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47th Annual Conference
(JOS 2022)

CONFERENCE PROCEEDINGS

THEME
SECURING ANIMAL AGRICULTURE AMIDST GLOBAL CHALLENGES

HISTOLOGICAL RESPONSES OF THE SHELL GLAND IN LOHMANN BROWN LAYERS ADMINISTERED WITH CRYSTALLINE PROGESTERONE

Ahmed, B.

Department of Animal Science, Umaru Musa Yar'adu University Katsina, P.M.B 2218

Corresponding Author:

Bishir Ahmed

+2347030286505

ABSTRACT

The study was conducted to determine the effect of Crystalline Progesterone (CP) on shell gland histology in eighteen Lohmann Brown layers. Completely randomized design was used with each treatment (0, 5, 10, 15, 20 and 25 mg per bird) administered intramuscularly via the breast muscle and replicated thrice for six weeks. Result revealed similar magnitude (>10 fat cell aggregates) of shell gland fat infiltration between 10 and 15 mg CP treatment groups. It also revealed non atrophic change at all doses. Beyond 15 mg CP, the magnitude of shell gland fat infiltration decreased to 6-10 fat cell aggregates and subsequently remained the same. Shell gland in the 5 mg CP group had no fat infiltration when compared to those in the control group which had 1-3 fat cell aggregates. It has been concluded that, CP affected, shell gland fat infiltration, Shell gland

Keyword: Layers, progesterone. Shell gland

INTRODUCTION

Productivity is the key to growth and reproduction status of our farm animals (Verma *et al.*, 2012). Sexual maturity and egg production in birds is controlled by many factors including hormones especially oestrogen that plays important role in reproductive performance while progesterone is related to ovulation process (Rozenboim *et al.*, 2004; Rozenboim *et al.*, 2007). Synthetic hormones have been used in animal agriculture to improve reproduction and performance (Ledda *et al.*, 1999). Despite the importance of progesterone in reproduction it is exogenous administration to laying birds were found to cause yolk to pass the shell gland thereby producing soft shelled or shell-less egg. This is the major economic problem of the poultry industry that is egg shell quality which steadily deteriorates after few months of egg production (NVRI, 1994). The specific objective of the study is to assess changes in shell gland histology induced by crystalline progesterone administration in Lohmann Brown laying hens.

MATERIAL AND METHODS

Experimental birds and their management

A total of eighteen 24-week old Lohmann Brown strain of layers were purchased from Sovet International Farm Limited, Tarauni, Kano. They were managed in battery cages at the Poultry unit of the Teaching and Research Farm of the Department of Animal Science, Faculty of Agriculture, Bayero University Kano (GPS Coordinates: 11.97643°N, 008.42995°E), the hens fed and watered *ad libitum* with. The birds were allowed to acclimatize to the new environment for two weeks before commencement of the experiment.

Experimental design progesterone administration and histological processing

The experiment was laid in a single factor completely randomized design with six treatment groups replicated three times. Six treatments were assigned as 0, 5, 10, 15, 20 and 25 mg Crystalline Progesterone levels corresponding to treatments A, B, C, D, E and F respectively.

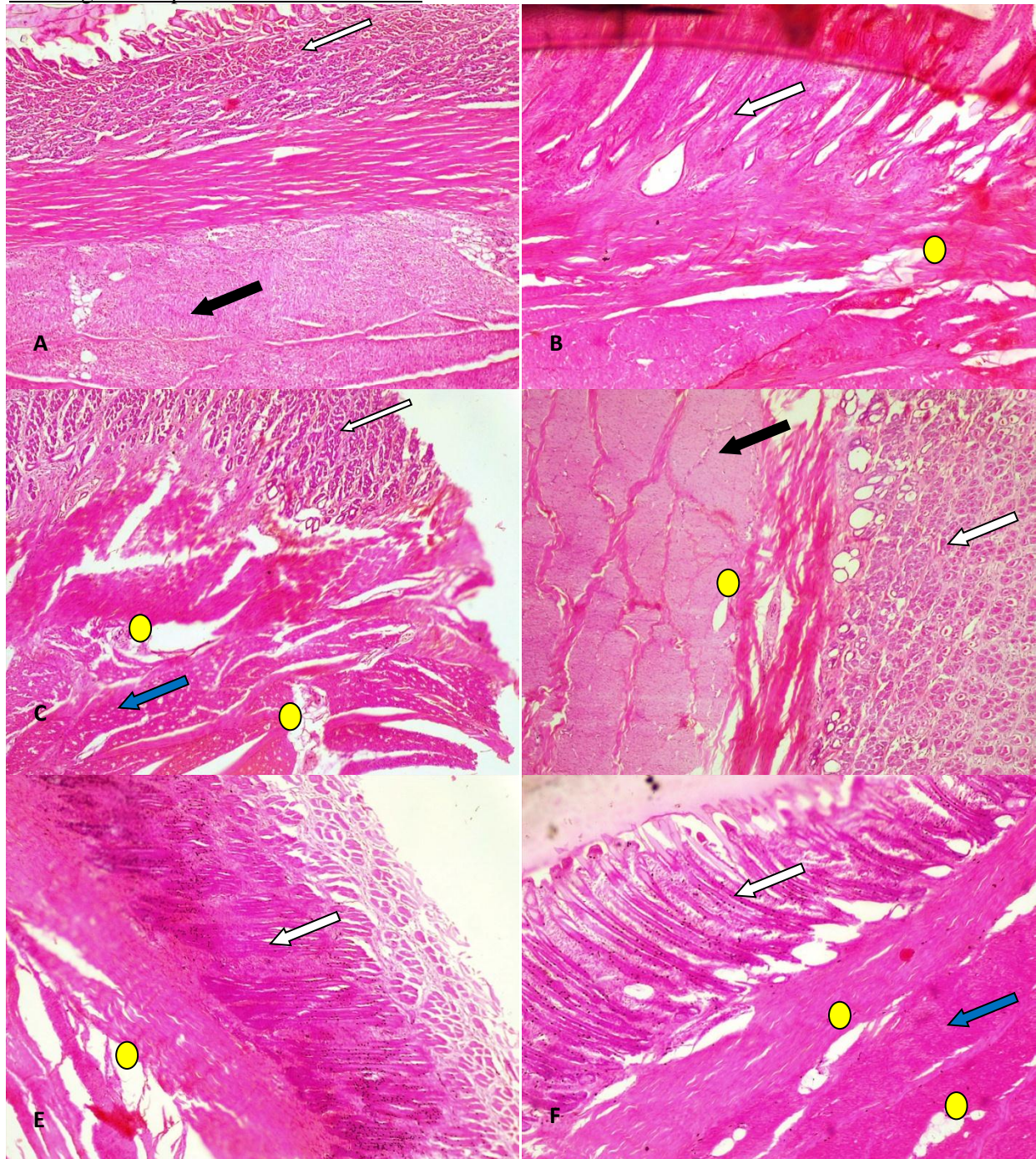
The treatment groups received (Gesteron-25[®]) injections intramuscularly via the breast muscle at 0, 5, 10, 15, 20 and 25 mg/bird twice per week at an interval of two days apart in the morning between 10.00 am and 11:00 am



At the end of the experiment, birds were slaughtered and shell glands were harvested using the procedure of Thierry (2000). Shell glands were fixed in 10% Neutral Buffered Formalin. The fixed samples were taken to histopathology laboratory for histological processing using standard histological techniques as described by Bancroft and Gamble (2008) and histological slides were prepared and observed for qualitative changes.

RESULTS

Histological Responses of the Shell Gland





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Plate I. Microphotograph of Shell Gland of Lohmann Brown Hens treated with Different Concentrations (A = 0 mg, B = 5 mg, C = 10 mg, D = 15 mg, E = 20 mg and F = 25 mg) of Crystalline Progesterone (Magnification – fat infiltrates x100, report x400). White arrows show mucosal glands with mostly empty lumen; black arrows show pale staining muscularis (denoting reduced protein content); blue arrows show muscularis with deeply pink cytoplasm (denoting normal protein content); yellow dots show fatty infiltrates. **A - Mucosa:** small-sized straight tubular glands lined by low cuboidal cells with luminal secretions not extending to basal glands. Thickness = 0.5 mm. **Muscularis:** moderate hypertrophy, fat infiltrates ++¹, Thickness = 12 mm. **B - Mucosa:** small-sized straight tubular glands lined by low cuboidal cells with abundant stroma and sparse secretions. Thickness = 0.5 mm. **Muscularis:** mild hypertrophy, fat infiltrates +, thickness = 10.5 mm. **C - Mucosa:** smaller-sized straight tubular glands lined by flattened cells with some secretions, moderate fibrous stroma. Thickness = 0.5 mm. **Muscularis:** moderate hypertrophy, cytoplasmic vacuoles in all fields, fat infiltrates +++++, thickness = not measured. **D - Mucosa:** small-sized straight tubular glands lined by cuboidal cells, abundant secretions, and fibrous stroma, thickness = 1.0 mm. **Muscularis:** no atrophy, cytoplasmic vacuoles in all fields, fat infiltrates +++++, thickness = not measured². **E -** Marked tissue folding obscuring slide. **Mucosa:** small-sized straight tubular glands lined by cuboidal cells, some secretions, abundant fibrous stroma, thickness = 0.5 mm. **Muscularis:** moderate hypertrophy, fat infiltrate +++, thickness - not measured. **F - Mucosa:** small-sized straight tubular glands lined by cuboidal cells, some secretions, abundant fibrous stroma, thickness = 1.0 mm. **Muscularis:** mild hypertrophy, fat infiltrates +++, thickness - not measured.

¹ Fat infiltrate + = no fat cells present/5 (x100) fields, ++ = 1-3 fat cell aggregates/5 (x100) fields, +++ = 6-10 fat cell aggregates/5 (x100) fields, and +++++ = >10 fat cell aggregates/5 (x100) fields.

² = No mucosa and no serosa identified on slide hence thickness is not measurable.

DISCUSSION

Progesterone treatment affects the differentiation of tubular gland cells (Oka & Schimke, 1969) and this depends on the stage of differentiation at which it was administered. If administered concomitantly with estrogen from inception of treatment, tubular gland cell differentiation will be abolished and by extension the growth of the oviduct (Boogaard, 1975). However, if onset of progesterone administration was delayed until when the birds are matured and actively laying as in the current study; it will not interfere with tubular gland cells because estrogen priming has already taken place (Boogaard, 1975). This sheds light on why exogenous crystalline progesterone (CP) in the present study showed clearly differentiated tubular glands across all CP treatment levels. The mild to moderate hypertrophy of the shell gland muscularis in the current work may point towards growth and functioning of the gland. Once the cells of the shell gland differentiate and become responsive to progesterone, they maintain this responsiveness even during the non-laying period (Yoshimura & Bahr, 1991). Progesterone receptor was reported by Yoshimura and Bahr (1991) to be present in the nuclei of the surface epithelial cells, tubular gland cells, stromal fibroblasts and smooth muscle cells in the arterial wall and myometrium of laying hens. Finally, the suggestion that estradiol and progesterone may regulate the growth of fat and fat-free tissues in female rats (Toth, Poehlman, Matthews, Tchernof & MacCoss, 2001) may explain the pattern of shell gland fat infiltration recorded in the present study.

In conclusion, crystalline progesterone affected the magnitude of shell gland fat infiltration. Further studies should be carried out on the effect of crystalline progesterone on the egg production in an *in vivo* study.

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