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I hereby recommend that the thesis prepared under my supervision by James Cecil Perry

entitled THE ANTAGONISTIC ACTION OF ADRENALIN ON THE REPRODUCTIVE CYCLE OF THE ENGLISH SPARROW, PASSER DOMESTICUS (LINNAEUS)

be accepted as fulfilling this part of the requirements for the degree of Doctor of Philosophy

Approved by:

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THE ANTAGONISTIC ACTION OF ADRENALIN ON THE REPRODUCTIVE CYCLE OF THE ENGLISH SPARROW, PASSER DOMESTICUS (LINNAEUS).

A dissertation submitted to the

Graduate School
of the University of Cincinnati

in partial fulfillment of the
requirements for the degree of

DOCTOR OF PHILOSOPHY

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by

James Cecil Perry

A. B. St. Louis University 1924
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CONTENTS

INTRODUCTION

Reproductive activity divides the vertebrate animals into two groups, continual breeders and seasonal breeders. The former group includes those that reproduce in cyclic fashion--those which breed throughout the year independently of seasonal or other environmental influence. The latter group, comprising by far the greater number of the vertebrate species, breed[5] at only one season of the year; usually the spring or summer. Among these is to be found the common house sparrow, Passer domesticus (Linnaeus). The reproductive mechanism of the continual breeders is explained by the reciprocal anterior pituitary-gonad relationship. Accordingly, the anterior pituitary stimulates the gonads by the formation of gonadotropic hormones. Under their influence mature germ cells are formed and the gonads, likewise, secrete their respective sex hormones. These sex hormones in turn stimulate the accessory reproductive organs and the secondary sex characteristics. Eventually the sex hormones inhibit the secretion of gonadotropic hormones by the anterior pituitary; thus the production of sex hormones by the gonads is in turn checked. But as the gonad check on the pituitary is removed, the gonadotropic hormones are again formed, the gonads are again stimulated, and the entire cycle is repeated cycle after cycle, interrupted only by an actual pregnancy or hatching period. To Moore and Price ('32) belong the credit for the elucidation of this recipro-

city between the anterior pituitary and the gonads.

In the annual or seasonal type of breeders the same reciprocal relationship exists, but at the conclusion of the cycle the gonads regress. No more germ cells mature until the following season. At this time the pituitary again secretes gonadotropic hormones and a cycle occurs.

Contrasting the continual and the seasonal cycles, we note that in the latter there is a long period of quiescence during which the pituitary does not stimulate the gonads. This quiescence is obviously not due to a check on the pituitary by the gonads themselves; for they are not secreting the inhibiting sex hormones, and as Wells ('35) reports, the hypophyses of male ground squirrels during quiescence do not contain stored gonadotropic hormones.

Investigators have long sought a cause of the resumption of the secretion of gonadotropic hormones by the pituitary and the consequent gonadal activity in seasonal breeders. During the past two decades certain factors in specific instances have been considered causes of this resumption. Among birds the increase in daylight hours has been considered such an inciting cause since the epochal experiments of Rowan ('25-'29). He showed that juncos and related birds exposed during the winter months to increased periods of daily illumination from electric lights after sundown came into the full spring breeding condition. Rowan ('38a), however, does not consider the increased light exposure to be the causal factor in avian gonadal development. Instead he maintains that the increased light causes increased wakefulness, and hence activity, which is the causal factor. Bissonnette ('30-'36)

Kirschbaum and Ringoen ('36), Benoit ('34-'37), and other workers in the field hold that light is the causal factor. It is important to remember that the cyclic forms exhibit cycle after cycle apparently independently of environmental changes. Thus, Whitaker ('38) has found that Eskimos are not sterile during the long arctic nights. Perry ('38), working on English sparrows, found that a diet of irradiated wheat caused gonadal development. Additional experimentation has usually confirmed the first results. The role of such a diet in stimulating gonadal development is now considered to be similar to light, that is, not as a primary factor, but a modifying factor. Witschi ('35) and Riley ('37), working with sparrows, conclude that there is an inherent breeding cycle coupled to the seasons through environmental factors. They indicate that this factor is to be sought in an inherent pituitary rhythm. As evidence, Riley adduces the fact that the hypophyses of adult males do not respond to light during the first half of the quiescent period. The closer the normal breeding season approaches, the more easily are they stimulated by light.

The idea has been prevalent for the past few years that the factors responsible for checking hormonal activity are certain anti-hormones. Thus, Collip and his co-workers ('35) consider that each hormone is kept in balance by an antagonistic or antihormonal substance. Thus prolonged administration of anterior pituitary gonadotropic preparations lead to a lowering of the stimulating effects of the animal's own pituitary. Gordon ('37) in collaboration with Charipper and Kleinberg relates the production of antihormones to the reticulo-endothelial system. Accordingly, the ovaries of splenectomized rats injected with

gonadotropic hormones continued to increase in weight, whereas those of controls, after an initial increase, lost weight. The implication is that the spleen liberates an antagonistic principle into the blood which acts as an antigonadotropic principle.

Thus in explaining the resumption of anterior pituitary gonadal relationship in seasonal forms we are brought to the position of considering certain environmental factors, such as increased light in birds as causal. Or, an alternate explanation is found in a more internally acting mechanism; certain antihormonal, preferably antagonistic principles, hold the secretion of gonadotropic hormones in abeyance for long periods of quiescence. As the natural breeding season returns these antagonists cease to act, and a cycle occurs. That these antagonists do not arise in the gonads, or in the internal rhythm of the pituitary itself is indicated by the existence of the reticulo-endothelial antihormones. In the following discussion evidence of the existence of another antagonist of the anterior pituitary gonadal mechanism will be presented. This antagonist is neither a gonadal antihormone nor an antihormone of the reticulo-endothelial system, but the hormone of the adrenal medulla, adrenalin. Its method of action affords not only an explanation of the annual reproductive cycles, but also may explain the mechanism of the continual cycles.

The two explanations of the annual cycles need not be exclusive. Increased light exposure of sufficient duration is indisputably followed by gonadal development. In the case of the antagonistic view, light and other possible environmental agencies are necessary to release the pituitary from the inhibition of the antagonist.

The writer wishes to express his grateful appreciation to Dr. Harry L. Wieman, Director of the Zoology Department of the University of Cincinnati, for making it possible to carry out the experimental work reported in this paper at the writer's convenience. To Dr. Wieman gratitude is also extended for his helpful and encouraging criticism. Especially is the writer indebted to Dr. Charles K. Weichert, Associate Professor of Zoology of the University of Cincinnati, for his patient and untiring direction of the experimental work involved. Thanks is also expressed to Dr. Weichert for his valuable suggestions in preparing the manuscript. The writer is further indebted and renders thanks to Dr. Max Gilbert of the Schering Corporation, Bloomfield, New Jersey, for a generous supply of Oreton (Testosterone Proprionate), Grateful acknowledgment is also made to Professor John I. Malone, formerly of the University of Detroit, for contributing very generous amounts of Antuitrin (Parke Davis, anterior pituitary gonadotropic hormone). The Adrenalin chloride used in the experiment was also a Parke Davis product, but was purchased. Gratitude is also expressed to Mr. James T. Clear of Cincinnati for running several of the metabolic rate tests reported in this paper; to Mr. William Gessing of Cincinnati for his aid in making the photographic reproductions; to Mr. Ray Fellinger of Cincinnati for typing the completed manuscript.

REVIEW OF LITERATURE

The literature treating of the reproductive activity of birds is voluminous. Excellent reviews of the subject are those of Marshall ('32), Bissonnette ('36), and Rowan ('38). Schafer ('07) first clearly formulated the theory that light was the stimulating factor in the sexual cycle of birds. He claimed that the decreasing hours of daylight were responsible for migration from north to south in the fall. Increase of light in the spring caused the northward migration.

Rowan ('25-'29) established the effect of light on the gonads of juncos. He exposed these birds to increased amounts of daily light in outdoor cages from October to January. At Edmonton, Alberta, Canada, where the experiments were performed, a minimum of 52^{0} below zero was encountered; but in spite of the low temperature the testes showed complete spermatogenic activity and the birds were singing. Temperature was ruled out, as juncos examined in California and the Edmonton controls exhibited neither condition. At this period and later, Rowan ('37) stated and gave evidence that light itself was not the cause of gonadal recrudescence in juncos. He obtained gonad development by keeping the birds in a state of wakefulness mechanically. This was accomplished by rotating their cages for increasing periods after sundown until they were receiving 4 hours of enforced wakefulness in addition to the 9 hours of daylight. He holds, therefore, that it is the greater activity brought about by increased light exposure that causes gonadal development.

Rowan, likewise, found at the times of the above experiments that diminution of light from a long length of day to a short length brought about regression of the gonads of juncos to the quiescent state. Identical results were obtained in other birds; finches, canaries, and crows. Bissonnette, working at Hartford, Connecticut ('30, '31) and using the starling as his experimental subject, found similar light effects on the gonads; recrudescence with increased light exposure, regression with decreased light exposure. He considers light to be the primary cause of gonadal stimulation and activity as a modifying factor. Bissonnette ('32) reports that red light stimulates testis development and green light inhibits it. Bissonnette ('36) and Clark, Leonard, and Bump ('36) independently report that increased light given to pheasants and quail during January caused them to lay fertile eggs in March when outside controls were not yet laying.

Kirschbaum and Ringoen ('36), working with sparrows during the winter months, demonstrated that increased light exposure of 6 $\frac{1}{2}$ hours for 6 weeks brings the males to a breeding condition. Ringoen and Kirschbaum ('39) report that a longer time, 90 days, is necessary to bring about the same condition in the females. They also find that the black beak, discussed below, is a characteristic of the developed male. They confirm the regressive effects of diminished light in this species. The writer has confirmed these results in his own investigations. However, spermatogenesis may occur after 35 days of such exposure. The same investigators find ('36) that decreased hours of light exposure administered to birds with developed gonads causes regression. The writer has found this to be the case in both males and females given an 8

hour light day at the height of the breeding season (April 15 to May 15). Witschi ('36) reports a testis activity indicator in sparrows. The beak, which is light colored in juvenile males, becomes jet black as the testis hormone is secreted by the testis. The same result follows the injection of the hormone into castrate males. Keck ('34) described the change in bill coloration at the onset of the breeding season from a drab gray or horny color to jet black. The beak remains permanently light in castrates. It is normal for the beaks of females to be light at all times. The writer has found that injections of 0.1 c.c. of Oreton (testosterone proprionate, Schering) given intramuscularly to females on alternate days will produce the jet black beaks after 6 to 9 injections. If the injections are stopped and the birds allowed to continue their normal life, the black coloration disappears in 6 to 7 weeks. Loss of black beak color also occurs in males subjected to 8-hour light days. Likewise, the male juvenile sparrow develops a black beak as a result of injected Antuitrin (Parke-Davis, anterior pituitary gonadotropic hormone). Hence the writer has confirmed and extended the beak-testis hormone-indicating-reaction. The changes in beak coloration have been used as reliable and trustworthy indicators of the states of pituitary gonadal conditions in the experiments considered in this paper. Benoit ('34-'37), in a series of experiments using Rouen ducks as subjects, confirms the effects of light on the gonads. He considers light as the stimulating factor, although he has not proved this to be the case according to Rowan ('38).

The manner in which light exercises its effects on the bird reproductive mechanism has been the subject of lengthy investigations.

Benoit ('34-'37) in the above cited series of experiments finds that ducks covered with light-proof cloth, except for the eyes, undergo pronounced testes development. He concludes that light acts on the pituitary by way of the eyes and optic tract. He next extirpated the eyes and found that such birds illuminated at night without any covering had developed testes. This development occurred also in birds with optic nerves severed and treated in like manner. Moreover in later work ('37) exposure of the pituitary to light through the thin wall of the orbit gave similar results. He infers the existence of a receptor in the head region, not necessarily a component of the optic tract which is concerned with pituitary stimulation. Ivanova ('35), working with sparrows, found testes development upon light exposure when the entire head was covered with light-proof material. This would indicate that the light acted through the entire body surface. Kirschbaum and Ringoen ('38), and indeed most investigators disagree. They fail, generally, to obtain development in such birds. They do obtain it if the eyes are left uncovered during light exposure even though the rest of the bird be covered with light-proof cloth. Rowan ('38), evaluating these contradictory results, holds that in the experiments of Benoit, Ivanova, and Kirschbaum and Ringoen, it is not the light that is stimulating the reproductive mechanism. He considers that the experiments tend to show that it is the increased wakefulness, and therefore physiological activity, that is the stimulating factor. He further supports his conclusions with the observations he has made on London Starlings. Rowan ('38a) states that these birds enter into the breeding season earlier than country starlings. He considers that the disturb-

ance of the city night life keeps the birds awake for longer periods, and as the light is too dim to affect them noticeably it is the increased activity that stimulates them earlier than the country starlings.

Concerning the specific investigation of this paper, the effects of adrenalin on the reproductive system of birds, there is a lack of literature. These experiments, as far as the writer can learn, are the first of their kind. The effects generally attributed to adrenalin that may have a bearing on the problem at hand are summarized from Best and Taylor ('39). The effects of subcutaneous injections of adrenalin, and presumably intramuscular injections, are less pronounced than intravenous injections. The arterioles of the brain are passively dilated by the general rise in blood pressure. Presumably the same is true for the anterior pituitary. Injected adrenalin causes hyperglycemia and glycosuria due to a mobilization of liver glycogen and a temporary loss from that organ ensues. The muscles and other tissues also lose glycogen. This glycogen is probably used to restore the liver glycogen by reconversion of the lactic acid, formed from the breakdown of muscle glycogen, into glycogen. Moreover, this reaction is reversible as follows:- Muscle Glycogen - Blood Lactic Acid - Liver Glycogen - Blood Glucose - Muscle Glycogen. In relatively large doses subcutaneous injections of adrenalin exert a dilating effect on the arterioles of the muscles, skeletal and cardiac. Likewise the vessels of the intestine usually dilate. Blood is moved into these structures, and due to a peripheral vaso-constriction is withdrawn from the vessels of the splanchnic areas, mucous membranes, and skin capillaries. The dilata-

tion of the vessels of the skeletal muscles would, therefore, be expected to occur after the injections described below. This result is of significance in the possible interpretation of the experimental results to be described. The question whether adrenalin is continuously discharged into the blood from the adrenal medulla is not settled. see p. 33 Rogoff ('35) is credited with estimating the concentration of adrenalin in the blood of a resting animal to range from 1 : 2,000,000,000 to 1 : 1,000,000,000. Cannon and Rapport ('11) found that in states of stress, the output of adrenalin amounted to 0.003 to 0.004 mg. per kilogram body wt. per minute. Probably in conditions of increased muscular activity there is an intermediate concentration of adrenalin secreted into the blood by the adrenal medulla. The secretion of adrenalin is under nervous control. The gland may be stimulated to exhaust its adrenalin content by stimulating the great splanchnic nerve. The center for control of adrenal secretion is stated by Cannon and Rapport ('21) to be in the upper part of the floor of the fourth ventricle. The hypothalamus is probably the highest center, however, for stimulation of this region will cause a discharge of adrenalin. This fact would seem to relate the pituitary and adrenal medulla very closely in a nervous as well as a n endocrine relationship. The theory of the emergency of adrenal function does not seem to bear on the normal function of reproduction. In a state of emergency in the human subject oxygen consumption and carbon dioxide production are increased. The respiratory quotient is raised. These effects are short lived. The calorogenic effect of adrenalin is correlated with the hyperglycemic effect.

MATERIALS AND METHODS

Sparrows were procured by trapping them in the Glenhaven-Standby type of trap. They were brought to the laboratory and kept in cages of 6 cubic feet capacity. Not more than 12 birds were alloted to a cubicle. Birds used in the experiments were transferred to experimental cages after they had become accustomed to captivity. All birds used in the experiments were juveniles with the exception of some of the birds used in the last series. Light stimulated controls were exposed to increased illumination as follows. Cubicles of 6 cubic feet capacity were made as light proof as possible with heavy black roofing paper. They were wired so that light in all the cubicles could be controlled at a main switch. During the actual periods of injection all experimental birds and their appropriate controls were kept in smaller cages, 12" x 8" x 8", two animals to a cage. Both experimental and control birds were supplied daily with plentiful amounts of scratch feed, consisting of cracked corn, oats, barley, wheat, and buckwheat. They were always supplied with fresh water and bird gravel.

The mortality of experimental birds was low and it did not greatly exceed that of control and supply birds. The weights of all birds were taken at the beginning of experimentation and at the time of sacrificing. At the time of sacrificing photographs were taken of the beaks of representative birds, both control and experimental. The posterior dorsal trunk region, containing the entire reproductive tract, was excised; fixed in Bouin's fluid, and run up to 70 % alcohol. Photographs of the trunk regions of representative birds were then taken. The gonads were then dissected free; the diameters measured;

the weights determined, and in the case of the left testes the volumes were computed. Computation of volume is necessary for the smaller testes as they are too small to be measured by methods involving displacement of fluid. Since the testes are ellipsoids the volume is obtained from the formula, $V = 4/3\pi ab^2$, where a is $\frac{1}{2}$ the long semi axis and b is $\frac{1}{2}$ the short semi axis. The volumes of the ovaries cannot thus be computed as their shapes are irregular; displacement methods are not generally practicable. However, the measurements of the long and short diameters are indicative of the volumes of the ovaries.

Histological study was made of representative and contrasting gonads, ducts, and adrenal glands. Where very similar macroscopic results were obtained, microscopic study was not made. Delafield's hematoxylin and eosin were the stains employed. Sections were cut at 10 micra. Histological preparations were also made of the livers and muscles of certain control and experimental birds. Fixation of such tissues for glycogen content was employed as follows. The tissues were taken from the living anesthetized birds in order to avoid the loss of glycogen. They were fixed in a solution consisting of 9 parts of absolute alcohol saturated with picric acid to 1 part of neutral formalin. They were stained according to Best's Carmine method. Control slides were made by allowing ptyalin to digest out possible contained glycogen. Sections of liver and muscle were also cut at 10 micra. Photomicrographs were taken of significant histological results.

EXPERIMENTAL PROCEDURE AND RESULTS

To test the action of adrenalin on the reproductive cycle 6 different series of experiments were carried out from December 1, 1939 to April 27, 1940.

Series 1. A group of 6 birds was taken from the supply cages, weighed and confined to the small experimental cages on December 1, 1939. These birds received the normal amount of daylight at the time of the experiment. Spacious windows facing south and east afforded abundant light all day. Daily injections of adrenalin chloride (Parke Davis) were commenced December 7. The dosage was 0.1 c.c. of 1 : 1,000, solution diluted 10 times. The injections were given intramuscularly in the breast muscles. Since these birds were in the quiescent stage of the cycle, the control birds were the untreated winter birds taken during the winter months. The purpose of this experiment was to determine the effects of adrenalin on the reproductive system of juvenile sparrows. As any change in this system would be expected in 2 or 3 weeks the birds were sacrificed at periods of 15 and 24 days after the start of injections. The results are summarized in Table 1. An examination of this table might seem to indicate that adrenalin injections had brought about a considerable reduction in the size, weight, and volume of the gonads. Possibly this was true to some extent. However, the averages given in Table 1 are for birds taken at intervals during the winter months. During December the gonads are approximately the same size, weight, and volume as that found for the experimental birds of this series. Only a slight regressive effect was indicated.

Table 1.
Series 1. Normal Light
Quiescent Winter Birds; Adrenalin Injected.
Dec.7 to Dec.31.

A. Males.

Bird.	Body Weights gms. Start	End	Diff.	Injs. 0.1cc.	Gonad Diameters mm. Right	Left	Gonad Wts. gms. Right	Left	Volume L.Test. cu.mm.	% Loss Wt.	Vol
Cont'l. 12 Avg. Winter. Expm'l		25.2		00	1.80x1.48	1.86x1.50	0.0021	0.0023	2.17		
T100	27.5	26.1	1.4	15	1.10x0.90	1.20x1.00	0.0016	0.0018	0.629	22	70
T102	26.7	25.3	1.4	16	1.00x0.90	1.10x1.00	0.0012	0.0014	0.578	41	73
T104	28.8	26.7	2.1	24	0.90x0.90	1.00x0.90	0.0013	0.0015	0.427	35	80
T106	29.5	27.0	2.5	24	1.50x1.40	1.60x1.50	0.0020	0.0021	1.833	13	15
Avg.	28.1	26.3	1.8	20	1.12x1.02	1.30x1.10	0.0015	0.0017	0.866	26	60

B. Females.

Cont'l 12 Avg. Winter Expm'l.		24.3		00		4.80x3.20		0.0129			
T101	25.1	24.4	0.7	24		3.00x1.90		0.0080		34	
T103	27.0	23.2	3.8	24		4.30x1,90		0.0126		2.3	
Avg.	26.1	23.8	2.3	24		3.70x1.90		0.0103		20	

Series 2. Concurrently with series 1, a second series of birds was injected with adrenalin in the same manner and with the same dosage. In addition to the injections of adrenalin, however, this series of birds was exposed to artificial illumination from a 50 watt Mazda bulb hanging 12 inches above the cages. Exposure was from 6 p. m. until 9 a. m., making a total of 15 hours. The small cages containing the experimental and control birds were placed inside the larger cages used to subject birds to increased illumination as described in the last section. The birds used as controls for this series were light stimulated for 40 days before sacrificing. The experimental birds were light stimulated for 31 days only, and injections were given only during the last 24 days (Dec., 7 to Dec., 31). Hence the control and experimental birds are not strictly comparable. It seems evident, however, from a consideration of the results given in " Table 2 " that the adrenalin had exerted an inhibitory effect on the development of the gonads. Moreover both beaks of the male controls had become black after 31 days of the added illumination; whereas the dorsal beaks and the tips and margins only of the ventral beaks of the experimental males were black. The remaining portions of the ventral beaks were still light in color. Microscopic examination likewise indicated that gonad development had been inhibited. Development of the reproductive system had, however, occured. It appeared, therefore, that while exerting an inhibiting effect the adrenalin dosage that was employed in this series had not been sufficiently great. New experiments, more accurate and controlled, were therefore undertaken.

Table 2.
Series 2. Daily Light 15 Hours.
Quiescent Winter Birds - Adrenalin Injected. Dec.7 - Dec.31.

A. Males

Bird.	Body Weights gms. Start	End	Diff.	Injs. 0.1 cc.	Gonad Diameters mm. Right	Left	Gonad Wts. Gms. Right	Left	Volume L.Test. cu.mm.	% Loss Wt.	Vol
Cont'ls. 40 days Light.	27.2	25.1	2.1	00.0	6.6x4.9	7.1x5.4	0.0772	0.1285	108.44		
Expm'l.											
S102	25.6	23.2	2.4	24	3.7x3.5	4.4x3.9	0.0254	0.0300	35.13	77	68
S104	27.9	25.1	2.8	24	4.4x3.7	4.4x3.7	0.0292	0.0320	31.43	76	76
Avg.	26.7	24.1	2.5	24	4.1x3.6	4.4x3.8	0.0273	0.0310	33.28	76	69

B. Females

Cont'ls. 40 days Light.	25.7	23.2	2.5	00		5.5x4.1		0.0264			
Expm'l.											
S103	24.8	22.8	2.0	24		5.9x3.2		0.0106		59	
S105	26.0	24.1	1.9	24		6.1x3.9		0.0102		76	
Avg.	25.4	23.5	1.9	24		6.0x3.6		0.0104		60	

Series 3. Birds were exposed to artificial light for 15 hours a day in the large 6 cubic feet capacity cages. Exposure time was again from 6 p. m. to 9 a. m. daily. From 9 a. m. to 6 p. m. the birds were in total darkness since the cubicles were kept in a photographic dark room. After 40 days of added daily light exposure the beaks of the males were black and the testes were in complete, or nearly complete, stages of spermatogenesis. The females were not comparably advanced, but their ovaries and oviducts were developed considerably beyond those of quiescent females. Such birds were now placed in the smaller type cages, again 2 to a cage. Like birds to serve as controls were similarly caged. The results of series 2 had indicated that a stronger dosage of adrenalin was advisable. A dosage of $\frac{1}{2}$ minim of 1 : 1,000 given daily was employed in this series. Immediately after the injections they were placed near a radiator. It was found that they overcame the immediate effects of the adrenalin much better if kept warm for a few minutes. As the injections and care required about 30 minutes, the nightly exposure was diminished $\frac{1}{2}$ hour. After the injections the birds were placed inside the dark cubicles and left undisturbed until the light was turned on. The daily injections were continued until the ventral beaks of the males had lost practically all their black coloration. This was usually 15 to 20 days after the start of the injections. At this time the birds were sacrificed together with their appropriate controls. " Table 3" presents the macroscopic results of this experiment in graphic form.

Table 3.
Series 3. Daily Light 15 Hours.
60 Day Light Stimulated Birds.
Adrenalin Injected 40th to 60th Days.

A. Males

Bird.	Body Weights gms. Start	End	Diff.	Injs. ½Min.	Gonad Diameters mm. Right	Left	Gonad Wts. gms Right	Left	Volume L.Test. cu.mm.	% Loss Wt.	Vol.
Cont'l. 60 Days Light. Expm'l.	27.6	25.7	1.9	00	6.9x5.5	8.0x6.3	0.1271	0.1613	166.26		
S112	21.6	19.8	1.8	19	1.6x1.0	1.7x1.0	0.0012	0.0016	0.89	99	99
S114	21.9	18.7	3.2	15	2.5x1.9	3.8x2.1	0.0038	0.0046	8.78	97	95
S116	22.1	20.4	1.7	20	2.7x2.1	3.5x2.2	0.0054	0.0066	8.88	96	94
S118	19.6	18.5	1.1	15	1.8x1.2	1.9x1.4	0.0012	0.0014	1.95	99	98
S120	21.9	20.7	1.2	16	2.1x1.8	2.3x1.9	0.0022	0.0022	4.36	98	97
S122	19.4	17.9	1.5	16	2.2x1.6	2.4x1.8	0.0026	0.0030	4.06	98	97
Avg.	21.1	19.3	1.8	17	2.0x1.5	2.6x1.7	0.0027	0.0030	4.83	98	97

B. Females

Cont'l. 60 Days Light. Expm'l.	26.0	23.2	2.8	00		8.3x4.9		0.0411			
S111	19.2	18.6	0.6	19		5.0x2.1		0.0048		88	
S113	21.5	19.9	1.6	19		6.0x3.0		0.0122		70	
S115	19.7	18.1	1.6	21		6.0x3.0		0.0056		86	
S117	23.5	21.2	2.3	15		4.2x2.8		0.0018		96	
S119	22.3	21.9	0.4	14		4.7x2.7		0.0168		59	
S121	23.6	22.3	1.3	16		5.0x2.1		0.0150		64	
Avg.	21.6	20.3	1.3	17		5.2x2.6		0.0095		77	

A comparison of the experimental males, S 112 to S 122, with the 60 day light stimulated controls brings out some remarkable differences. These birds have an average left testis volume of 4.83 cubic millimeters contrasted with 166.26 cubic millimeters for the 60 day controls, a loss of 161.43 cubic millimeters or 97 % . Likewise the average weight loss of the left testis is 0.1583 gms., or 98 % . A comparison of the average testis volume and weight losses of these experimental birds with the average testis volume and weight of the 40 day light stimulated birds, " Table 2 ", reveals a volume loss of 103.61 cu. mm., or 96 % . Like - wise the average weight loss is 0.1255 gms., or 97 % . Compared to the normal quiescent males the volumes and weights of the testes of the experimental males are of the same order, but slightly in excess. (Cf. Table 1). Inspection of females,S 111 to S 121, shows that similar regressive changes have occured in the ovaries. It is to be noted, how- ever that females do not respond to light stimulation as quickly as do males of this species. Hence the differences in diameters between 40 day light stimulated and experimental females are not as striking as in the males. The ovaries of the experimental birds,when compared to those of 60 day light stimulated controls, show an average loss of 3.1 x 2.3 millimeters. The average loss of ovarian weight in the series is 0.0316 gms., or 76 %, when compared to the average weight of the 60 day light stimulated controls. Again,the experimental ovaries are diminished in weight to an extent even below the average of the quiescent females. The average size of such quiescent ovaries is slightly less than the average size of the experimental ovaries of this series.

Striking as are the weight and volume losses of these experimental birds, and striking as the macroscopic differences are seen to be; still more remarkable are the microscopic changes. Contrasting the testes of the experimental birds with those of 40 and 60 day light stimulated controls we find that instead of complete spermatogenesis having occured, as would be expected from the light stimulation, a pronounced regression has occured. The experimental testes approach quite closely the condition found in the testes of quiescent winter birds. Actually we find only spermatogonia in the testes of the adrenalin injected birds. The degree of regression can best be observed by comparing fig. 2 plate III with figs. 3 and 4 plate III. The close approximation to the quiescent condition is manifest from a comparison of fig.2 with fig. 1 of plate III.

A similar regression is evident in the ovaries pictured on plate IV. The follicles in fig. 2, the experimental ovary, are more nearly the size of those in fig. 1, the quiescent ovary. They are much smaller than the follicles of the two control ovaries in figs. 3 and 4. The total size reduction in the experimental ovary is evident since the sections pictured in all instances are approximately median. Moreover, the follicles of the control ovaries are less compact and a more nearly approaching the surface position preliminary to ovulation.

An examination of the reproductive ducts illustrates the regression that has occured in the accessory reproductive organs. Sections of the male ducts are through the spermatic glomus region. Single tubules of a 40 and of a 60 day light stimulated control are shown in figs. 3 and 4 plate V. Obviously each is characterized by a large lumen and a high type of secretory epithelium. Spermatozoa are not

found in any number in such tubules of 40 day birds; they are abundant in the tubules of 60 day birds. Two tubules of the glomus region of an experimental male are pictured in fig. 2 plate V. Remarkably different, they reveal only a small lumen, a low flat non-secretory epithelium, and a complete absence of spermatozoa. In these characteristics they agree with the tubules of the quiescent bird seen in fig. 1 plate V.

The oviducts pictured on plate VI illustrate the regressive effects on the female accessories. The duct of the 40 day light stimulated bird shows the beginnings of crypt and gland formation in the duct walls. The duct of the 60 day light stimulated bird shows extensive crypt formation and glandular development. The epithelium is becoming secretory, the lumen has greatly increased. In the duct seen in fig. 2, taken from bird S 111, these progressive changes have almost completely disappeared. The lack of pronounced crypts, the low flat non-secretory epithelium, and the narrow lumen show that it is similar to the condition found in the normally quiescen t oviduct, fig. 1.

In conformity with the changes in the gonads and accessory organs marked changes occur in the beaks of the males. Thus the secondary sex characteristics likewise share in the regressive effects produced by the adrenalin injections. Fig. 1 plate I shows these changes in contrast to the controls. From left to right the beaks are those of a 60 day light stimulated control, a 40 day light stimulated control, and that of a typical experimental male. The black coloration has practically disappeared from the experimental bird beak. It had been just as black as the control beaks at the start of the adrenalin injections. The loss of the black coloration is a definite sign of testis hormone failure and

Table 2.
Series 2. Daily Light 15 Hours.
Quiescent Winter Birds; Adrenalin Injected. Dec. 7 - Dec.31.

A. Males.

Bird.	Body Weights gms. Start	End	Diff.	Inje. c.c.	Gonad Diameters m.m. Right	Left	Gonad Wts. gms. Right	Left	Volume c.m.	% Loss Wt.	% Loss Vol.
Cont'l. 40 Days											
Light.	27.2	25.1	2.1	00	6.6x4.0	7.1x5.-	0.0778	0.1225	108.44		
Cont'l.											
3102	25.6	23.2	2.4	24	3.7x3.5	4.4x3.0	0.0254	0.0300	25.13	77	68
3104	27.3	25.1	2.2	24	4.4x3.7	4.4x3.7	0.0288	0.0320	31.43	73	73
Avg.	26.7	24.1	2.3	24	4.1x3.6	4.4x3.3	0.0273	0.0310	28.28	73	69

B. Females.

Cont'l. 40 Days											
Light.	27.7	25.7	2.0	00		5.5x4.1		0.0274			
Cont'l.											
3103	24.3	22.3	2.0	24		5.0x3.2		0.0106		80	
3105	26.0	24.1	1.9	24		6.1x3.0		0.0102		76	
Avg.	25.1	23.5	1.9	24		6.0x3.6		0.0104		60	

As far as can be determined, the glycogen content of the liver and muscles of adrenalin injected birds does not differ radically from controls. The adrenal glands of experimental and control birds do not show any marked histological differences. The weights of the adrenal glands are practically the same for all kinds of birds studied. Thus the average adrenal gland weights for males and females were found to be as follows; for quiescent birds 0.00125 gms.; for 40 day light stimulated birds 0.0011 gms.; for 60 day light stimulated birds 0.0011 gms.; for adrenalin injected birds of this series 0.00117 gms. Measurements of the adrenal gland diameters were practically the same for the 4 classes of birds - 2.40 x 1.55 mm. The data presented in the various tables show that the total average weight loss of the experimental birds is about the same as that found in the case of the various controls. A local destruction of muscle tissue is encountered at the place of the injections in some instances, due no doubt to local irritation. Surrounding muscle is, however, normal and healthy. As the metabolic rate of the various types of birds investigated was not found to be appreciably different, the regressive changes occuring in the reproductive system are presumably not due to changes in the metabolic rate. Metabolic rate readings were taken by using the Haldane method whereby the amount of water and carbon dioxide produced by the animal are directly determined by weight, and the oxgen consumed is determined by the difference of this combined water and carbon dioxide weight and the weight lost by the animal during the time of running the determination.

The following table represents an average of 3 determinations on each bird reported. The respiratory quotient was determined according to the following formula -

$$R.\ Q. = \frac{\text{weight } O_2 \text{ (in } CO_2 \text{) in expired air}}{\text{weight } O_2 \text{ consumed}}$$

Table 4.
Metabolic Rate Determinations.

A. Quiescent Winter Birds.

Bird	Sex	H_2O produced gm./ kg./ hr.	CO_2 produced gm./ kg./ hr.	O_2 consumed gm./ kg./ hr.	R. Q.
N101	Female	9.8510	8.8830	6.6950	.965
N103	"	7.8031	9.5152	7.4656	.927
N104	Male	3.0909	8.2141	8.6004	.741
N106	"	4.8777	7.5151	6.3589	.859
Avg.		6.9057	8.5318	7.2800	.873

B. Light Stimulated (40 - 60 days).

L101	Female	5.3239	7.1016	6.4519	.800
L103	"	6.0066	7.7961	6.1700	.919
L104	Male	4.3177	6.6906	6.7744	.718
L106	"	4.0578	7.3615	6.3650	.841
Avg.		4.9240	7.2377	6.4403	.820

C. Light Stimulated (40 to 60 days) Adrenalin Injected.

S111	Female	3.3868	7.8183	7.1275	.797
S113	"	4.5579	9.1159	7.3653	.900
S112	Male	5.5209	9.8389	8.3890	.853
S116	"	4.6482	9.9534	8.2418	.878
Avg.		4.5284	9.1816	7.7809	.857

All readings were taken under the conditions in the laboratory at the time of running. They were taken between the hours of 1 p. m. and 5 p. m. The considerable variations in individual readings are probably due to changes in laboratory conditions. Despite individual

changes the average respiratory quotients covering considerable periods of time, which the readings represent, are very close for the three kinds of subjects.

In order to determine, if possible, the mode of antagonism of adrenalin on the reproductive system another series of birds was injected with antuitrin.

Series 4. Birds were first given daily injections of antutrin in order to bring them into the breeding condition. Juvenile birds were injected with 0.2 c.c. intramuscularly, again in the pectoral muscles. These injections were carried out during February 1940. When the beaks of the males in this series had become uniformally black, injections of adrenalin were commenced. The dosage used was again $\frac{1}{2}$ minim. The antuitrin injections were, however, continued. It was not deemed expedient to give injections of both hormones on the same day; they were, therefore given on alternate days. The birds of this series were confined in the small type experimental cages and received only the normal number of daylight hours. The 2 birds that received only 9 injections of adrenalin died due to accident. With the exception of the male J 104 all of the experimental birds of this series were found to have regressed almost to the normal condition of resting winter birds. The antagonistic action of adrenalin on the reproductive system of the birds of this series will be evident from an inspection of " Table 5". Only 1 male and 1 female control is given in the table as the writer had frequently determined the efficacy of antuitrin in this species.
As a further check on the antagonism of adrenalin for injected antuitrin effects another series of birds was treated with the 2 hormones.

Series 5. Quiescent juvenile males were confined in the small experimental cages, given the normal daylight exposure, and injected on alternate days with antuitrin and adrenalin. The dosages were as in series 4. They were sacrificed after they had received sufficient antuitrin to cause the blackening of the beaks if it had been injected alone. Contrasting the gonadal condition of these birds with the condition found in average resting winter males it will be seen from an inspection of " Table 6 " that the weights and volumes are slightly in excess of the average. This is to be expected as the gonads of sparrows begin to enlarge by the end of February normally. Although only 2 birds are reported for this series it can be safely concluded that adrenalin will inhibit the action of antuitrin on quiescent birds in view of the results of series 4.

Series 6. As a final step in testing the antagonism of adrenalin on the reproductive system of sparrows a group of birds was given daily injections of adrenalin at the time of the natural breeding season. Birds trapped during the first week of April were confined in experimental cages, exposed to the normal number of daylight hours, and injected with adrenalin as in series 3. Controls were similarly caged, but did not receive the adrenalin injections. The results appear graphically in "Table 7 ". With the exception of the male P 102 all birds in this series underwent a marked regression. Even in the case of P 102 the regression is evident, especially in the weight.

Table 5.
Series 4. Normal Daylight, February.
Antuitrin Injected Birds; 0.2 c.c. Feb.,1 -Feb.,16.
Antuitrin and Adrenalin Injected; Alternate Days to
Time of Sacrificing (Early March).

A.Males.

Bird.	Body Weights gms. Start	End	Diff.	Injs. Ant. c.c.	Adr. ½Min.	Gonad Diameters mm. Right	Left	Gonad Wts. gms. Right	Left	Volume L.Test cu.mm.	% Loss Wt.	Vol
Cont'l.												
J100	26.2	23.4	2.8	6.8	00	6.5x5.0	7.3x5.2	0.0860	0.1070	103.37		
Expm'l.												
J102	26.5	24.6	1.9	6.8	18	2.3x1.8	2.7x1.9	0.0045	0.0048	5.07	95	95
J104	21.8	20.5	1.3	6.0	14	3.8x2.8	3.9x3.3	0.0078	0.0115	17.23	89	85
J106	22.4	21.7	0.7	4.2	9	3.4x3.2	3.9x2.8	0.0188	0.0188	15.17	82	81
J108	23.9	22.5	1.4	4.2	9	3.0x2.8	3.2x2.8	0.0142	0.0146	13.16	85	87
J110	19.5	17.6	1.9	5.4	15	1.8x1.6	1.9x1.5	0.0022	0.0024	2.24	97	97
J112	21.8	19.4	2.4	5.6	16	2.1x1.7	2.2x1.9	0.0032	0.0032	4.14	97	96
Avg.	22.7	21.0	1.7	5.4	14	2.7x2.3	3.0x2.3	0.0085	0.0092	9.50	91	91

B. Females.

Cont'l												
J101	22.4	19.8	2.6	6.8	18		9.2x7.5		0.0722			
Expm'l.												
J103	20.6	18.7	1.9	6.8	18		5.4x3.6		0.0124		83	
J105	22.7	20.3	2.4	5.2	10		5.0x3.8		0.0118		84	
Avg.	21.9	19.6	2.6	5.5	14		5.2x3.7		0.0121		83.5	

Table 6.

Series 5. Normal Daylight, February.
Antuitrin and Adrenalin Injected Birds Contrasted with Quiescent Birds. Injections on Alternate Days;
Feb. 17 to March 3.

Males.

Bird.	Body Weights gms. Start	End	Diff.	Injs. Ant. c.c.	Adr. ½Min.	Gonad Diameters mm. Right	Left	Gonad Wts. gms. Right	Left	Volume L.Test. cu.mm.	% Diff. Wt.	Vol
Cont'l. 12 Avg. Winter.		25.2		00.	00.	1.80x1.48	1.86x1.50	0.0021	0.0023	2.17		
Expm'l.												
J200	22.5	20.2	2.3	3.0	15	1.90x1.30	1.90x1.60	0.0026	0.0030	2.55	+23	+15
J202	21.4	18.5	2.9	3.2	15	1.80x1.50	2.30x1.50	0.0024	0.0026	2.71	+12	+20
Avg.	21.9	19.4	2.6	3.1	15	1.85x1.40	2.10x1.55	0.0025	0.0028	2.63	+18	+17

Table 7.
Series 6. Normal Daylight, April.
Birds at Height of Breeding Cycle; Adrenalin Injected.

A. Males

Bird.	Body Weight gms. Start	End	Diff.	Injs. ½Min.	Gonad Diameters mm. Right	Left	Gonad Wts. gms. Right	Left	Volume L.Test cu.mm	% Loss Wt.	Vol.
Cont'l. 4 April.	25.8	24.1	1.7		6.5x5.4	7.9x5.6	0.2468	0.2587	129.76		
Expm'l.											
P100	25.8	22.8	3.0	22	1.7x1.4	2.2x1.5	0.0028	0.0032	2.59	98	98
P102	28.9	25.5	3.4	20	3.9x3.5	4.7x3.6	0.0278	0.0296	31.88	88	75
P104	26.5	24.8	1.7	22	2.6x1.7	2.8x1.9	0.0052	0.0056	5.30	96	95
P106	25.5	21.9	3.6	18	2.5x1.4	2.8x1.8	0.0046	0.0048	4.73	97	96
P108	23.8	20.3	3.5	18	2.4x1.6	2.7x1.8	0.0048	0.0056	4.58	96	97
Avg.	26.1	23.1	3.0	20	2.6x1.9	3.1x2.1	0.0090	0.0098	9.82	95	92

B. Females

Cont'l. 4 April.	26.3	23.2	3.1			7.5x5.2		0.0620			
Expm'l.											
P101	24.1	20.4	3.7	22		5.4x2.6		0.0122		80	
P103	21.7	17.4	4.2	20		4.6x2.9		0.0078		87	
P105	24.6	20.5	4.0	18		5.1x3.2		0.0084		86	
P107	22.1	18.6	3.5	15		4.8x2.7		0.0064		90	
P109	25.9	22.6	3.3	18		4.4x2.5		0.0058		90	
Avg.	23.7	19.9	3.8	19		4.9x2.8		0.0081		87	

DISCUSSION.

From the results discussed in the preceding section the antagonistic action of adrenalin on the anterior-pituitary-gonad mechanism stands out indubitably. This antagonism is manifest in the cessation of gonadal activity; first in the checking of gametogenesis and the regression from the complete breeding condition of the germ cells to the resting, out-of-cycle condition; second, in the checking of sex hormone secretion as evidenced by the inhibiting effect on the accessory organs and secondary sex characteristics. Since gonad development is caused directly by the anterior pituitary gonadotropic hormones; it is quite possible that gonad regression is due to their absence, or their destruction, or their failure to be released. Accordingly the antagonistic action of adrenalin is interpreted as being against these hormones; the antagonism for the gametogenic (FSH) hormone being responsible for the cessation of gametogenesis and the consequent regression of the germinal line; the antagonism for the luteinizing hormone (interstitial-cell-stimulating hormone) being responsible for the cessation of the sex hormone secretion - androgen or estrogen. The failure of these latter hormones then is undoubtedly responsible in turn for the regression of the accessories and secondary sex characteristics. The experimental evidence for this interpretation is born out by three lines of evidence. First, adrenalin causes regression in light stimulated birds. In such birds it is evident that despite the continued exposure to increased light the regression occurs. Hence the adrenalin in some way suppresses gonadotropic hormone formation,

or activity. Second, the regression brought about in antuitrin injected birds again indicates a possible antagonistic action of adrenalin on the bird's own gonadotropic hormones. Third, the regression brought about in the birds at the height of the breeding season is additional evidence that the gonadotropic hormones are being destroyed or prevented from stimulating the gonads.

That these regressive and inhibitory changes are produced by the antagonism of adrenalin for the gonadotropic hormones themselves, and that such changes are not due to other possible indirect methods of action seems evident for the following reasons. The metabolic rate of the experimental birds is not appreciably different from that of the controls. There is no appreciable loss of weight in adrenalin injected birds as compared to the control birds. Nor are there discoverable differences in liver or muscle glycogen between the two sorts of birds. Finally, confinement in the small experimental cages cannot be invoked to explain the regressive changes in the adrenalin injected birds. Regression did not occur in the control birds in the same type of cages.

The nature of the mode of action of adrenalin may be considered as directly chemical or indirectly nervous. If chemical, it must be by the destru ction of the gonadotropic hormones or by a prevention of their secretion or release. This latter mode may also be nervous. Conceivably the hypothalamic centers, or even the medulla oblongata, which control the functioning of the medulla of the adrenal gland may be affected in such a way that hypophyseal inhibition results and the pituitary ceases to form gonadotropic hormones. Whether this be the actual mode of action of the adrenalin or whether a chemical antagonism

occurs, the antagonistic action is clear and unmistakable. Cytological studies on the anterior pituitary, which should do much to clear up the mode of action of adrenalin, are now in progress.

How may this antagonistic action explain the annual breeding season of the sparrow and other seasonal breeders? How may it fit in with the theory that increased length of day light hours is the fundamental cause of the annual breeding season in sparrows? In corroboration of Rowan, we affirm that as the hours of day light increase the hours of wakefulness increase,and hence physiological activity increases. Goldzieher ('39) states, concerning the question of the existence of adrenalin in the circulating blood, -

" The much debated question has been conclusively answered by Tournade and Chabrol in their experiments on vaso-anastomosed dogs. As soon as the circulation is established between the adrenal vein of the healthy donor and the jugular vein of the adrenalectomized animal, the blood pressure of the latter rises, and it decreases again if the flow through the anastomosis is temporarily interrupted. Identical response of the heart rate and the blood sugar level confirm the exactitude of the experiment which proved that the discharge of adrenalin from the adrenal is continuous, and in addition to a mere massive secretion in case of emergency."

Goldzieher also states, -

" Muscular exertion reduces the adrenalin content of the gland, while the pheochrome substance disappears simultaneously from the medullary cells."

And again he writes, -

" Muscular activity is the common physiological stimulus of adrenal secretion."

Thus we are assured of two facts, namely, that adrenalin is continuously secreted in effective amounts into the circulating blood; and that muscular activity diminishes the available amount appreciably. Hence the more prolonged physiological activity in the birds kept awake by the long light day exhausts the supply of adrenalin from the adrenal

medulla, or at least diminishes the amount in the circulating blood to a level whereby, according to this conception, it can no longer prevent the gonadotropic activity of the anterior pituitary. The gonadotropic hormones would thus again become active; the gonads would be stimulated to the formation of germ cells and sex hormones; and the breeding condition would be attained. However, after the last brood of young is hatched and leaves the nest, the activity involved in care of the young diminishes. With this diminution of physiological activity the adrenalin in the bird's body may increase to a point above its antagonistic threshold value. The formation of gonadotropic hormones would thus be checked and consequently the entire reproductive mechanism would regress. In nature this regression actually occurs at a time when the hours of day light would still be sufficiently great to stimulate gonadal activity, if light alone were the causal stimulus. The above offers a plausible explanation of seasonal reproduction in English sparrows and reconciles the two opposing theories in vogue at the present time. Similar experiments in other seasonal breeding vertebrates should bring some interesting and new facts to light.

SUMMARY

1. Adrenalin injected in daily $\frac{1}{2}$ minim doses into sparrows brought to breeding condition by added daily light, by injections of antuitrin, and into normally breeding birds, produces a regression to the resting or juvenile condition of the gonads, accessories, and secondary sex characteristics.

2. The evident antagonism is interpreted as being directed against the secretion, release,or activity of the gonadotropic hormones of the ant-

erior pituitary.

3. Changes in metabolic rate, glycogen storage and release, and toxic effects are excluded as possible sources of this antagonism.

4. The mode of action of the adrenalin antagonism, whether chemical or nervous, has not been determined.

5. An explanation of the annual breeding season of the English sparrow is given in view of the proved antagonism of adrenalin on the reproductive cycle and the indubitable stimulating effects of increased daily light exposure, whether experimental or normal, and antuitrin injections.

CONCLUSION

This thesis supports the theory that increased daily exposure to the spring day light in the English sparrow brings about a greater expenditure of physiological activity. This increased energy makes for a diminished amount of adrenalin in the body. It thus falls below an anterior pituitary antagonistic value. When this occurs the breeding season is at hand. When physiological activity diminishes, even though the hours of daylight have not decreased appreciably, adrenalin again reaches this threshold value in the body and the anterior pituitary gonad mechanism is suppressed.

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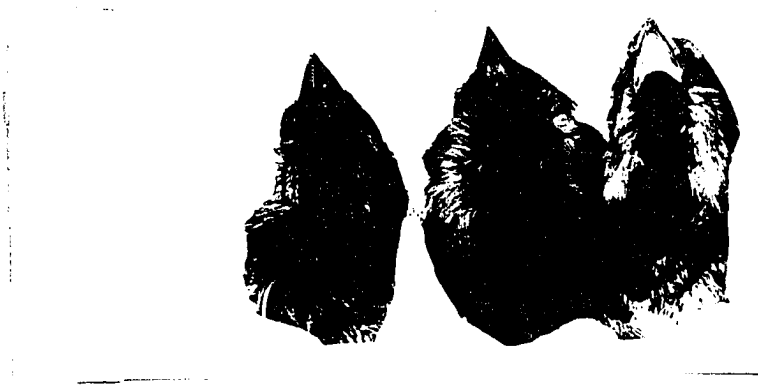
Plate I.

1. The beaks of 3 male birds illustrating the regressive effects of adrenalin on beak coloration. From left to right a 60 day light stimulated male, a 40 day light stimulated male - both have the jet black beak due to testis hormone secretion - a 60 day light stimulated adrenalin injected bird (S112); the beak has regressed to the juvenile coloration.
2 From left to right are shown the reproductive tracts of a normal quiescent or juvenile male; a 40 day light stimulated male (one testis was removed for sectioning); a 60 day light stimulated male; a 60 day light stimulated but adrenalin injected (40 to 60 days) male; the regression to the juvenile condition is evident.
3 Female reproductive systems are shown in the same manner as for the males in 2. Regression is especially noted in the oviduct of the adrenalin injected bird (S113). Note absence of conspicuous follicles in the experimental ovary.

PLATE I



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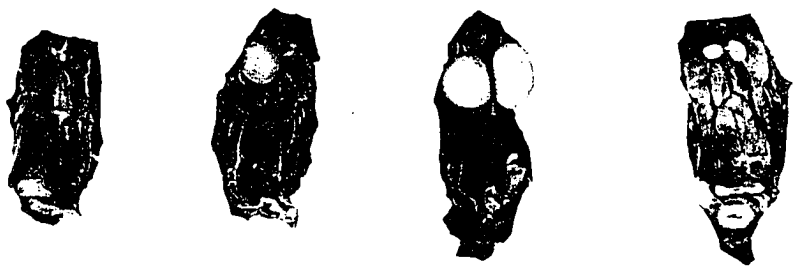

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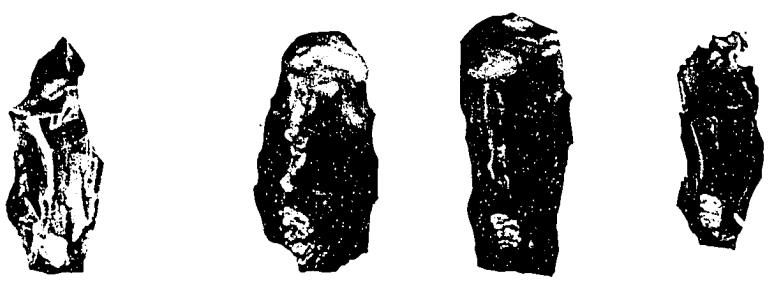

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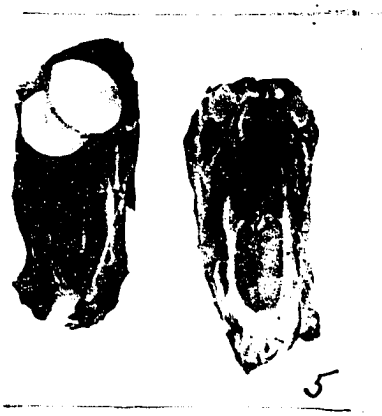
Plate II.

1 Two male beaks. The black is that of the antuitrin injected control J 100, series 4. The light beak is that of an adrenalin injected bird, J 102, given alternate injections of antuitrin and adrenalin after being brought to the breeding condition by antuitrin. Note the juvenile coloration.
2 The reproductive tracts of the same 2 males as in 1. The regression of the experimental bird is marked.
3 Two female reproductive tracts of birds J 101, control series 4, and J 103 series 4. Treated in the same manner as the males. Regression is obvious in both the ovary and oviduct of the latter.

4 Two male beaks. The black beak is that of a male at the height of the breeding season (April 15). The light beak is that of a male injected with adrenalin during the month of April. Again the juvenile coloration has resulted.
5 The reproductive tracts of the same males. Again regression is obvious.
6 The reproductive tracts of like females. Marked regression is evident.

PLATE II



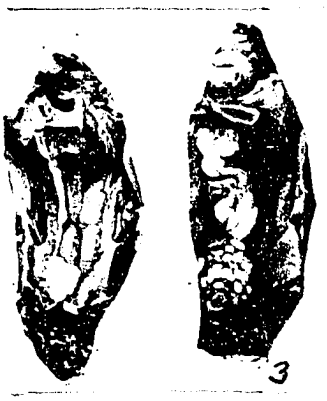


Plate III

All sections cut at 10 micra. All pictures taken with 10 x ocular and 4 mm. objective.

1 Portion of a normal quiescent juvenile testis. Note absence of all spermatogenesis; spermatogonia only are present.
2 Portion of the testis of male S 112 which received adrenalin injections from 4oth to 60 th day of light stimulation. The two testes are closely comparable. Regression is obvious when compared to 3 and 4.
3 Portion of testis of 40 day light stimulated bird.
4 Portion of testis of 60 day light stimulated bird.

PLATE III

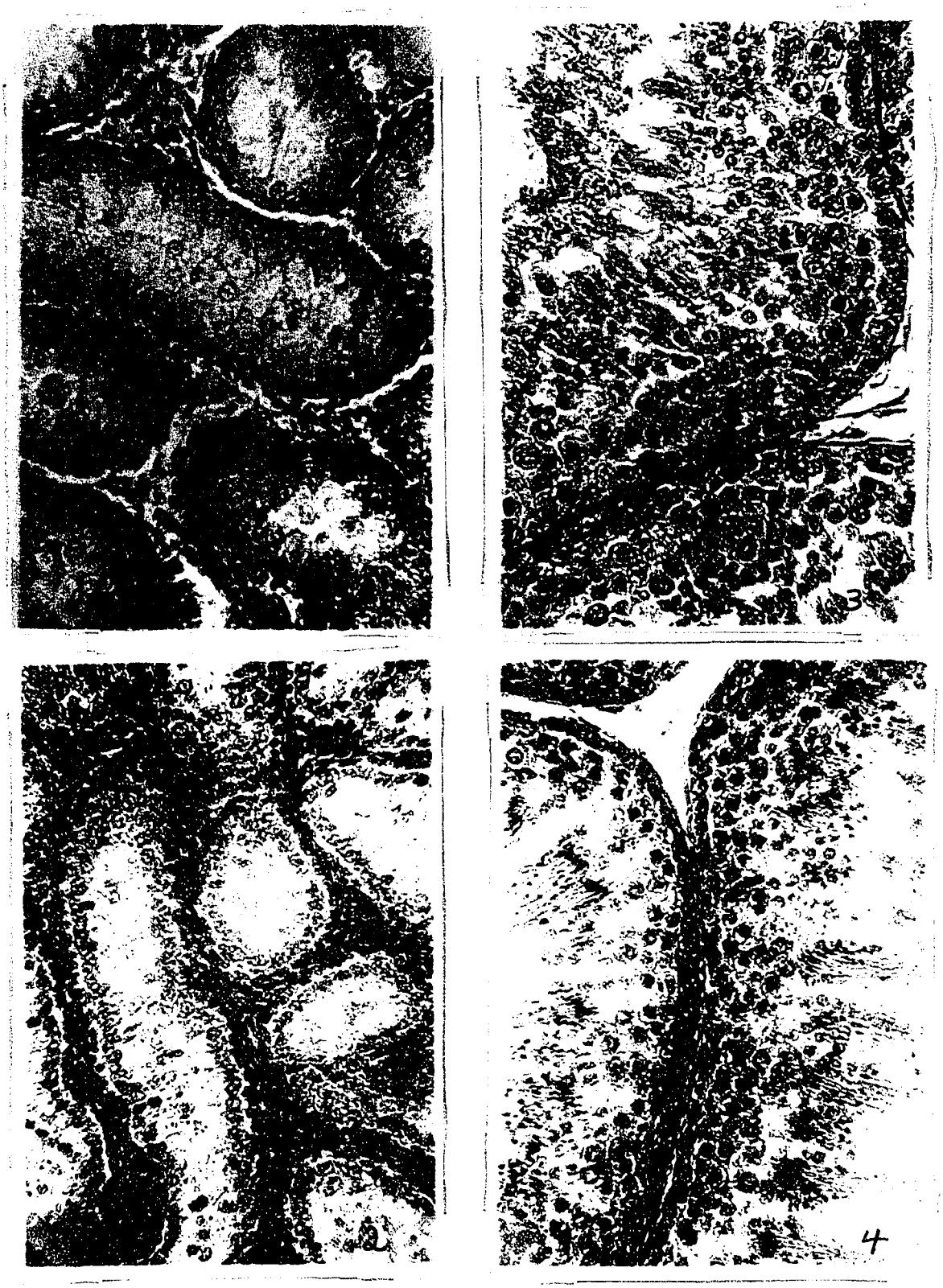
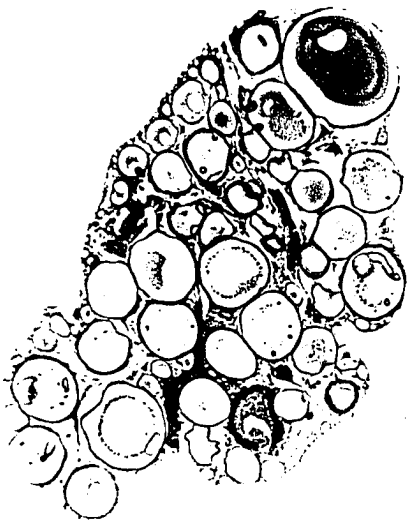


Plate IV

All sections cut at 10 micra. All pictures taken with 10 x ocular and 24 mm. objective.

1 An approximately median section of a normal quiescent juvenile ovary.
2 An approximately median section of the ovary of female S 111 which received adrenalin injections from the 40th to 60 th day of light stimulation. The two ovaries are closely comparable especially in size of follicles and in compactness of the same. Regression from the conditions observed in 3 and 4 is marked.
3 An approximately median section of an ovary of a 40 day light stimulated bird. Note enlarging follicles and yolk accumulation, also the peripheral positions of the larger follicles. Likewise an increase in the interfollicular tissue is obvious.
4 An approximately median section of an ovary of a 60 day light stimulated bird. The characteristics of 40 day bird are present and accentuated.

PLATE IV



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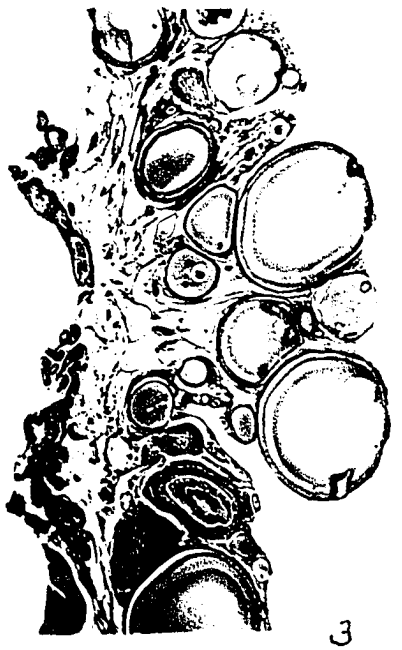

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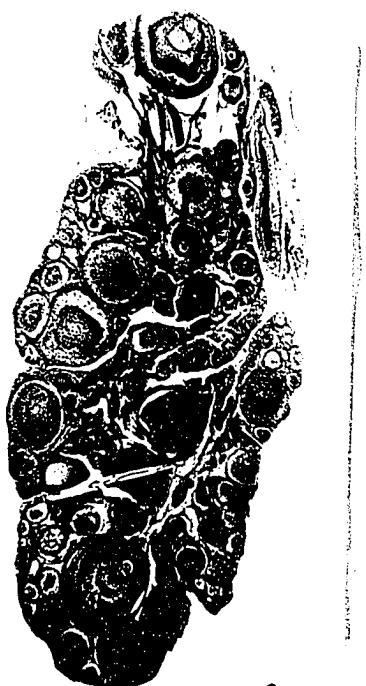

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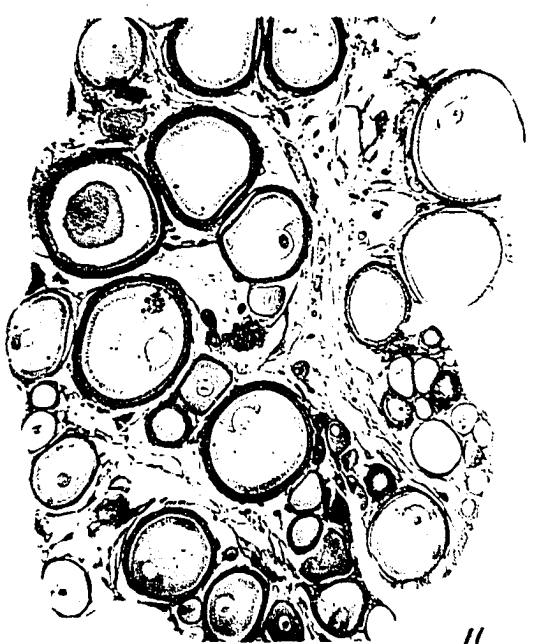

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Plate V

All sections cut at 10 micra. All pictures taken with 10 x Ocular and 4 mm. objective.

1 Two tubules of the spermatic glomus region of a quiescent juvenile bird.
2 Two tubules of the same region taken from the male bird S 112 which received adrenalin from the 40th to the 60th day of light stimulation. The juvenile condition is obvious.
3 A single tubule of the spermatic glomus t aken from a 40 day light stimulated bird. Note the great increase in the lumen and the high secretory epithelium.
4 A similar tubule taken from a 60 day light stimulated bird. In addition to the characteristics of the 40 day tubule note the numerous sperm in the lumen.

PLATE V

1

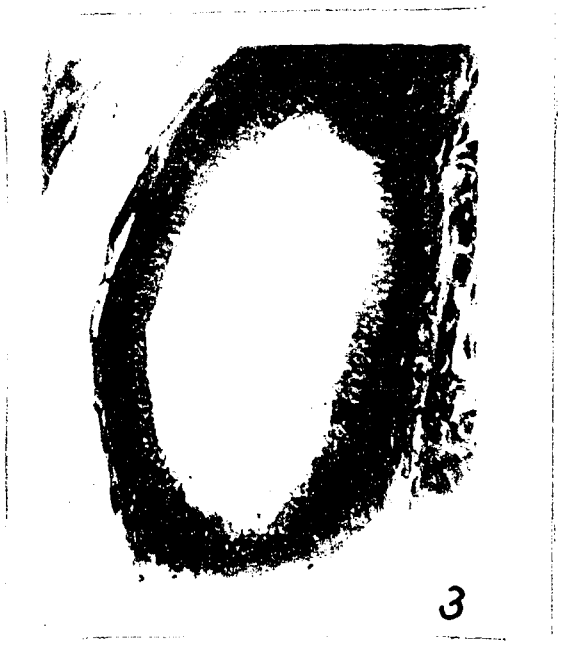

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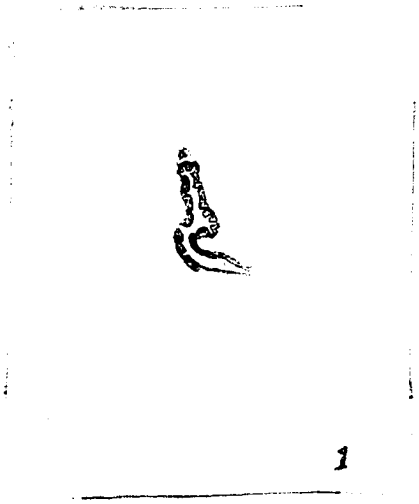
Plate VI

All sections cut at 10 micra. All pictures taken with 10X ocular and 24 mm. objective.

1 A section of the normal quiescent juvenile oviduct.
2 A section of the experimental bird oviduct - S 111. This bird was injected with adrenalin from the 40 th to 60 t h day of light stimulation. Note the close approximation to the juvenile condition and the manifest regression from the oviducts of 3 and 4.
3 A section of the oviduct of a 40 day light stimulated bird.
4 A section of the oviduct of a 60 day light stimulated bird.

PLATE VI



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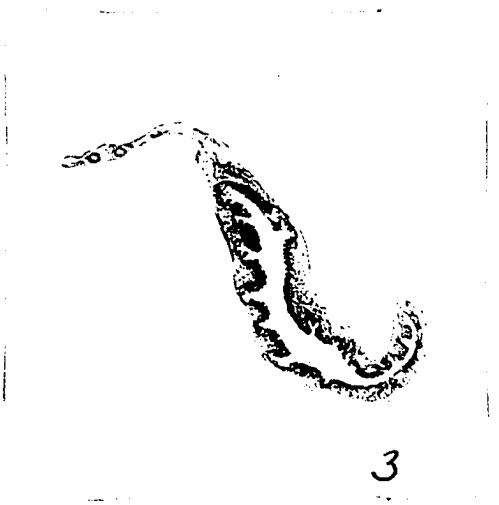

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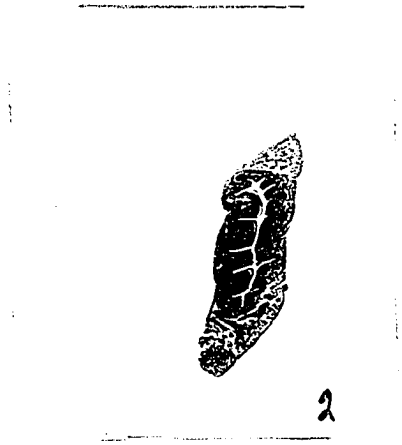

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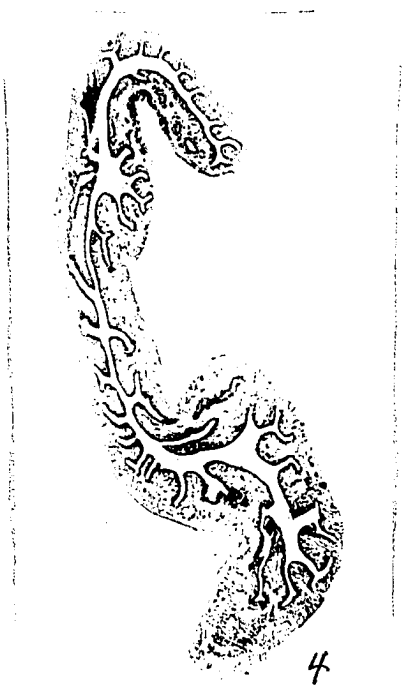

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