BIOLOGY OF REPRODUCTION 13, 603–609 (1975)

Role of Catecholamines in Photoperiodically-Induced Gonadal Development in Coturnix Quail[1]

M. E. EL HALAWANI and W. H. BURKE

Department of Animal Science,
University of Minnesota,
St. Paul, Minnesota 55108

ABSTRACT

The involvement of central biogenic amines in the mechanisms controlling photoperiodically-induced testicular development in Coturnix quail were studied employing pharmacological approaches. Daily administration of alpha-methyltyrosine (MT), which blocks catecholamine (CA) biosynthesis, to 6-week-old quail exposed to long daily photoperiods markedly depleted brain dopamine (DA), norepinephrine (NE) and epinephrine (E). This was associated with a partial suppression of testicular growth 7 days after light stimulation. Combined treatment with MT and L-dihydroxyphenylalanine (L-DOPA), a precursor of DA, NE and E, elevated the monoamine levels in the brain and prevented the blocking effect of MT on testicular growth. Selective blockade of NE and E biosynthesis with diethyl-dithiocarbamate (DDC) depleted both NE and E, while it elevated DA level. Furthermore, such treatment reduced testicular weight. In DDC-treated birds, D,L-dihydroxyphenylserine (DL-DOPS) administration, which bypasses the DDC block, restored only brain NE level and reversed the blocking action of DDC on testicular weight.

It is concluded that central NE plays a role in transformation of the photoperiodic information affecting gonadal development in Coturnix quail.

INTRODUCTION

Gonadal growth in birds is regulated by pituitary gonadotropins. Either LH or FSH can stimulate avian gonadal growth and this response has often been used as the end-point for "total gonadotropin" bioassays.

Extra retinal photoreception has been shown to be necessary for the photoperiodic induction of gonadal development in some avian species (Menaker, 1971). Homma and Sakakibara (1971) have demonstrated the presence of deep brain photoreceptors in Coturnix quail. Monoamine containing neurons have been demonstrated and localized in bird brains (Sharp and Follett, 1968; Ikeda and Gotoh, 1971; Warren et al., 1973; Calas et al., 1974) but their role in the regulation of avian gonadotropic activity, and the nature of the chemical transmitter(s) which play a role in this function is not known. High norepinephrine (NE) levels have been associated with conditions that increase LH release in the chicken (Graber and Nalbandov, 1972), and Campbell and Wolfson (1974) have shown that hypothalamic NE turnover is increased in light-stimulated quail. Photoperiodically-induced gonadal growth in quail is accompanied by elevated serum LH (Nicholls et al., 1973) and it can be blocked by lesions of the hypothalamus (Sharp and Follett, 1969) or intraventricular infusion of 6-hydroxydopamine (Davies and Follett, 1974).

The objective of these studies was to investigate the role of brain catecholamines as mediators of light induced gonadal growth. Photoperiodically-induced gonadal growth of Japanese quail *(Coturnix coturnix japonica)* was studied after administration of drugs designed to manipulate the level of catecholamines (CA) in the central nervous system. The mechanisms behind the observed changes in gonadal growth were studied by determination of brain dopamine (DA), NE, epinephrine (E) and serotonin (5-HT) levels.

MATERIALS AND METHODS

Male Coturnix quail were used in all experiments. During the first 3 weeks of life the birds were housed in electrically heated batteries and exposed to continuous illumination. After this time the birds were subjected to a nonstimularory light schedule, i.e. 6 h of light and 18 h of darkness (0900–1500 h) daily. When the birds were 6 weeks of age (body weight 100–120 g) and had been subjected to the nonstimulatory light regimen for 3 weeks, they were provided with 14 h of light and 10 h of darkness (0900–2300

Accepted September 16, 1975.
Received May 27, 1975.

[1] Minnesota Agricultural Experiment Station Scientific Journal Series number 9121.

h) per day. In experiments 1, 2 and 3 one group which served as a negative control was kept on a nonstimulatory light regime and another control group of light-stimulated quail were treated with physiological saline.

Starting from the day of light stimulation, 0.15 M saline solutions or suspensions of the following compounds were prepared daily and administered: DL-alpha-methyl-P-tyrosine methyl ester HCl (MT, 250 mg/kg ip); L-3,4-dihydroxyphenylalanine methyl ester HCl (L-DOPA , 125 mg/kg ip); sodium diethyl-dithiocarbamate (DDC, 250 mg/kg ip); DL-threo-3,4-dihydroxyphenylserine (DL-DOPS, 200 mg/kg ip); and synthetic gonadotropin releasing hormone (LRH, Spectrum Med. Ind., Los Angeles; 1.25 ug/bird, im).

Birds were injected daily at 0900 and 1600 h. Injections were given for 7 days beginning on the first day of stimulatory light and continuing for 7 days. The birds were sacrificed 3–4 h after the last injection in all experiments (i.e. 3–4 h before the end of the light period). The negative control birds, which were in the dark phase of the photoperiod (4 h after the end of the light period) at the time of sacrifice, were brought into a lighted room and killed within 30 seconds, by decapitation. Birds in other experimental groups were mixed and killed at random within about 1 h period to avoid confounding the data with known diurnal changes in CA. Body and testicular weights were recorded at the time of sacrifice. The brain from each bird was quickly removed, weighed and frozen on dry ice. Brains were stored at −20°C until analyzed for CA. The details of group arrangement and drugs employed for each experiment are given in Tables 1–4.

Biochemical Analyses

After homogenization of brains in perchloric acid and centrifugation at 30,000 g, the supernatant was subjected to cation-exchange chromatography as described by Barchas et al. (1972). The following spectrophotofluorometric analyses were performed: NE and E (Anton and Sayre, 1962), using a Turner III filter spectrophotofluorometer, DA (Anton and Sayre, 1964), and 5-HT (Bogdanski et al., 1956), using an Aminco Bowman spectrophotofluorometer. Recovery estimates were made in duplicate for each monamine for each assay. The values reported are corrected for recovery. The percent recoveries (mean ± standard deviation) for each amine were: DA, 87.9 ± 1.3; NE, 83.4 ± 0.9; E, 80.0 ± 1.0 and 5-HT, 93.6 ± 2.7.

The data were statistically analyzed using analysis of variance and significant ($P<.05$) differences between means were determined using Student-Newman-Keul's test (Snedecor and Cochran, 1967).

RESULTS

Body weights were obtained at the termination of each experiment, as a check for potential toxicity. In no case did the agents used (MT, DOPA, DDC, DOPS) or combinations of them significantly change the body weights from those of saline controls.

Experiment 1. Effects of MT and LRH on Testicular Growth and on Brain DA, NE, E, and 5-HT Levels (Table 1)

In this and all subsequent experiments, control birds not exposed to stimulatory light were found to have significantly higher brain monoamine levels than light stimulated ones. While this appears to be at variance with the findings of Campbell and Wolfson (1974) it is very likely due to the relative time in their respective photoperiod that these birds were sacrificed.

Treatment with MT produced a significant inhibition of testicular response to light and a marked depletion of DA, NE, and E levels in the brains of birds subjected to 14 h of light per day. Brain DA, NE, and E were reduced by an average of 79 percent, 62 percent and 55 percent but the 5-HT level was not significantly changed by such treatment. The MT-induced depletion of CA was associated with a significant inhibition of the testicular response to light stimulation.

LRH administration significantly increased testicular weight of MT treated birds even though CA and 5-HT levels in the brains of both groups were comparable.

Experiment 2. Effects of L-DOPA in MT-Treated Quail (Table 2)

In this experiment we sought to determine if L-DOPA, a precursor of CA could bypass the MT block of CA synthesis, increase brain CA levels and restore testicular growth.

As in Experiment 1, the MT-induced inhibition of amine synthesis caused the levels of brain DA, NE, and E to fall and the treatment decreased the light-stimulated testicular growth to 25 percent of the light-stimulated controls.

Administration of L-DOPA alone, caused a slight reduction in testicular growth of light stimulated birds. It also increased DA and E levels in the brain, whereas NE and 5-HT remained unchanged. L-DOPA treatment significantly increased testicular weight of MT treated birds (compare groups 3 and 5, Table 2) but the weights remained lower than those of the light stimulated control group.

Experiment 3. Effect of Preferential Depletion of NE and E on Testicular Growth (Table 3)

Inhibition of dopamine-B-oxidase (the enzyme necessary for the conversion of DA to

TABLE 1. Effects of MT and LRH on testis weight and brain monoamine concentrations of Coturnix quail.

Group	Treatment	Testis wt. (mg)	Monoamines (ug/g) DA	NE	E	5-HT
	Nonstimulated (6L:18D)					
1	Saline	12.9 ± 0.9[a]	1.325 ± 0.070[a]	0.978 ± 0.068[a]	0.314 ± 0.015[a]	2.233 ± 0.139[a]
	Light stimulated (14L:10D)					
2	Saline	221.1 ± 29.9[b]	0.865 ± 0.059[b]	0.757 ± 0.470[b]	0.268 ± 0.011[b]	1.775 ± 0.096[b]
3	MT	80.7 ± 4.6[c]	0.184 ± 0.024[c]	0.287 ± 0.017[c]	0.119 ± 0.018[c]	1.564 ± 0.179[bc]
4	MT + LRH	168 ± 14.8[d]	0.287 ± 0.056[c]	0.227 ± 0.007[c]	0.093 ± 0.007[c]	1.236 ± 0.137[c]

Results are expressed as means ± S.E. (8 birds per group). Monoamine concentrations were determined in brain tissue collected 3-4 h after final MT and/or LRH administration. Means within a column with different superscripts are different at the 5% level of probability.

TABLE 2. Antagonism by L-DOPA of MT effects on brain monoamine concentrations and testis weight of Coturnix quail.

Group	Treatment	Testis wt. (mg)	Monoamines (ug/g) DA	NE	E	5-HT
	Nonstimulated (6L:18D)					
1	Saline	16.2 ± 0.4[a]	0.999 ± 0.051[a]	0.976 ± 0.053[a]	0.332 ± 0.025[ab]	2.145 ± 0.089[c]
	Light stimulated (14L:10D)					
2	Saline	179.5 ± 42.0[b]	0.811 ± 0.059[b]	0.797 ± 0.035[b]	0.294 ± 0.021[a]	1.385 ± 0.093[a]
3	MT	44.4 ± 13.1[a]	0.333 ± 0.035[c]	0.397 ± 0.019[c]	0.098 ± 0.009[c]	1.493 ± 0.143[ab]
4	L-DOPA	141.1 ± 14.1[c]	0.976 ± 0.055[a]	0.811 ± 0.042[b]	0.363 ± 0.013[b]	1.281 ± 0.122[a]
5	MT + L-DOPA	80.5 ± 4.9[d]	0.536 ± 0.028[d]	0.587 ± 0.028[d]	0.158 ± 0.014[d]	1.844 ± 0.173[bc]

Results are expressed as means ± S.E. (8 birds per group). Monoamine concentrations were determined in brain tissue collected 3-4 h after final MT and/or L-DOPA administration. Means within a column with different superscripts are different at the 5% level of probability.

TABLE 3. Effects of DDC on brain monoamine concentrations and testis weight of Cotumix quail.

Group	Treatment	Testis wt. (mg)	Monoamines (ug/g) DA	NE	E	5-HT
	Nonstimulated (6L:18D)					
1	Saline	15.1 ± 0.9[a]	0.983 ± 0.059[a]	0.902 ± 0.035[a]	0.359 ± 0.022[a]	2.027 ± 0.089[a]
	Light stimulated (14L:10D)					
2	Saline	101.0 ± 5.1[b]	0.811 ± 0.041[b]	0.661 ± 0.020[b]	0.308 ± 0.020[a]	1.534 ± 0.184[b]
3	DDC	43.6 ± 6.1[c]	1.017 ± 0.059[a]	0.368 ± 0.028[c]	0.174 ± 0.008[b]	1.495 ± 0.090[b]

Results are expressed as means ± S.E. (8 birds per group). Monoamine concentrations were determined in brain samples collected 3-4 h after final DDC administration. Means within a column with different superscripts are different at the 5% level of probability.

NE) by DDC caused a 44 percent reduction in both NE and E, while it increased DA levels by 25 percent. 5-HT levels remained unchanged in DDC treated quail. The DDC-induced depletion of central NE and E and elevation of dopamine were accompanied by a significant reduction in growth of the testes.

Experiment 4. Restoration of NE in DDC-Treated Quail by DL-DOPS (Table 4)

In this experiment the DDC block of NE and E was bypassed by providing an alternate precursor for their synthesis (DL-DOPS).

Again, as in Experiment 3, injection of DDC into light stimulated quail produced a marked depletion in brain NE and E levels and an increase in DA. These changes were associated with testicular growth inhibition. In the absence of DDC, DL-DOPS administration caused the DA level to fall 41 percent, while NE and E levels remained unchanged. These quail, in which DA was preferentially depleted, showed augmented testicular growth in comparison to the corresponding saline treated controls. Administration of DL-DOPS to DDC treated quail, resulted in restoration of NE, but not E, essentially to control levels. Multiple mean comparisons revealed that the testicular weight of the DDC + DL-DOPS-treated group was significantly greater than that of DDC-treated birds.

DISCUSSION

The results of the present study, obtained in quail treated with drugs that modify brain CA, suggest that central monoamines play a role in the mediation of photoperiodically-induced testicular development.

Treatment with MT, an inhibitor of tyrosine hydroxylase (Spector et al., 1965) induced a marked decrease of brain DA, NE, and E levels which was associated with significant reduction in testicular growth. Both MT and DDC reduced photoperiodically-induced gonadal stimulation. However, the two drugs have a different mode of action: while MT blocks the first enzymatic step (tyrosine-DOPA) of CA biosynthesis, DDC inhibits dopamine-B-oxidase (Carlsson et al., 1966). By using DDC, NE and E could be depleted while normal DA levels were maintained (Tables 3 and 4).

The presence of CA and 5-HT-containing neurons have been established in the brain of birds (see introduction). Moreover, there are

TABLE 4. Antagonism of DDC effects on brain monoamine concentrations and testis weight of Coturnix quail by DL-DOPS.

Group	Treatment	Testis wt. (mg)	Monoamines (ug/g) DA	NE	E	5-HT
1	Saline	115.9 ± 11.2[b]	0.961 ± 0.065[b]	0.646 ± 0.049[a]	0.318 ± 0.02[a]	1.351 ± 0.037[b]
2	DDC	26.7 ± 3.3[a]	1.244 ± 0.117[c]	0.252 ± 0.020[b]	0.151 ± 0.021[b]	1.244 ± 0.081[bc]
3	DL-DOPS	145.7 ± 14.9[c]	0.568 ± 0.089[a]	0.716 ± 0.048[a]	0.310 ± 0.02[a]	1.155 ± 0.033[c]
4	DDC + DL-DOPS	94.9 ± 10.2[b]	1.035 ± 0.049[b]	0.593 ± 0.045[a]	0.174 ± 0.018[b]	1.219 ± 0.057[bc]

Results are expressed as means ± S.E. (6 to 8 birds per group). Brain monoamine concentrations were determined in brains collected 3-4 h after final DDC and/or DL-DOPS administration. Means within a column with different superscripts are different at the 5% level of probability.

indications that the synthesis and turnover of CA is dependent on the flow of nervous impulses in central monoamine neurons (Anden et al., 1966; Anden et al., 1971). Thus, a disruption of central mechanism(s) involving CA-containing neurons which could influence testicular growth in response to stimulatory photoperiodic schedule seemed probable.

It seems unlikely that a direct action of MT on the pituitary, gonads or peripheral nerves might be responsible for the decreased gonadal weight. This is supported by the observation that the testes of MT-treated quail responded to LRH at a time when brain CA were depleted. The testes are not directly responsive to this hormone, since it has no effect on testis weight of quail held on a nonstimulatory light regime (unpublished observations). Similarly, a toxic effect of the drugs is unlikely to play a significant role in the present study since CA precursors, L-DOPA and DL-DOPS prevented the effects of monoamine inhibitors and restored testicular growth and no detrimental effect on body weights were seen.

Restoration of CA levels in the brain, after their previous depletion by MT and DDC treatments, and the concomitant increase in testis weight gives additional evidence for the specificity of the effect observed. L-DOPA treatment in MT-treated birds partially restored brain CA levels and testicular weight. The observed inability of L-DOPA to completely restore brain CA to control values might be explained by earlier findings that much of the systemically administered L-DOPA is converted into peripheral CA, which are much diminished when combined with inhibitors of extracerebral decarboxylase (Bartholini and Pletscher, 1968; Butcher et al., 1972). Normalization of central NE level, as a consequence of the action of DL-DOPS (a precursor of NE) given at the same time as DCC is in accord with previous findings in the rat (Creveling, 1968). Such treatment essentially prevents the blocking effect of DDC on photoperiodically-induced testicular stimulation.

Although the present findings suggest that brain CA participates in the mechanism(s) of gonadal development in Coturnix, identification of the specific amine which mediates the photoperiodically-induced gonadal growth is complex. Due to the difficulties in interpreting the observed changes in central monoamines, the implications that can be drawn from their analyses are speculative at best.

The involvement of brain NE in mediating light-induced testicular stimulation is substantiated by the results with the NE precursor, DL-DOPS. Thus DL-DOPS selectively restored brain NE levels, previously diminished by administration of DDC, and also prevented the testicular inhibition induced by the latter compound. Moreover, the observation that DL-DOPS administration did not reverse the DDC depletion of E, further favors the view that a noradrenergic activating mechanism intervenes in the process of gonadal growth. It might be argued, however, that the increased testicular weight in response to stimulatory light is caused by an inactivation of central dopaminergic mechanisms. The arguments for this view are based on findings that light stimulated testicular growth was significantly reduced by L-DOPA treatment at a time when brain DA levels were increased above the controls, and NE remained unchanged (Table 2; group 4). In addition, treatment with DL-DOPS depleted brain DA levels, but augmented the testicular response to light stimulation (Table 4; group 3). However, if light induces testicular development by interfering with dopaminergic neurons only, testicular weight of MT treated birds should have been increased over the control quail, since MT markedly depleted the endogenous DA level. Testicular inhibition rather than stimulation was produced by such treatment (Tables 1 and 2).

In view of the failure of reduced DA level in MT-treated birds to augment or even to maintain normal testicular growth at time when NE is reduced, and since L-DOPA will partially reverse the MT-induced testicular block while both NE and DA levels increase, it appears that the presence of an intact positive noradrenergic mechanism is essential in mediating the photoperiodically-induced gonadal stimulation. Whether dopaminergic neurons exert a restraining influence over such processes requires further investigation. There are indications that DA has an inhibitory effect on gonadotropic function in both rabbit and rat (Sawyer et al., 1974; Fuxe et al., 1972a; Fuxe et al., 1972b).

Since the testicular weight increase following exposure to a stimulatory photoperiod in quail is due to an increased gonadotropin secretion (Follett et al., 1972; Nicholls et al., 1973), our findings imply that a disruption of central noradrenergic mechanisms and/or augmentation of dopaminergic mechanisms block gonadotropin secretion. This may occur by influencing the release of hypothalamic releasing substances (Follett and Sharp, 1969; Smith and Follett, 1972). Indeed, it has been reported that hypothalamic NE level shows variations associated with luteinizing hormone releasing factor activity in light stimulated quail (Campbell and Wolfson, 1974) and LH release in the chicken (Graber and Nalbandov, 1972), whereas 6-hydroxydopamine inhibits LH release in light stimulated quail (Davies and Follett, 1974). Such a concept, however, needs to be substantiated by further work involving the determination of catecholaminergic activity during various phases of gonadotropic hormone secretion.

REFERENCES

Anden, N.-E., Fuxe, K. and Hokfelt, T. (1966). The importance of the nervous impulse flow for the depletion of the monoamines from central neurons by some drugs. J. Pharm. Pharmacol. 18, 630-632.

Anden, N.-E., Corrodi, H., Fuxe, K. and Ungerstedt, U. (1971). Importance of nervous impulse flow for the neuroleptic induced increase in amine turnover in central dopamine neurons. Europ. J. Pharmacol. 15, 193-199.

Anton, A. H. and Sayre, D. F. (1962). A study of the factors affecting the aluminum oxide-trihydroxyindole procedure for the analysis of catecholamines. J. Pharmacol. Exptl. Therap. 138, 360-375.

Anton, A. H. and Sayre, D. F. (1964). The distribution of dopamine and dopa in various animals and methods of their determination in diverse biological material. J. Pharmacol. Exptl. Therap. 145, 326-336.

Barchas, J., Erdelyi, E. and Angwin, P. (1972). Simultaneous determination of indole- and catecholamines in tissues using a weak cation-exchange resin. Anal. Biochem. 50, 1-17.

Bartholini, G. and Pletscher, A. (1968). Cerebral accumulation and metabolism of ^{14}C-dopa after selective inhibition of peripheral decarboxylase. J. Pharmacol. Exptl. Therap. 161, 14-20.

Bogdanski, D. F., Pletscher, A., Brodie, B. B. and Udenfriend, S. J. (1956). Identification and assay of serotonin in brain. J. Pharmacol. Exptl. Therap. 117, 82-88.

Butcher, L. L., Engel, J. and Fuxe, K. (1972). Behavioral, biochemical and histochemical analyses of the central effects of monoamine precursors after peripheral decarboxylase inhibition. Brain Res. 41, 387-411.

Calas, A., Hartwig, H.-G. and Collin, J. P. (1974). Noradrenergic innervation of the median eminence. Microspectrofluorimetric and pharmacological study in the duck. *Anas platyrhynchos*. Z. Zellforsch. 147, 491-504.

Campbell, G. T. and Wolfson, A. (1974). Hypothalamic norepinephrine, luteinizing hormone releasing factor activity and reproduction in the Japanese quail, Coturnix coturnix japonica. Gen. Comp. Endocr. 23, 302-310.

Carlsson, A., Lindquist, M., Fuxe, K. and Hokfelt, T. (1966). Histochemical and biochemical effects of

diethyldithio-carbamate on tissue catecholamines. J. Pharm. Pharmacol. 18, 60-62.

Creveling, C. R., Daily, J., Tokuyama, T. and Witkop, B. (1968). The combined use of alpha-methyltyrosine and three-dihydroxyphenylserine-selective reduction of dopamine levels in the central nervous system. Biochem. Pharmacol. 17, 65-70.

Davies, D. T. and Follett, B. K. (1974). The effect of intraventricular administration of 6-hydroxydopamine on photo-induced testicular growth in Japanese quail. J. Endocr. 60, 277-283.

Follett, B. K., Scanes, C. G. and Nicholls, T. J. (1972). The chemistry and physiology of the avian gonadotrophins. In: Hormones Glycoproteigues Hypophysaires, p. 192. Ed. M. Jutisz. Inserm, Paris.

Follett, B. K. and Sharp, P. J. (1969). Circadian rhythmicity in photo-periodically induced gonadotrophin release and gonadal growth in the quail. Nature 223, 968.

Fuxe, K., Hokfelt, T. and Nilsson, O. (1972a). Effect of constant light and androgen-sterilization on the amine turnover of the tubero-infundibular dopamine neurone: Blockade of cyclic activity and induction of a persistent high dopamine turnover in the median eminence. Acta Endocr. 69, 625-639.

Fuxe, K., Hokfelt, T., Sundstedt, C.-D., Ahrin, K. and Hamberger, L. (1972b). Amine turnover changes in tubero-infundibular dopamine (DA) neurons in immature rats injected with PMS. Neuroendocrinology 10, 282-300.

Graber, J. W. and Nalbandov, A. V. (1972). Relationship of hypothalamic catecholamines and gonadotrophin levels in the chicken. Neuroendocrinology 10, 325-337.

Homma, K. and Sakakibara, Y. (1971). Encephalic photoreceptors and their significance in photoperiodic control of sexual activity in Japanese quail. In: Biochronometry, pp. 333-341. Ed. M. Menaker. National Academy of Sciences, Washington, D.C.

Ikeda, H. and Gotoh, J. (1971). Distribution of monamine containing cells in the central nervous system of the chicken. Jap. J. Pharmacol. 21, 763-784.

Menaker, M. (1971). Rhythms, reproduction and photoreception. Biol. Reprod. 4, 295-308.

Nicholls, T. J., Scanes, C. G. and Follett, B. K. (1973). Pituitary and plasma luteinizing hormone in Japanese quail during photoperiodically induced gonadal growth and regression. Gen. Comp. Endocr. 21, 84-98.

Sawyer, C. H., Hilliard, J., Kanematsu, S., Scaramuzzi, R. and Blake, C. A. (1974). Effects of intraventricular infusions of norepinephrine and dopamine on LH release and ovulation in the rabbit. Neuroendocrinology 15, 328-337.

Sharp, P. J. and Follett, B. K. (1969). The effect of hypothalamic lesions on gonadotrophin release in Japanese quail (Coturnix coturnix japonica). Neuroendocrinology 5, 205-218.

Sharp, P. J. and Follett, B. K. (1968). The distribution of monoamines in the hypothalamus of the Japanese quail, Coturnix coturnix japonica. Z. Zellforsch 90, 245-262.

Snedecor, G. W. and Cochran, W. G. (1967). Statistical methods. Iowa State University Press, Ames, Iowa.

Smith, P. M. and Follett, B. K. (1972). Luteinizing hormone releasing factor in the quail hypothalamus. J. Endocr. 53, 131-138.

Spector, S., Sjoerdsma, A. and Udenfriend, S. (1965). Blockade of endogenous norepinephrine synthesis by alpha-methyltyrosine, an inhibitor of tyrosine hydroxylase. J. Pharmacol. Exptl. Therap. 147, 86-95.

Warren, S. S., Farner, D. S. and Oksche, A. (1973). Fluorescence microscopy of neurons containing primary catecholamines in the ventral hypothalamus of white-crowned sparrow, Zonotrichia leucophyrys gambelii. Z. Zellforsch 141, 1-17.

RECOMMENDED REVIEWS

Lofts, B. and Lam, W. L. (1973). Circadian regulation of gonadotrophin secretion. J. Reprod. Fert., Suppl. 19, 19-34.

Kordon, C. and Glowinski, J. (1972). Role of hypothalamic monoaminergic neurones in the gonadotrophin release-regulating mechanisms. Neuropharmacology 11, 153-162.