The Institute of Biology's
Studies in Biology no. 103

Sex Hormones and Behaviour

P. J. B. Slater

SP. 575
1

The Institute of Biology's
Studies in Biology no. 103

[redacted]

Sex Hormones and Behaviour

P. J. B. Slater
B.Sc., Ph.D.
Lecturer in Animal Behaviour,
University of Sussex

University Park Press
Baltimore

© Peter J. B. Slater 1978

First Published 1978 by Edward Arnold (Publishers) Ltd, London
First Published in the USA in 1979 by
University Park Press
233 East Redwood Street
Baltimore, Maryland 21202

Library of Congress Cataloging in Publication Data

Slater, Peter J. B.
Sex hormones and behaviour.

(The Institute of Biology's studies in biology; no. 103)
Bibliography: p.
1. Hormones, Sex—Physiological effect. 2. Animals, Habits and behavior of. 3. Reproduction. I. Title. II. Series: Institute of Biology. Studies in biology; no. 103.
QP572.S4S55 1978 599'.05 78-13706

ISBN 0-8391-0253-4

All rights reserved. No part of this publication may be reproduced, stored in a retrieval system, or transmitted in any form or by any means, electronic, photocopying, recording or otherwise, without the prior permission of Edward Arnold (Publishers) Ltd.

Printed in Great Britain

General Preface to the Series

Because it is no longer possible for one textbook to cover the whole field of biology while remaining sufficiently up to date the Institute of Biology has sponsored this series so that teachers and students can learn about significant developments. The enthusiastic acceptance of 'Studies in Biology' shows that the books are providing authoritative views of biological topics.

The features of the series include the attention given to methods, the selected list of books for further reading and, wherever possible, suggestions for practical work.

Readers' comments will be welcomed by the Education Officer of the Institute.

1978

Institute of Biology
41 Queen's Gate
London SW7 5HU

Preface

There are many texts on reproductive physiology, but few of them devote any space to behaviour. This little book aims to fill this gap at an introductory level. It is a foolhardy enterprise to attempt to cover so large a field in so few pages, and not all will agree with my choice of material or ways of simplifying it. The reward will be if less expert readers are encouraged to seek out more details by moving on to books which provide fuller treatment. The first two chapters provide a brief account of reproductive physiology as a background to understanding the behavioural effects of hormones: readers already familiar with the relevant physiology may prefer to move straight to Chapter 3.

I am grateful to Professor Richard Andrew, Dr Lynda Birke, Mrs Rosemary Lewis and Mrs Elisabeth Slater, all of whom made helpful comments on the manuscript. Thanks are also due to the many people whose work has provided me with examples: my only regret is that space has not allowed me to mention them by name or cite references to them.

Sussex, 1978

P. J. B. S.

Contents

此为试读，需要完整PDF请访问：www.ertongbook.com

1 The Brain and Hormone Secretion

1.1 Hormones and nerves

As the main concern of this book is to outline the effects of hormones on behaviour, it is as well to start by considering why hormones are necessary at all in this context. After all, the main function of the nervous system is to produce behaviour and it is an extremely efficient carrier of messages between various parts of the body. Why then have a second system of communication based on hormones?

The answer to this question is that both the nervous and endocrine systems have special characteristics which make them useful for specific tasks to which the other would be poorly suited. The nervous system has the advantages of speed, specificity and transience. A message may be sent from one end of the body to the other in a fraction of a second. It can contact a single muscle without influencing others and the activity of that muscle can be altered quickly by the pattern of impulses passing down the nerve. Hormones act very differently. They are not generally secreted quickly nor can they be rapidly removed from the bloodstream; their speed of movement is limited by that of the blood. Yet, just because of these characteristics, a given hormone level can easily be maintained in the bloodstream to provide constant stimulation over days, months or even years. This stimulation is also general. Unlike nerve impulses with their specific destinations, hormones are able to contact every cell in the body (and may indeed alter the functioning of different cells in different ways).

Thus the nervous and endocrine systems are adapted for complementary roles. In the control of behaviour hormones are of importance largely in bringing about long term changes in responsiveness: this is nowhere more obvious than in reproduction, where hormones are mainly involved in the rather slow changes taking place during the course of days rather than in moment to moment switches in behaviour.

Not surprisingly, the two systems of communication influence each other strongly. The nervous system senses changes in both the internal and external environments and is able, through its close contact with the endocrine system, to modify hormone secretion in response to the information it receives. This is achieved through the phenomenon of neurosecretion. Certain nerve cells are modified to synthesize hormones and release them into the bloodstream in response to nervous signals.

The existence of neurosecretion is widespread in the animal kingdom and it is the main way in which nervous activity may modify hormone output. We will therefore consider the effect that it has on hormone production in some detail here. The other side of the relationship – the way in which hormones influence the behavioural output of the brain – must wait for later chapters.

1.2 The pituitary gland

In vertebrates the major influence of the nervous system on hormone secretion is through the pituitary gland, which lies just above the roof of the mouth and immediately below an area of the brain called the hypothalamus (Fig. 1–1). The pituitary used to be known as the

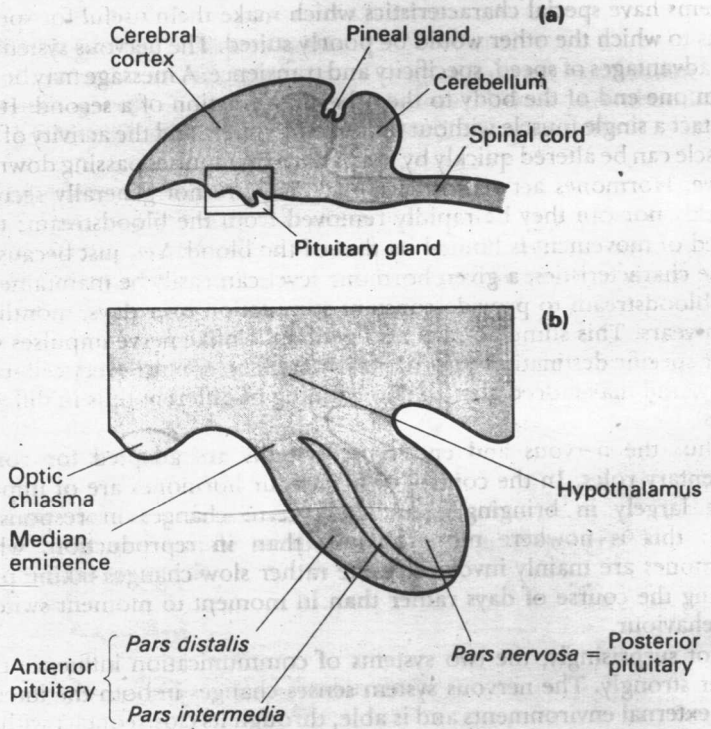

Fig. 1–1 The mammalian pituitary gland. (a) Its position in relation to the brain as a whole in the rabbit. (b) The main divisions of the pituitary and its attachment to the hypothalamus.

'conductor of the endocrine orchestra', but recent research suggests that it would be more appropriately called the 'leader' as, isolated from the controlling influence of the hypothalamus, its functioning is drastically changed.

The pituitary is in fact not one gland but two, each controlled by the brain in a different way. Table 1 lists the hormones they produce in mammals, with their confusing variety of pseudonyms, and summarizes the way in which the brain controls them and the main functions which they have. Some of these functions will be considered in more detail in later chapters.

The posterior pituitary or neurohypophysis is the simpler of the two parts. It is a neurosecretory organ producing two hormones which are synthesized in cell bodies within the hypothalamus and travel down their axons to be secreted into the blood from nerve endings within the gland. One of these hormones, vasopressin, is largely concerned with water regulation by the kidney, while the other, oxytocin, is involved in labour and suckling. We shall therefore return to it later.

The anterior pituitary or adenohypophysis produces no less than seven hormones. Four of these are not directly concerned with reproduction and so only deserve brief mention. Melanocyte stimulating hormone (MSH) is secreted from the pars intermedia immediately in front of the neurohypophysis; thyroid stimulating hormone (TSH), adrenocorticotropic hormone (ACTH) and growth hormone (GH) emanate from the more anterior section known as the pars distalis.

The remaining three anterior pituitary hormones also come from the pars distalis and are known collectively as the gonadotropins because of their effects on the gonads. Follicle-stimulating hormone (FSH) is primarily involved in stimulating growth of the ovaries and secretion of oestrogen from them in females, while in males it brings about spermatogenesis. Luteinizing hormone (LH) also affects ovarian growth, but its main function in females is to stimulate ovulation; in males it leads to secretion of the hormone testosterone from the testes. FSH and LH are thus responsible for stimulating growth of the gonads and the release of male and female sex hormones from them. Although, as outlined above, some of these changes are primarily due to one rather than the other, their effects overlap considerably and they normally act together. The third gonadotropin is prolactin, which has various functions, the best known being the stimulation of milk production from the mammary glands.

Secretion from the anterior part of the pituitary is controlled in a very different way from that of the posterior part. The secretory activity of the latter depends on an intact nervous connection with the hypothalamus, for it is down these nerves that the hormones are passed for release from the gland. By contrast, the anterior pituitary continues to function normally if its nerve supply is cut but ceases to do so if the blood vessels to

Table 1 The hormones of the mammalian pituitary gland.

Hormone	*Control of secretion*	*Effects*
ANTERIOR PITUITARY = Adenohypophysis		
Pars distalis		
Adrenocorticotropic hormone (ACTH)	ACTH releasing factor (CRF)	Stimulation of hormone secretion from the adrenal cortex
Thyroid stimulating hormone (TSH)	TSH releasing factor (TRF)	Stimulation of thyroxine secretion from the thyroid
Growth hormone (GH = Somatotropic hormone (STH))	GH releasing factor (GH–RF) GH inhibiting factor (GIF)	Metabolic effects throughout body include promotion of growth
Gonadotropins:		
Follicle stimulating hormone (FSH)	FSH releasing factor (FSH – RF = GnRF?)	Ovary – growth and oestrogen secretion Testis – growth and spermatogenesis
Luteinizing hormone (LH)	LH releasing factor (LH – RF = GnRF)	Ovary – ovulation and may support corpus luteum Testis – testosterone secretion
Prolactin (= Luteotropic hormone (LTH) = Lactogenic hormone)	Prolactin inhibiting factor (PIF) Prolactin releasing factor (PRF)	Milk production May support corpus luteum
Pars intermedia		
Melanocyte stimulating hormone (MSH)	MSH releasing factor (MRF) MSH inhibiting factor (MIF)	Uncertain function in mammals (skin darkening in lower vertebrates)
POSTERIOR PITUITARY = Neurohypophysis		
Pars nervosa		
Oxytocin	Synthesized in hypothalamus and released from nerve endings in gland	Contraction of smooth muscle in uterus and mammary glands
Vasopressin (= Antidiuretic hormone (ADH))	Synthesized in hypothalamus and released from nerve endings in gland	Water resorption in kidney

it from the hypothalamus are severed. Although this discovery, made over twenty years ago, suggested chemical control of the anterior pituitary, in much the same way as its own hormones control secretion of the thyroid, gonads and adrenal cortex, the substances exerting this influence have proved hard to isolate. Collectively, they are known as releasing and inhibiting factors or, especially the better known ones, as releasing and release-inhibiting hormones. They are synthesized in the hypothalamus and secreted into the pituitary portal vessels at the median eminence just above the pituitary (see Fig. 1–1). Acting at such short range, they are produced in very small quantities and appear to be quickly diluted or destroyed elsewhere in the body. Those that have been identified are polypeptides: TSH releasing factor (TRF) consists of only three amino-acids, while the releasing factor controlling LH (LH–RF) consists of ten. The main evidence for the existence of many of the others listed in Table 1 is that substances with activity appropriate to their names have been found in extracts of the hypothalamus. Not all of them may exist as separate chemicals, and the list in the table is therefore a tentative one. It is, for example, still doubtful if FSH has a releasing factor of its own, for LH–RF stimulates FSH secretion also and, because of this, is sometimes referred to as gonadotropin releasing factor (GnRF).

Unlike most other anterior pituitary hormones, prolactin is primarily controlled by an inhibiting factor in mammals: if the pituitary is transplanted to a point in the body far from the hypothalamus, or kept in culture, more prolactin is produced due to removal of the suppressing influence of this substance. Smaller quantities of a prolactin releasing factor have also now been isolated from hypothalamic extracts and it seems likely that this hormone, GH and MSH each have both a releasing and an inhibiting factor. It is interesting that these three hormones are the three anterior pituitary ones which do not stimulate the release of other hormones elsewhere in the body. They are thus not subject to the negative feedback control typical of the others. In the absence of this, both releasing and inhibiting factors may be necessary to achieve fine modulation of their output.

Negative feedback is a widespread and important controlling mechanism in physiological systems. Amongst hormones it occurs where one stimulates production of another and this in turn suppresses the first so that a balance between them is reached. Thus FSH leads to oestrogen secretion from the ovary and oestrogen feeds back to lower FSH output. If oestrogen levels rise too high they suppress FSH production and, as a result, less oestrogen is produced. If too little oestrogen is secreted, it will not suppress FSH so that more is released, thus raising the oestrogen level once more. Relationships of this sort enable hormone levels to be set very precisely and changes in them to be tightly controlled. Negative feedback may act at various points in a series of hormones each of which stimulates another. Thus it has been found that the main feedback effect of

oestrogen is not on FSH itself but on the production of its releasing factor (FSH–RF or GnRF) from the hypothalamus. The effect is, however, the same, for suppression of the releasing factor leads to lower FSH output.

1.3 The hypothalamus and gonadotropin secretion

What areas of the hypothalamus are involved in controlling release of LH and FSH through their influence on the production of releasing factors? Three methods have been commonly used to try to answer this question. By causing tiny lesions in specific areas, usually by passing a current between two electrodes close together, parts of the brain can be destroyed and the effect of this noted. Alternatively, by stimulating electrically in specific locations with a very much smaller current than that required to produce a lesion, increased neural activity can be induced. If the part stimulated has a positive effect on hormone secretion, greater amounts of hormone should result. Lastly, small quantities of hormone can be introduced into the brain on the tip of a fine needle or through a thin tube. Oestrogen is the hormone most often used in this way because of its negative feedback effect on FSH production. As this effect is via the hypothalamus, oestrogen placed in the relevant area should cut down on production of FSH by the pituitary. The effects of oestrogen on LH are more complicated, but one is that it stimulates a burst of LH from the pituitary which in turn leads to ovulation.

What then have studies of these three types shown? Figure 1–2 is a simplified diagram of those nuclei in the hypothalamus relevant to the following discussion.

Cells in the pre-optic nucleus and anterior hypothalamus are important in the control of ovulation. In the rat, lesions in this area stop ovulation, while electrical stimulation leads to it. Neither of these treatments affects the weight of the ovaries, and thus it seems that this area is mainly important in controlling the pulse of LH which is responsible for ovulation rather than the steady output of FSH and LH which maintains the condition of the ovaries. The fact that oestrogen implants in the anterior hypothalamus also give ovulation supports this idea: it appears to be here that oestrogen acts to stimulate the ovulatory peak of LH. In addition, the effects of some external stimuli on hormone secretion involve this area. For example, in common with many other species (see section 1.5), ferrets are normally brought into breeding condition by increased day length in spring. Females with anterior hypothalamic lesions come into condition in winter: the inhibiting effect of short days on FSH and LH secretion has been removed. But despite the fact that these ferrets are receptive to males, they do not produce eggs because, as with other species, the pulse of LH leading to ovulation has been removed.

The basal tuberal area of the hypothalamus, which contains the arcuate

此为试读，需要完整PDF请访问：www.ertongbook.com

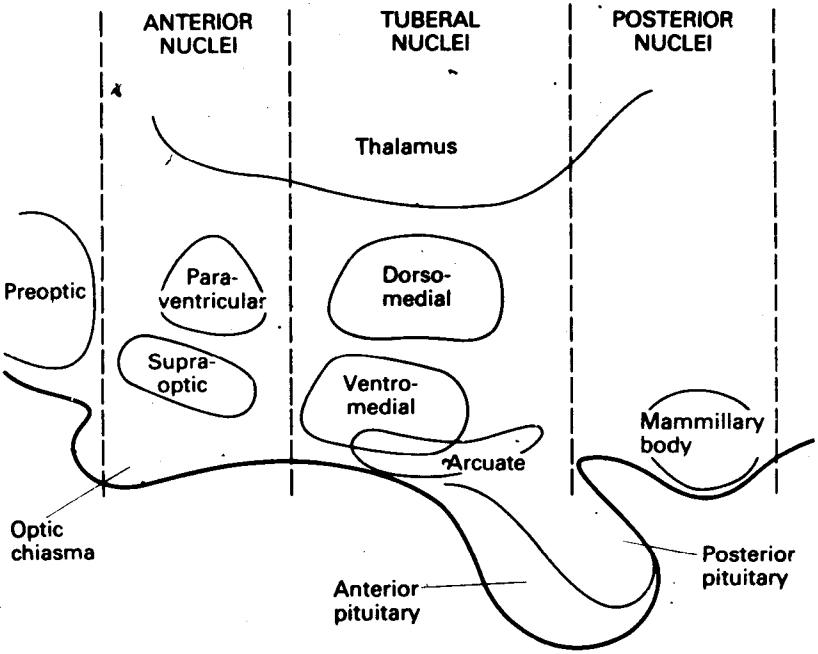

Fig. 1–2 A schematic diagram of the regions of the hypothalamus and some of its main nuclei as seen in midline section.

and ventro-medial nuclei, has a different role. Lesions here lead to ovarian atrophy and oestrogen implants also have this effect. These results point to this region as being important in the production of the tonic levels of FSH and LH which support the gonads and also being the major site of negative feedback by oestrogen. As the basal tuberal area lies between the anterior hypothalamus and the pituitary, it is not surprising that lesions in it also block ovulation. Electrical stimulation has the opposite effect: ovulation in the rabbit can be elicited by a small current passed into this area for only three minutes. This presumably leads to release of LH–RF from the median eminence and thus LH from the pituitary. Certainly the result is not due to some indirect effect of the current on the pituitary itself, for seven and a half hours of equivalent stimulation applied directly to the pituitary did not induce ovulation.

The general picture which emerges is that the anterior hypothalamus and pre-optic nucleus control the cyclical release of LH leading to ovulation, while the basal tuberal areas are responsible for the more constant, low level, secretion of FSH and LH which gives gonad growth and sex hormone secretion in both males and females. Negative feedback of oestrogen is largely to the basal tuberal area, while this hormone has

the effect of enhancing LH output through the anterior hypothalamus. All these effects on FSH and LH are of course indirect, acting through changes in the secretion of their releasing factors from the median eminence. The different roles of these two main brain areas are summarized in Fig. 1–3.

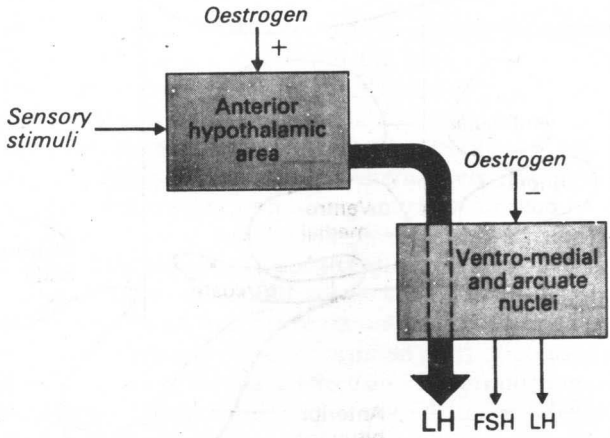

Fig. 1–3 Schematic diagram showing the main areas of the hypothalamus controlling the release of LH and FSH in female mammals.

Less is known of the control of prolactin secretion. The evidence, even for the better known of the factors controlling it, PIF, is circumstantial as this substance has not yet been isolated and identified. Lesions in various parts of the hypothalamus have been found to affect prolactin output suggesting that the brain mechanisms controlling it are diffuse.

1.4 The development of the reproductive system

The gonadotropins and the sex hormones do not suddenly start to be secreted at puberty. They are also involved in the process of sexual development even in the embryo. Testosterone, in particular, plays a vital part in determining the primary sexual characteristics. If testosterone is absent at this early stage female genitalia develop, whereas in its presence the growth of male features occurs. Up to this point (before the sixth week of gestation in man), the embryo has rudiments of both ovary and testis, but testosterone secretion then starts in individuals which are genetically male and causes sex differences to emerge. If no testosterone is present, female ducts and genitalia develop: this occurs normally in females but will also happen in males whose embryonic gonads have been removed. The female state is therefore the neutral one, which is masculinized in

the presence of testosterone. Another factor is also involved, however. Female embryos treated with testosterone do not become totally male but develop both male and female gonads and ducts. Experimental evidence points to the existence of another hormone produced by the embryonic testis, which diffuses across to the ovarian rudiment and suppresses its development. Female embryos lack this, so their ovaries will develop even if they are given testosterone.

Another difference between males and females which is brought about by testosterone concerns the control of gonadotropin release by the hypothalamus. In adult males LH and FSH are continuously released, whereas in females, superimposed upon these low levels is the cyclical peak, primarily of LH, which stimulates ovulation. This difference between the sexes has been shown to depend upon whether or not testosterone is present at a critical stage of development: in most species this is before birth but in rats, which are very immature when born, it is in the first week of life. Treatment of young rats with testosterone in the few days after they are born has very dramatic effects. While males show normal subsequent development, in females the cyclical release of LH is found to be absent when adulthood is reached. The ovaries grow and eggs develop, but ovulation does not occur and nor do other cyclical changes in physiology and behaviour typical of adult females. This same effect of testosterone occurs normally in males, as they produce small quantities of this hormone naturally just after birth. But if young male rats are castrated at birth, so that testosterone is not present subsequently, and they are given ovarian implants as adults, these ovaries show cycles as they normally would only in females. From these results one can conclude that testosterone is responsible for abolishing the potential for cyclical LH release in males, while the absence of this hormone in young females allows the hypothalamus to develop this capacity. Evidence that the effect of testosterone is on the hypothalamus rather than the pituitary stems from the discovery that female rats, whose pituitaries are replaced by ones transplanted from males, still show ovulation. These pituitaries are thus showing the female pattern of secretion as determined by the sex of the hypothalamus that overlies them.

Sex determination of both the reproductive system and the hypothalamus is therefore largely dependent on the presence or absence of testosterone during critical periods of development. That oestrogen does not have a role to play in this in mammals may be due to the fact that the embryo receives a certain amount of this hormone across the placenta from the mother: if oestrogen had a feminizing effect, all embryos might become female!

The effects so far discussed leave the young animal poised to become either male or female, but a period of immaturity follows during which the capacity for reproduction remains latent. Then, at puberty, sex hormones start to be secreted in larger quantities causing the

development of secondary sexual characteristics and the onset of changes in behaviour and physiology related to reproduction. It is, however, wrong to suppose that reproductive hormones are not secreted between infancy and puberty. In females FSH and oestrogen are in delicate balance with each other, as they are in the adult, but the levels of both hormones are very much lower before puberty. If the ovaries of a young rat are removed, so that the negative feedback cannot act, FSH output increases markedly. Higher FSH levels such as this will cause growth of the ovaries of other immature females. Thus both ovary and pituitary are fully capable of functioning in an adult manner before puberty but inhibit each other from doing so: too little FSH is produced to give ovarian growth and FSH output is kept low by the small amount of oestrogen that the ovaries do secrete. At puberty, the balance changes and the quantities of FSH and oestrogen start to rise. The mechanism underlying this is unclear, but the hypothalamus may for a time react to oestrogen by increasing rather than lowering FSH output. The hypothalamus is certainly, once again, closely involved: an unfortunate human condition called pubertas praecox, in which very young children become sexually mature, often results from tumours or cysts damaging this part of the brain.

Table 2 summarizes the role of hormones in each of the three main stages of reproductive development discussed in this section.

1.5 Development within the breeding season

Most animals, unlike man, do not breed the whole year round, but only produce young when conditions are favourable for their rearing. In higher latitudes this usually means during the spring and summer, but nearer the equator such conditions may persist for much of the year. Here some bird species may be found nesting in every month, others do so only in the rainy season and in others breeding seasons are not annual. An example of this last situation is in the wideawake tern of Ascension Island which breeds every nine months.

Where breeding is restricted to one season of the year, the gonads regress after this period but grow again at the start of the new season. In temperate zone birds this growth occurs in the spring, one or more breeding cycles then take place, and the animals go out of breeding condition again in the late summer. Though many small mammals behave similarly, in large ones the situation is often more complicated because of the long period of development within the uterus. In sheep, for instance, breeding condition is attained in the autumn, and copulation occurs then, but the young are not born until the following spring.

What factors control these annual cycles of reproduction? In many of those birds and mammals which attain breeding condition in spring it is

Table 2 The effects of hormones on reproductive development in male and female mammals.

Stage of Development	*Male*	*Female*
Early in foetal life	*Testosterone* gives male primary sexual characteristics	Female primary sexual characteristics and ovaries develop if testosterone and local factor absent
	Local factor from testes suppresses ovarian development	
At around time of birth	*Testosterone* makes hypothalamus male, suppressing mechanism for cyclical LH release	Mechanism for cyclical LH release develops in absence of testosterone
At puberty	Increase in *FSH and LH* secretion leads to growth of testes and higher testosterone output	Increase in *FSH and LH* secretion leads to growth of ovaries and higher oestrogen output
	Testosterone gives male secondary sexual characteristics	*Oestrogen* gives female secondary sexual characteristics
		Cyclical LH release starts, leading to ovulation

known that the most important single influence is the increasing day length at this time. Experiments in which such animals are given longer days artificially a month or two earlier than usual have found that this will make them come into breeding condition prematurely. Somehow the increased light is stimulating FSH and LH output through the hypothalamus and pituitary. This may happen by a number of possible routes. As there is a pathway from the eyes to the hypothalamus, this is perhaps the simplest, but it is known that the reproductive system of some species can respond to increased light even in animals which are blinded. Surprisingly, there is some evidence that this may be because the hypothalamus itself responds to the very low levels of light which penetrate to it through the skull and brain. A further route is much more roundabout. It is known that the tiny pineal gland, lying on the roof of the forebrain (see Fig. 1–1), secretes the hormone melatonin, which suppresses gonadotropin output. In birds and mammals, the pineal is not itself sensitive to light, but its secretion is suppressed by light entering the eyes. Thus, when day length gets longer, it will produce less melatonin and

thereby remove the inhibition which this substance has on FSH and LH secretion.

Although it is well established that light can affect hormone secretion, and something is known of the neural pathways involved, a more difficult question remains which is relevant to other external stimuli which influence hormone secretion as well as to light. Do these stimuli act simply on physiological processes or do they act by modifying the behaviour of the animal which in turn stimulates the change in hormone output? Perhaps, for example, increased day length allows animals to be active for longer and it is this which enhances gonadotropin secretion. By the use of cycles which diverge from 24 hours long, gonad growth can sometimes be obtained even when animals are kept in the dark for much of the time. In species where this is the case, the likelihood of gonad growth seems to depend on how active the light schedule makes the animal become. The bird whose activity is plotted in Fig. 1–4 underwent

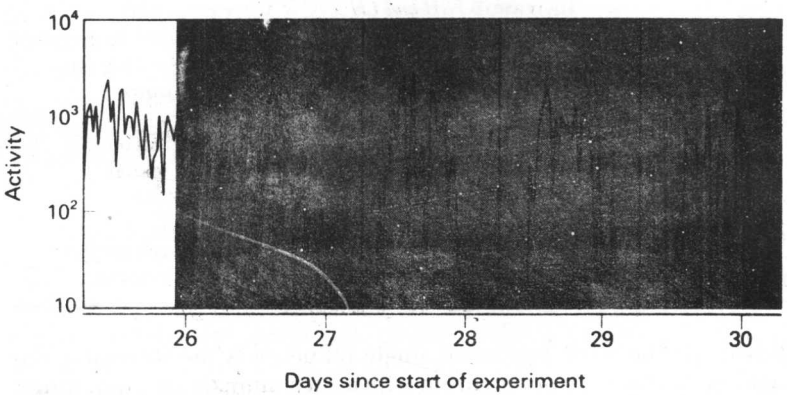

Fig. 1–4 The activity pattern of a white-throated sparrow on a light regime involving 16 hrs light every five days. (After WOLFSON, A. (1966). *Rec. Progr. Hormone Res.*, 22, 227.)

gonad growth despite receiving only sixteen hours of light in every five days. The schedule did, however, make it active for about sixteen hours in each of those days. This activity may have been responsible for the gonad growth, but some caution is required, for it is also possible that gonad growth and activity result from a common mechanism rather than one causing the other.

The role of behaviour in giving gonad growth is clearer in some other bird species. For example, the testes of male budgerigars regress if the birds are kept in isolation, but will not do so if they are able to hear other males singing. This is not, however, a direct effect of the other males on their gonadotropin secretion, for it depends on their own ability to sing:

此为试读,需要完整PDF请访问: www.ertongbook.com