

## Effect Of Supplementary Doses Of Luteinizing Hormone And Estradiol On Female Grey Quail, *Coturnix-Coturnix*

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### Abstract

Thirty female birds were segregated from the stock and divided into five groups of six birds each. Birds were injected intramuscularly with two different doses of luteinizing hormone (LH) (0.167mg and 0.334 mg) and estradiol (0.167mg and 0.33 mg) for ten days. It was found that 0.167 mg LH even though stimulated the growth of largest follicle it was not upto any significance where as the same dose of estradiol significantly inhibited body weight and development of follicle of ovary. Treatment with LH and estradiol resulted in the inhibition of pituitary ovary during the reproductive phase of grey-quail.

**Key words :** Hormone, follicle, estradiol, inhibition.

### Introduction

Estrogens are very effective inhibitors of pituitary gonadotrophin secretion but studies in female rats by Docke *et al.*, (1984b) strongly suggest that estradiol itself can induce desensitization to its suppressive effect on gonadotrophin release. This estrogen induced desensitization of the negative feed back of estrogen, which is probably required for the control of final maturation of the preovulatory follicles does not seem to be operative in male rats (Docke *et al.*, 1988). Reproduction in females is regulated by hormones (Christensen *et al.*, 2012). Birds lay eggs with significant amounts of yolk androgens which are transferred during the rapid growth of ovarian follicles (Okuliarova *et al.*, 2010).

Effects of estradiol on different mechanisms is well established in mammals (Rozell and Keisler 1990) and Japanese quail (Somogyiova *et al.*, 1983). Administration of LH in rat causes initiation of follicular maturation during mid-pregnancy (Taya and Sasamoto 1989). In female quail LH, LHRH or progesterone had induced ovulation and oviposition (Onagbesan and Peddie 1988). Inter female differences are associated with environmental and social factors encountered by the female during the breeding season, such as mating partner (Garcia - Fernandez *et al.* 2010), breeding density (Pilz and Smith, 2004), immunological loads (Gil *et al.*, 2006), food availability (Benowitz-Fredericks *et al.*, 2013) and stressful events (Henriksen *et al.*, 2011, Okuliarova *et al.*, 2010) and with female's age as well (Guibert *et al.*, 2012). Gonadotropin-releasing hormone (GnRH) is largely responsible for the initiation of sexual behaviors; one form of GnRH activates a physiological cascade causing gonadal growth and gonadal steroid feedback to the brain, and another form is thought to act as a neurotransmitter to enhance sexual receptivity. In contrast to GnRH, gonadotropin-inhibitory hormone (GnIH) inhibits gonadotropin release (Bentley *et al.* 2006). In this experiment the effect of LH and estradiol was studied on body weight and diameter of largest follicle of ovary for a period of ten days.

**Table 1 : Different groups, treatments and total doses**

Group	Treatment (Dose/bird/day)	Total dose in 10 days
Group I	Control group 0.1 ml sesame oil	1 ml sesame oil
Group II	0.167 mg LH in 0.1 ml sesame oil	1.67 mg LH
Group III	0.334 mg LH in 0.1 ml sesame oil	3.34 mg LH
Group IV	0.167 mg estradiol	1.67 mg estradiol
Group V	0.334 mg estradiol	3.34 mg estradiol

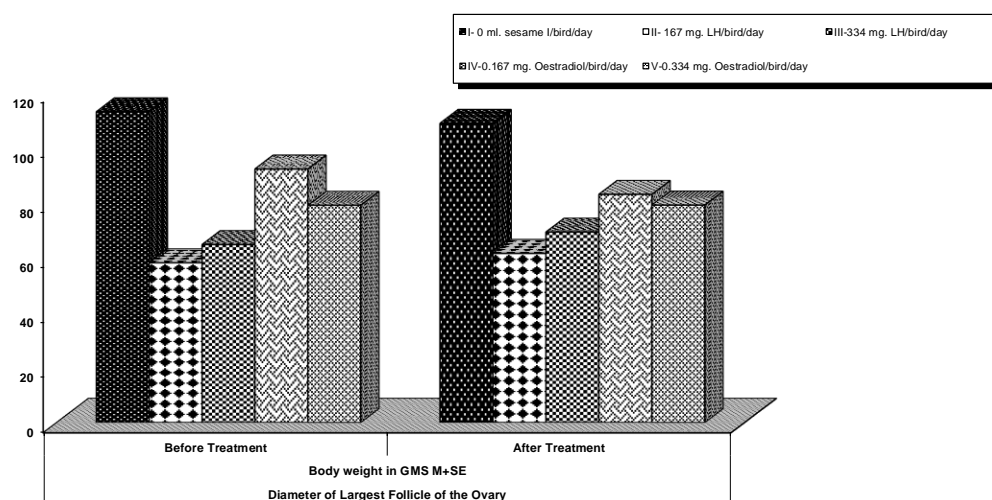
### Materials and Methods

Birds were purchased locally and acclimatized to laboratory conditions for over a period of four weeks. Thirty female birds were separated, tagged and weighed individually and were laparotomized to note the diameter of largest follicle of ovary. These birds were divided into five groups comprising six birds in each group. They were kept into iron wire net cages and treated as explained in Table 1. Birds were injected intramuscularly for ten days during morning hours. They were fed with mixed feed and water *ad libitum*. The birds were sacrificed by decapitation.

### Results :

#### Body weight

Fig. 1 and Table-2 shows the graphical and tabular representation of body weight before and after treatment. Marginal ( $P > 0.025$ ) decrease (group 1) and increase (group III) was found on comparing post treatment vs pre treatment. Significant reduction ( $P > 0.005$ ) was observed in group IV. No apparent difference was noted in group II and V. It can be concluded from the results that both the doses of LH increased the body weight.



**Fig. 1 Showing the effect of Luteinizing hormone and Estradiol on mean body weight of female Grey quail, *Coturnix coturnix***

**Diameter of largest follicle of the ovary**

Slight changes were found in group I and II after treatment when compared to pre treatment (Fig. 2 and Table 2) No alteration was found in group III and V. Fast decrease ( $P > 0.005$ ) was observed in 0.167 mg estradiol treated birds.

**Table 2 :** Showing the effect of Luteinizing hormone and Estradiol on mean body weight and gonad size (Diameter of the largest follicle of the ovary) of female Grey quail, *Coturnix coturnix*.

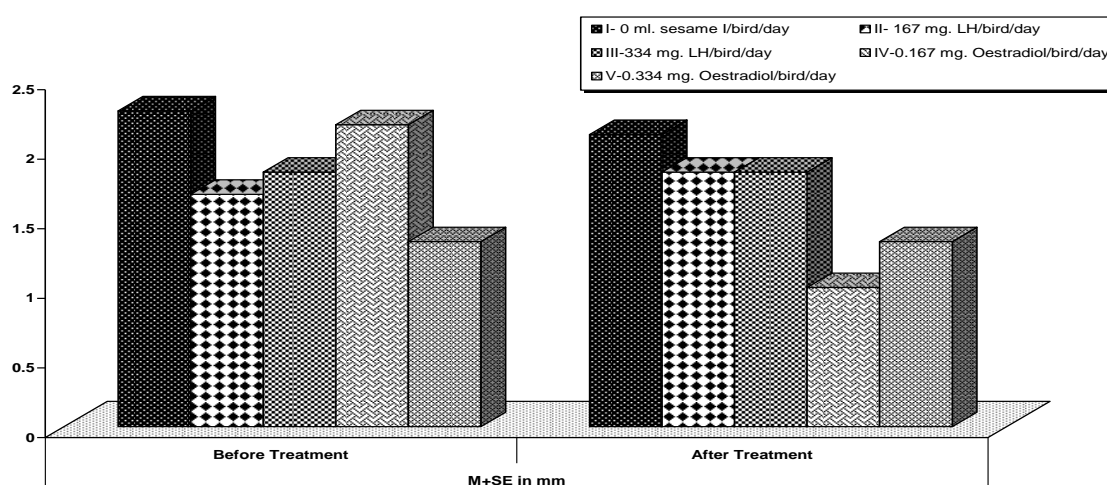
Groups	Body weight in GMS $M \pm SE$		Diameter of Largest Follicle of the Ovary in mm $\pm SE$	
	Before Treatment	After Treatment	Before Treatment	After Treatment
I 0.1 ml. sesame oil /bird/day	112.87 $\pm 4.24$	108.67 $\pm 0.75^*$	2.27 $\pm 0.14$	2.10 $\pm 0.24$ NS
II 0.167 mg. LH/bird/day	58.00 $\pm 7.29$	61.43 $\pm 2.22$ NS	1.67 $\pm 0.14$	1.83 $\pm 0.14$ NS
III 0.334 mg. LH/bird/day	64.63 $\pm 3.46$	69.17 $\pm 2.52^*$	1.83 $\pm 0.14$	1.83 $\pm 0.27$
IV 0.167 mg. Estradiol/bird/day	92.07 $\pm 3.67$	82.87 $\pm 1.97^{***}$	2.17 $\pm 0.14$	1.00 $\pm 0.24^{***}$
V 0.334 mg. Estradiol/bird/day	78.90 $\pm 2.55$	78.97 $\pm 2.14$ NS	1.33 $\pm 0.14$	1.33 $\pm 0.14$

Values expressed as Mean  $\pm$  SE

Number of birds in each group is 6.

\* $P > 0.025$ , \*\* $P > 0.01$ , \*\*\* $P > 0.005$ , NS = Not significant

Significant test - Before treatment Vs. After treatment



**Fig. 2:** Showing the effect of Luteinizing hormone and Estradiol on mean gonad size (Diameter of the largest follicle of the ovary) of female Grey quail, *Coturnix coturnix*.

**Discussion :**

It has been well documented that estradiol is the active metabolite to induce male sexual behaviour in mammals (Christensen and Clemens 1974,) and also in birds (Adkins 1975). Activation of quail mating behaviour by TP was blocked by concurrent administration of the antiestrogens.

All these studies indicate that estradiol is the effective metabolite of testosterone in the central nervous system.

During breeding sex hormones control female reproductive physiology and behaviour through the hypothalamic - pituitary-gonadal (HPG) axis (Johnson 2015).

LH releasing hormone has been shown to stimulate reproductive activity in some species of birds as shown by an increase in plasma LH levels in Japanese Quails (Bicknell and Follett 1975). Studies also indicate antifertility effects of LH releasing hormone or its agonists in various species of mammals. High dose of LH releasing hormone caused premature termination or inhibition of pregnancy in rat (Bex and Corbin, 1981). Change in the sensitivity of pituitary to LH releasing hormone has been reported during different stages of reproduction in many species of birds (Balthazart *et al.* 1980) and sexual maturation of rat.

In avian species, ovulation is regulated by a pre-ovulatory surge of reproductive hormones, mainly by the positive feed back between LH and progesterone (Johnson *et al.*, 1985). Quail lay eggs in the afternoon (Houdelier *et al.*, 2002) in contrast to hens, which usually lay eggs in the morning. The pre ovulatory peak of LH in Japanese quail controls deposition of maternal testosterone in egg (Okuliarova *et al.*, 2018). Estradiol plays important role in the activation of reproductive behaviour in quail (Balthazart *et al.*, 2009).

Results of the present study indicated that body weight increased in LH treated birds where as 0.167 mg estradiol decreased the body weight and diameter of largest follicle of the ovary.

As the experiment was conducted during breeding phase of the birds it might be possible that during this period hormones secreted by the ovary made the pituitary highly responsive to LH which resulted in increased gonadotrophin secretion and faster rate of ova growth. It seems that both hormonal treatment produced inhibitory effect. Inhibitory effects of LH releasing hormone have not so far been reported in sub mammalian species, but in some mammalian species a few reports show its antifertility effects (Bambino *et al.* 1980). The mechanism of antifertility action of LH releasing hormone is not very clear. It may be due to the formation of antibodies (auto-immunization). It is, therefore, possible that in this bird too, some such mechanism operates and causes antigonadal effects but it mainly appears due to changes in pituitary functions rather than direct effect on ovary.

Breneman (1955) investigated the effects of injections of 0.5, 1.0, 5.0 and 25.0 µg estradiol per day for ten days on the ovary of 30 days old pullets and found no significant difference from control ovarian weight.

Phillips (1959) injected 12.5 mg DES per week into 6 week old pullets and obtained a 30% increase in ovarian weight ( $P < 0.01$ ). Similarly, 10 mg DES per day given to adult, on breeding black ducks, *Atlas platyrhynchos* resulted in a 95% increase in ovarian weight ( $P < 0.1$ ) Chu and You (1946) also failed to induce maturation of follicles by estrogen injections in hypophysectomized pigeons. It is not clear whether any stimulation of ovarian weight occurred or not. Estrogen causes gonadotrophin inhibition by way of the hypothalamus in mammals.

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