Effect of Prolactin and Progesterone on Gonads of Breeding California Quail (33831)

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Although much is known about avian reproductive endocrinology (1), the hormonal influences involved in the transition of the sexually active bird (egg laying female, sperm producing male) to the sexually inactive, parental bird are poorly understood. Ever since Riddle and his co-workers found that prolactin stopped egg laying in the domestic fowl and pigeon, produced incubation behavior in hens, and caused collapse of the testes in roosters (2), the general opinion has been that this hormone plays a causative role in this transition. However, further work on the same and other species has not always substantiated this opinion (3). Lehrman (4) and others have implicated progesterone as the hormone which produces incubation behavior in the domestic pigeon, and this hormone also stops egg laying in the domestic fowl if administered early in the ovulatory cycle (1). The purpose of the present study was to determine the effects of prolactin and progesterone on the gonads of breeding California quail (*Lophortyx californicus*) and to compare these results with those obtained in other bird species.

Materials and Methods. The 18 quail pairs used in this experiment were yearling or adult birds obtained from Poisal's Rare Bird Farm, Pleasanton, California, in late February, 1965. They were housed as pairs in small (2 $\times$ 2 $\times$ 1 ft) wire-mesh cages, supplied with food (Purina game chow Layena) and water *ad libitum,* and subjected to artificial 15L–9D photoperiod. The females began laying eggs after about 40 days, and the hormone treatments were begun after each hen had laid approximately 5 eggs. The males were producing sperm at the time of hormone administration or immediately previous to it as indicated by the fertility of their mates' eggs.

The pairs were divided into three groups of 6 pairs each: group 1 (control) received 0.2 ml of saline; group 2 received 0.5 mg of progesterone in 0.2 ml of saline; group 3 received 15 IU of prolactin (NIH-P-S8) in 0.2 ml of saline. All injections were administered intramuscularly daily for 12 days at about the fourth hour of light, and a record was kept of eggs laid.

After sacrificing the birds on day 13, the ovaries, oviducts, and left testes were fixed in 10% neutral-buffered formalin, blotted dry, and weighed on a Mettler balance. The diameters of the 3 largest ovarian follicles and the length and width of each testis were measured to the nearest 0.1 mm with a dial micrometer. Testis volume was calculated using the formula for the volume of an ellipsoid. The testes were then embedded in paraffin, sectioned at 6 μ and stained with Delafield's acid alum–hematoxylin and eosin; the stage of spermatogenesis was recorded using the criteria of Lewin (5), except that his "early" and "late" regression stages were termed stages 6 and 7 in this study. The longest diameters of 30–50 interstial cell nuclei were measured at random under oil immersion with an ocular micrometer. Also, in order to estimate the condition of the gonaduct complex, the height of the epididymal epithelium was measured in 15–20 areas selected at random.

Results. The control breeding females laid one egg a day throughout the experiment. Prolactin stopped laying in one hen, interrupted laying in two hens, and had no effect in three hens. The follicle diameters and ovary and oviduct weights of the prolactin-treated and control hens did not differ significantly (Table I). Progesterone halted laying in four of six females, interrupted laying in one, and had no effect in another. The ovary

TABLE I. Effect of Prolactin and Progesterone on Ovaries and Oviducts of Quail.

Treatment	No. of birds	Ovary wt (g)[a]	Pollicle diameter (mm)[a]	Oviduct wt (g)[a]
Saline	6	5.17 ± 0.61	13.2 ± 0.6	6.44 ± 1.34
Prolactin	6	5.95 ± 1.00	13.4 ± 1.4	6.37 ± 0.56
Progesterone	6	2.43 ± 0.47[b]	8.7 ± 1.0[b]	5.02 ± 0.50

[a] Mean ± SE mean.
[b] Differ significantly from control values ($p < .01$).

weights and follicle diameters of the progesterons-treated hens were significantly smaller than control values (Table I). The mean oviduct weight of the progesterone—treated females was smaller than that of the controls, but the difference was not significant.

The testes of three of the control breeding males were in the initial stage of regression (stage 6), presumably because of chronic exposure to artificial long photoperiod. Prolactin may have caused a slight increase in the rate of regression, because 2 of the 6 birds had testes in stage 7. However, this hormone had no effect on testis volume, interstitial cell nuclear diameter, or epididymidal epithelial height (Table II). On the other hand, the values for interstitial cell nuclear diameter were significantly lower than the control values in the progesterone-treated males (Table II). Because only one of the testes of the latter group was regressing, the other being in stages 4 or 5, it appears that progesterone stimulated spermatogenesis but had a negative effect on interstitial cell nuclear size. The heights of the epididymidal epithelium tended to be smaller in the progesterone-treated birds, but the values were not significantly different from control values (Table II).

Discussion. Evidence gained from experiments on the domestic fowl and pigeon indicate that prolactin inhibits egg laying with resultant follicular atresia (6, 7). However, prolactin only interrupted but did not stop egg laying in the quail. Similarly, Juhn and Harris (8) found that, in domestic fowl, prolactin blocked the antagonistic effect of progesterone on egg laying and only temporarily interrupted laying when given alone. Several hormones (e.g., thyroxin, progesterone, growth hormone) injected into laying domestic fowl will stop egg laying (9), but these materials may play no role in the termination of ovulation in the natural situation. Therefore, the data obtained from early experiments using high doses of relatively impure prolactin must be interpreted with caution.

Prolactin inhibits testis activity in some birds but not others (10). When this hormone does antagonize testis activity, it presumably operates by inhibiting pituitary gonadotropin secretion, because FSH given simultaneously prevents testis collapse (7, 11, 12). However, there was no evidence in the present study that prolactin has an antagon-

TABLE II. Effects of Prolactin and Progesterone on Testis and Epididymis of Breeding Male Quail.

Treatment	No. of birds	Left testis volume (mm^3)[a]	Stage of spermatogenesis[b]	Interstitial cell nuclear diameter (μ)[a]	Height of epididymidal epithelium (μ)[a]
Saline	6	607.6 ± 73.9	5.5 (5–6)	5.00 ± 0.23	11.1 ± 1.7
Prolactin	6	680.2 ± 99.1	6.0 (5–7)	4.70 ± 0.34	11.4 ± 3.4
Progesterone	6	443.6 ± 93.6	4.7 (4–6)	4.23 ± 0.13[c]	7.8 ± 0.1

[a] Mean ± SE mean.
[b] Mean (range); stage 4: spermatids; stage 5: sperm; stage 6: early regression; stage 7: mid-regression, after Lewin (5).
[c] Differ significantly from control values ($p < .02$).

istic effect on the quail testis of the magnitude seen in other birds. The prolactin injected into both the males and females was physiologically active, at least in regards to the induction of incubation patch formation (13). Prolactin also has no measurable effect on the testes of breeding bobwhite quail, *Colinus virginianus* (Jones, unpublished). Recently, Meier and Dusseau (10) claimed that prolactin antagonizes testis activity in nonmigratory but not in migratory species and subspecies. The California quail is not migratory; nevertheless, exogenous prolactin did not antagonize testis activity in this species. Hence, it is difficult to generalize about the "antigonad" effect of prolactin in birds.

In domestic fowl, progesterone administration inhibits egg laying if given early in the 24-hr ovulatory cycle, but if given late in the cycle this hormone causes release of LH and premature ovulation (1). Because, in this study, all of the injections were given early in the daily ovulatory cycle, it was not surprising that this steroid stopped ovulation. The smaller ovary weights and follicle diameters in the progesterone-treated hens may indicate that this hormone inhibited FSH as well as LH release. However, a direct antagonistic effect of progesterone on the ovary cannot be ruled out. Cycling levels of progesterone play an important role in the ovulatory cycle of domestic fowl (14). Validation of this concept in quail would require further study of the effects of this hormone in relation to dose levels and time of administration.

The injection of progesterone into breeding male quail stimulated spermatogenesis but inhibited interstitial cell activity. However, it was not possible to determine if these effects were the result of a direct effect on the testis or an indirect effect on pituitary gonadotropin release.

Summary. Prolactin and progesterone were administered to egg laying female and sperm producing male California quail (*Lophortyx californicus*) in order to ascertain the effects of these hormones on gonad function. In females, prolactin interrupted but did not halt egg laying and had no effect on ovary and oviduct weight and follicle diameter. Progesterone, on the other hand, halted egg laying and decreased ovary weight and follicle diameter. In males, prolactin had no measurable effect on testis activity, but progesterone stimulated spermatogenesis and decreased interstitial cell nuclear diameter.

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