autoradiographic studies of sex hormones. In J. Money (Ed.), Sex research: New developments. Chicago: Holt, Rinehart and Winston, 1965. Pp. 219-234.

Price, D. and Williams-Ashman, H. The accessory reproductive glands of mammals. In W. C. Young (Ed.), Sex and internal secretions. Vol. I (3rd ed.) Baltimore: Williams and Williams, 1961. Pp. 366-448.

Selye, H. The stress of life. New York: McGraw-Hill, 1956.

Steinhaus, A. H. Toward an understanding of health and physical education. Dubuque, Iowa: William C. Brown, 1963.

Steinhaus, A. H., Hoyt, L. A., and Rice, H. A. Studies in the physiology of exercise: X, The effects of running and swimming on the organ weights of growing dogs. American Journal of Physiology, 1932, 99, 512-520.

Valenstein, E. S., Riss, W., and Young, W. C. Experimental and genetic factors in the organization of sexual behavior in male guinea pigs. Journal of Comparative and Physiological Psychology, 1955, 48, 397-403.

Villee, C. A. Some problems of the metabolism and mechanism of action of steroid sex hormones. In W. C. Young (Ed.), Sex and internal secretions. Vol. I (3rd ed.) Baltimore: Williams and Williams, 1961. Pp. 643-665.

Young, W. C. Sex and internal secretions. 2 Vols. (3rd ed.) Baltimore: Williams and Williams, 1961.