Serum sexual steroid hormones and lipids in commercial broilers (*Gallus domesticus*) in Costa Rica

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Primary Audience: Veterinarians, Nutritionists

SUMMARY

The poultry-breeding industry worldwide has focused on the development of hybrids for meat or egg production. In Costa Rica, there is a popular belief that commercial broilers have a high growth rate because they are raised with steroid hormones. This study was conducted to assess BW, nutritional state, and serum concentrations of some steroid hormones in a representative sample of 600 male and female Cobb 500 broilers from commercial farms and to compare them with a control group of 38 Cobb 500 broilers of both sexes reared in an experimental station. Control birds were weighed weekly during the 40-d experiment. Feed for the control group was prepared by personnel at the Investigation Center in Animal Nutrition of the University of Costa Rica and was supplied ad libitum. Commercial birds were blood sampled, weighed, and slaughtered at 40 d of age. All biochemical analyses were performed at the Faculty of Microbiology or the Hematological Investigation Center and Related Sicknesses of the University of Costa Rica. Compared with commercial birds, control birds had lower mean BW $(2,004 \pm 168 \text{ vs}, 2,127 \pm 260 \text{ g}; P = 0.004)$ and higher serum concentrations of total cholesterol $(3.72 \pm 1.01 \text{ vs}, 3.12 \pm 0.49 \text{ mmol/L}; P < 0.001)$. No significant differences were observed in mean serum concentrations of triglycerides and steroid hormones between the control and experimental groups. We conclude that the high BW and growth rate reached by these broilers in approximately 6 wk was not due to circulating steroid hormone levels.

Key words: broiler, cholesterol, 17β -estradiol, growth hormone, poultry breeding, progesterone, testosterone, triglyceride

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DESCRIPTION OF PROBLEM

Throughout the world, consumption of chicken meat continues to increase in both developed and developing countries. Scheuermann et al. [1] predicted that chicken will become the overall meat of choice by the year 2020 because of its nutritive qualities and availability for many people at affordable prices [2]. Therefore, in the last decades, the broiler industry worldwide

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has focused on the development of hybrids or genetically modified birds for meat or egg production. Meat birds have been selected over the last 50 yr for rapid growth and high yield [3, 4]. Nevertheless, there has also been increasing concern regarding the use of hormones as growth promoters in several fattening animals reared for human nutrition, especially cattle [5]. Some authors have also noted a possible association between these exogenous substances found in meat, dairy products, and eggs and an increase in the incidence of different types of cancers (breast, ovarian, or testicular) and associated diseases [6]. Some experts have even questioned the effect of these hormones over a long period of time [7]. In Costa Rica, there is a popular belief that commercial broilers have a high growth rate because they are raised with steroid hormones. However, no scientific studies have been performed that support this belief because investigations in Costa Rica have focused more on consumer perceptions regarding the nutritional value of poultry meat.

Growth enhancers, such as antibiotics, antifungals, and probiotics, have been widely used as an economical and safe way to increase BW and improve the nutritional status of many animals, to reduce the rate of diseases, and to improve the preservation of meat products, thereby diminishing costs and increasing producer profits [8].

Steroid hormones are very powerful compounds that have wide biological effects in animals and humans. In both, estradiol, progesterone, and testosterone occur naturally and in identical molecular forms [9]. These steroids, when ingested by humans via meat consumption, would have the same biological activity in the human body as the hormones produced naturally by the organism [10]. Additionally, these natural or synthetic steroids have a very limited linkage or bonding to human plasma proteins, remaining free in the bloodstream and thereby increasing their potential effects [5]. Recent studies in prepubertal children have demonstrated that even small differences in hormone levels and very low doses of steroid hormones can have significant biological effects [6].

Both the US Food and Drug Administration (**FDA**) and the USDA have established a limit on acceptable exposure to these hormones (1%)

or less), which is based on their daily production (**DP**) in the populations with a smaller DP, which means prepubertal boys for estradiol and progesterone, and prepubertal girls for testosterone [5, 9, 11, 12]. In these studies, the FDA stated, "Although not all sex steroids are demonstrated carcinogens, current evidence supports our conclusion that all endogenous sex steroids and synthetic compounds with similar biological activity should be regarded as suspect carcinogens" [9]. The FDA also concluded that this 1% value was supported by scientific evidence, was reasonable, and reflected sound public health policy [9]. Equally, the USDA concluded in its study that "the combined USDA, FDA, and EPA programs to control residues in meat and poultry have been effective in reducing the incidence of violative residues in meat and poultry products and in reducing potential risk" [12].

The limit values mentioned above are also found in the "Thirty-Second Report of the Joint Committee of Food Additive Experts of the FAO/WHO: Evaluation of Certain Remainders of Veterinary Drugs in Foods" [6], which considered the DP of estradiol and progesterone in prepubertal boys to be 6 and 150 μ g/d, respectively, and the DP of testosterone in prepubertal girls to be 32 μ g/d [9, 10]. Nevertheless, this maximal secure intake of natural sex steroids has been questioned because the DP rates on which it is based are very doubtful [5, 6].

Because of improvements in BW and FE of meat-producing animals, sexual steroid administration has been used as a practice in animal production, mainly in beef cattle, increasing in the United States over almost 50 yr; however, in the United States, sex steroids are not used for rearing poultry. In addition, the use of sex hormones for rearing livestock, either poultry or cattle, has been officially prohibited in the European Community since 1989 [5]. In Costa Rica, no prohibitions exist on the use of hormones in either poultry or cattle, except for Decree 7269-A of September 11, 1975, which bans the use of diethylstilbestrol for poultry or cattle rearing [13].

The substances that have been more widely used are the estrogens, especially 17β -estradiol, or progesterone or testosterone in combination with estradiol [6]. In many publications, a significant difference has been demonstrated be-

tween the hormonal levels of treated and nontreated cattle [11]. However, the use of steroid hormones in poultry is not a legal practice in the United States, and studies in broilers are practically nonexistent or null, which may be a result of the substances being banned.

In Costa Rica, a popular belief is that commercial broilers grow very rapidly because they are raised with steroid hormones. To our knowledge, to date no studies have been performed in Costa Rica related to this statement. Therefore, this study was conducted to assure consumers that steroid hormones are not being administered to birds.

MATERIALS AND METHODS

Selection of the Bird Sample

A total of 646 broilers (Cobb 500) [14] at 40 d of age were collected live from different broiler producers in the country between August and November 2007. One-half the birds were males and one-half were females. The average industry broiler density in Costa Rica is 13 to 15 birds/m². All the bird procedures followed were approved by the Institutional Council on Animal Care of the University of Costa Rica.

The participation of the different broiler producers in the study was promoted by the National Camera of Poultry Farmers of Costa Rica. The 5 main broiler-producing companies in the country participated in the investigation. For each producer, the number of farms visited was calculated proportionally to the participation of that company in national production. The farms were selected randomly on the day before the birds were weighed and blood sampled, and the companies were notified on the same day as the experimental procedures. A total of 19 commercial farms, distributed among 5 provinces in Costa Rica, were visited. On each farm, 34 broilers were randomly selected (one-half males and one-half females).

Birds in the Control Group

The control group was composed of 38 oneday-old Cobb 500 broilers from the hatchery of the Industrial Bird Processing Plant [15], from eggs produced by a single lot of breeders. These control birds were grown at the Experimental Station Fabio Baudrit Moreno at the University of Costa Rica. Broilers received 24 h of light. They were maintained in a $3-m^2$ pen with a placement density of 12.5 birds/m² and were raised to 40 d of age. All birds received a combined vaccine against infectious bronchitis (Massachusetts soft strain), infectious bursal disease, and Newcastle (live virus strain B1) viruses [16]. Water and food were supplied ad libitum daily during the experiment. Feed was prepared by the staff of the Investigation Center in Animal Nutrition of the University of Costa Rica. The starter, grower, and finishers diets of the control group are specified in Table 1. Nutrient levels were according to Cobb 500 requirements.

On d 40 of the experiment, birds in the control group were weighed with a digital scale, with a sensitivity of ± 2 g, just before they were blood sampled using a sterile 5-mL syringe and 21-gauge × 1.5-in. needle.

Data and Blood Sample Collection in Control and Commercial Broilers

The sex of birds was determined by direct observation of the superior crest of the bird. The BW of the birds was determined with a digital scale (sensitivity of ± 2 g). A sample of blood was collected from each broiler from the wing or radial vein using a sterile 5-mL syringe and 21-gauge × 1.5-in. needle. Samples were collected into plain Vacutainer tubes [17], and serum was obtained by centrifugation at 4,025 × g for 5 min at 25°C. Samples were stored individually at -20°C until biochemical tests were performed. All hemolyzed serums were eliminated from the study to avoid interference with the hormone and lipid analyses.

Biochemical Analysis

Serum hormone levels were determined using quantitative enzyme-linked immunoassays, according to the instructions of the manufacturers [18, 19]. The assay sensitivities for estradiol, progesterone, growth hormone, and testosterone were 36.7 pmol/L, 0.16 nmol/L, 0.66 ng/L, and 0.14 nmol/L, respectively. The serum levels of total cholesterol and triglycerides were determined by enzymatic colorimetric reactions using an automated liquid chemistry analyzer [20].

Table 1.	Ingredient an	d nutritional	composition	of diets c	of birds in the	e control aroup
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Item	Starter	Grower	Finisher
Ingredient, %			
Corn, yellow	52.30	64.49	67.12
Soybean oil, acidulated	3.40	2.80	3.06
Soybean meal (48%)	39.00	27.80	25.54
Limestone	1.60	1.58	1.42
Sea salt	0.50	0.46	0.39
DL-Methionine	0.33	0.28	0.24
L-Lysine hydrochloride	0.14	0.22	0.22
L-Threonine	0.03	0.07	0.07
Mono-dicalcium phosphate (21% phosphorus)	1.40	1.30	1.20
Broiler premix ¹	1.30	1.00	0.75
Calculated nutritional composition			
ME, kcal/kg	2,980	3,075	3,125
CP, %	22.75	18.5	17.5
Methionine, %	0.65	0.55	0.50
Methionine + cysteine, %	1.01	0.85	0.79
Lysine, %	1.37	1.15	1.10
Threonine, %	0.90	0.77	0.73
Calcium, %	1.00	0.95	0.87
Available phosphorus, %	0.46	0.42	0.40
Sodium, %	0.19	0.18	0.17

¹Broiler premix supplied (per kg of starter, grower, and finisher diets): vitamin A, 12,000, 10,000, and 8,000 IU; vitamin D₃, 4,000, 3,500, and 3,000 IU; vitamin E, 40, 25, and 20 mg; vitamin K, 3.5, 3.0, and 2.5 mg; vitamin B₁, 3.5, 3.0, and 2.5 mg; vitamin B₂, 8, 7, and 6 mg; niacin, 60, 50, and 40 mg; pantothenic acid, 14, 12, and 10 mg; vitamin B₆, 4, 3.5, and 3.0 mg; biotin, 0.15, 0.125, and 0.10 mg; folic acid, 1.5, 1.1, and 0.8 mg; vitamin B₁₂, 0.02, 0.015, and 0.013 mg; choline, 375, 300, and 250 mg; manganese, 100, 100, and 90 mg; iron, 45, 40, and 37 mg; zinc, 100, 100, and 90 mg; copper, 15, 15, and 13 mg; iodine, 1.0, 1.0, and 1.0 mg; and selenium, 0.3, 0.3, and 0.3 mg. The coccidiostat in the starter and grower diets was salinomycin sodium (60 ppm). The growth promoter in the starter diet was bacitracin methylene disalicylate (75 ppm).

Intraassay CV for total cholesterol and triglycerides were 2.0 and 3.1%, respectively.

Data Analysis

Statistical analysis was performed with SPSS, version 15.0 for Windows [21]. Student's t-test was used for the analysis of normally distributed data, and the Mann-Whitney U-test was used for skewed data. Outliers (mean \pm 3 SD) were excluded from the analysis. Data were categorized by sex of the broilers, and results are presented as means \pm SD, with 95% CI as ranges. Nonparametric Spearman's correlation coefficients were calculated to determine the association between BW and biochemical variables. To detect whether mean serum levels of biochemical variables and BW varied across the different broiler-producing companies, one-way ANOVA analysis was performed. All statements of significance were based on testing at a P < 0.05 level.

RESULTS AND DISCUSSION

Control Bird Group

The control group consisted of 38 broilers (23 females and 15 males; 60.5 and 39.5%, respectively). Male birds presented a mean BW significantly higher than female broilers of the same age (40 d). Likewise, male birds had higher mean serum levels of testosterone than female birds $(3.33 \pm 2.77 \text{ vs. } 0.82 \pm 0.92 \text{ nmol/L}; P <$ 0.001) and a tendency for higher triglyceride levels, although the difference was not significant in the latter case (P = 0.068; Table 2). No significant differences were observed among the other hormones and total cholesterol between female and male control birds. Wide variability in testosterone and growth hormone levels was observed in this group. In the control group, mean feed intake during the first 21 d was 1,036 g with the standard diet (data not shown).

Commercial Bird Group

The commercial group sample consisted of 600 broilers, one-half females and one-half males (Table 2). Male birds presented a mean BW significantly higher than female broilers of the same age (40 d). Male birds also had higher mean serum levels of testosterone (1.99 ± 2.17) vs. 1.35 ± 1.59 nmol/L; P < 0.001) and total cholesterol $(3.20 \pm 0.57 \text{ vs.} 3.11 \pm 0.53 \text{ mmol/L};$ P = 0.035) than female chicks. In contrast, female birds had significantly higher mean levels of estradiol than male broilers (44 \pm 17 vs. 37 \pm 3 pmol/L; P < 0.001). No differences were observed in mean serum levels of progesterone, growth hormone, and triglycerides between the 2 groups. Wide variability in testosterone and growth hormone levels was also observed in commercial fowl.

Table 3 shows the mean values for BW, lipid profile, and steroid hormones according to the commercial groups or the control group. Significant differences were observed only between the control and experimental groups for mean BW and serum concentrations of total cholesterol and triglycerides. Control birds presented lower BW and triglyceride levels than commercial birds. Serum total cholesterol levels were higher in the control group than in the different commercial groups. No significant differences were observed in mean serum concentrations of steroid hormones between the different commercial groups and the control group of broilers.

When the total populations of commercial and control birds were compared, significant differences were observed in mean BW and total cholesterol concentrations. Control birds had a mean BW lower (by 123 g) and serum concentrations of total cholesterol higher $(3.72 \pm 1.01$ vs. 3.12 ± 0.49 mmol/L; P < 0.001) than birds in the commercial group. No significant differences between the control and experimental groups were observed for mean serum concentrations of triglycerides and steroid hormones (estradiol, growth hormone, progesterone, and testosterone; Table 4).

Correlations (r < 0.200) were low between the BW of the commercial birds and the observed steroid hormone levels. Likewise, triglyceride concentrations correlated weakly with total serum cholesterol levels (r = 0.212) and BW of the broilers (r = 0.071; data not shown).

The large-scale broiler industry in Costa Rica represents approximately 52 million chickens raised for human consumption each year. Several commercial broiler hybrids have been used in Costa Rica, such as Ross, Cobb 500, and Hubbard. However, the most frequently used hybrid is currently Cobb 500 because it seems to adapt

	Control group			Commercial group			
Variable	Female $(n = 23)$	Male (n = 15)	P-value*	Female (n = 300)	Male (n = 300)	P-value*	
BW, g	$1,938 \pm 143$ (1,666 to 2,316)	$2,105 \pm 154$ (1,850 to 2,430)	0.002	$1,953 \pm 178$ (1,122 to 2,636)	$2,291 \pm 208$ (1,636 to 2,860)	< 0.001	
Estradiol, pmol/L	38 ± 4 (37 to 55)	37 ± 1 (35 to 38)	0.351	44 ± 17 (37 to 128)	37 ± 3 (37 to 73)	< 0.001	
Progesterone, nmol/L	0.21 ± 0.14 (0.16 to 0.80)	0.18 ± 0.06 (0.16 to 0.32)	0.415	0.22 ± 0.15 (0.16 to 0.80)	0.24 ± 0.16 (0.16 to 0.80)	0.160	
Testosterone, nmol/L	0.82 ± 0.92 (0.17 to 3.47)	3.33 ± 2.77 (0.35 to 10.41)	< 0.001	1.35 ± 1.59 (0.17 to 7.63)	1.99 ± 2.17 (0.17 to 10.41)	< 0.001	
Growth hormone, ng/L	22 ± 26 (1 to 100)	20 ± 30 (1 to 120)	0.829	18 ± 28 (1 to 170)	17 ± 29 (1 to 180)	0.483	
Total cholesterol, mmol/L	3.64 ± 0.83	3.85 ± 1.25	0.537	3.11 ± 0.53	3.20 ± 0.57	0.035	
Triglycerides, mmol/L	(1.14 to 5.21) 1.10 ± 0.31 (0.56 to 1.71)	(1.35 to 6.84) 1.28 ± 0.25 (0.86 to 1.71)	0.068	(1.14 to 6.71) 1.24 ± 0.57 (0.07 to 5.75)	(1.35 to 6.84) 1.25 ± 0.46 (0.06 to 3.23)	0.801	

Table 2. Serum hormonal levels and nutritional status parameters of broilers in the control and commercial groups at 40 d of age $(n = 638)^1$

¹Values presented are the means \pm SD of the different variables; n = number of birds. Numbers in parentheses are ranges. *Significant difference at P < 0.05 (Mann-Whitney).

Table 3. Comparison of n 40 d of age $(n = 638)^1$	rean serum hormona	I levels and nutritional	status parameters (±'	SD) of birds from the (different broiler compa	nies and birds from the	control group at
Variable	Company A $(n = 360)$	Company B $(n = 30)$	Company C $(n = 90)$	Company D $(n = 30)$	Company E $(n = 90)$	Control group (n = 38)	<i>P</i> -value*
BW, g	2,111 ± 267 (1.122 to 2.752)	2,167 ± 263 (1 796 to 2,682)	$2,149 \pm 262$ (1.684 to 2.860)	$2,103 \pm 228$ (1 774 to 2 586)	$2,159 \pm 238$ (1.574 to 2.780)	2,004 ± 168 (1.666 to 2.430)	0.021
Estradiol, pmol/L	41 ± 15 (37 to 128)	(37 to 55)	41 ± 13 37 to 92)	39 ± 6	(37 to 128)	37 ± 3 (37 to 55)	0.393
Progesterone, nmol/L	0.24 ± 0.19	(0.16 to 0.32)	(0.16 ± 0.07)	0.20 ± 0.07	(0.16 ± 0.13)	0.20 ± 0.11	0.354
Testosterone, nmol/L	1.74 ± 2.13	1.08 ± 1.56 (0.17 to 6.94)	(0.17 to 6.94)	1.12 ± 1.63 (0.17 to 7.63)	1.64 ± 1.53 (0.35 to 6 94)	1.81 ± 2.22 (0.17 to 10.41)	0.304
Growth hormone, ng/L	16 ± 27 (1 to 180)	26 ± 44 (1 to 160)	19 ± 24 (1 to 135)	11 ± 19 (1 to 100)	21 ± 33 (1 to 165)	21 ± 27 (1 to 120)	0.124
Total cholesterol, mmol/L	3.12 ± 0.50 (2.10 to 6.71)	3.25 ± 0.36 (2.64 to 4.12)	3.15 ± 0.51 (2.02 to 4.27)	2.90 ± 0.42 (2.25 to 4.01)	3.08 ± 0.44 (1.89 to 4.30)	3.72 ± 1.01 (1.14 to 6.84)	<0.001
Triglycerides, mmol/L	(0.06 to 5.75)	(1.02 to 2.02)	(0.46 to 3.23)	(1.12 to 2.67)	0.99 ± 0.30 (0.49 to 1.82)	(0.56 to 1.71)	<0.001
¹ n = number of birds. Numb *Significant difference betw	ers in parentheses are r reen means at $P < 0.05$	anges. (1-way ANOVA).					

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Variable	Commercial group (n = 600)	Control group $(n = 38)$	P-value*
BW, g	$2,127 \pm 260$ (1,122 to 2,860)	$2,004 \pm 168$ (1.666 to 2.430)	0.004
Estradiol, pmol/L	(1,122) (1	37 ± 3 (37 to 55)	0.114
Progesterone, nmol/L	(37 to 123) 0.23 ± 0.16 (0.16 to 0.80)	(371033) 0.20 ± 0.11 (0.16 to 0.80)	0.190
Testosterone, nmol/L	(0.10 to 0.30) 1.65 ± 1.90 (0.17 to 0.02)	(0.10 to 0.80) 1.81 ± 2.23 (0.17 to 10.41)	0.603
Growth hormone, ng/L	(0.17 ± 29)	(0.17 to 10.41) 21 ± 27 (1 ± 120)	0.442
Total cholesterol, mmol/L	(1 to 180) 3.12 ± 0.49	(1 to 120) 3.72 ± 1.01	< 0.001
Triglycerides, mmol/L	(1.89 to 6.71) 1.25 ± 0.53 (0.06 to 5.75)	(1.14 to 6.84) 1.17 ± 0.30 (0.56 to 1.71)	0.331

Table 4. Serum hormonal levels and nutritional status parameters in broilers from the commercial and control groups at 40 d of age (n = 638)¹

¹Values presented are the means \pm SD of the different variables. n = number of birds. Numbers in parentheses are ranges. *Significant difference at P < 0.05 (Mann-Whitney).

satisfactorily to climatic conditions in the country and to present an excellent growth rate and FCR.

The broiler industry worldwide has genetically refined the broiler chicken for rapid growth, efficient feed conversion, and development of broad-breasted muscles. However, the birds are subject to congestive heart failure and heart attacks and often have difficulty walking because of leg disorders [14]. Because of the rapid growth of these birds, they reach an average market BW of 2.2 kg in 6 or 7 wk [22]. Recently, Radu et al. [23], who evaluated some features of meat production in Cobb 500 chicken hybrids, reported mean BW of $2,296 \pm 50$ g in males and $2,169 \pm$ 44 g in females, with a mean BW of $2,232 \pm 37$ g in both sexes. These data are consistent with the mean BW reached by our bird control group and are close to the average BW for live broilers marketed in Costa Rica. Although a significant difference was found in BW between the commercial and control groups in our experiment, the differences could be explained in terms of the diet or space in the pens where the birds were raised. Commercial poultry houses in Costa Rica are for flocks of 10,000 to 20,000 broilers each (average placement density of 13 to 15 birds/ m^2), limiting the space and contributing to a sedentary life, and thereby a rapidly increasing BW in the birds

Very little information is available in the literature concerning serum steroid hormone concentrations in Cobb 500 broilers. Both female and male birds produce progesterone, testosterone, and estradiol, but in different proportions according to the sex of the broiler [24]. Secondary sexual characteristics in male and female chicks are under the control of androgens and estrogens, respectively. In the male, androgens are responsible for the growth, size, and coloring of the comb; the plumage and bill color; the structure of the feathers, vocalizations; and behavior [25]. Testosterone is the principal steroid produced and secreted by the testes of sexually mature chickens [26]. In the female, estrogens control the color and shape of plumage and the sexual behavior of the birds. As expected, in our study significant differences in testosterone and estradiol concentrations were found between commercial female and male birds. Male broilers naturally have higher serum testosterone concentrations; thus, they present a greater BW than female birds. This characteristic was observed in both the commercial and control male groups in the present study. No differences were found in serum estradiol concentrations between female and male control birds, probably because the number of broilers was low (n = 38).

Blood levels of testosterone have also been demonstrated to exhibit a diurnal rhythm [27,

28] and pulsatile release [29, 30]. This behavior of steroid hormones can explain the wide range of growth hormone and testosterone concentrations found in commercial and control broilers.

No important differences were observed in serum concentrations of steroid hormones between the commercial and control birds. We suggest that the high BW and growth reached by these broilers by 6 wk of age was not related to the administration of steroid hormones as growth promoters. Several additional factors support the idea that hormones are not good growth promoters in broilers. First, steroid hormones in nonlaying birds are very short-lived in the bloodstream because they have a higher metabolic clearance rate than in laying birds. The biologically active half-life of progesterone in the circulation of birds is approximately 11 min. The half-life of estradiol in the circulation has been calculated at approximately 28 min [31]. Second, genetically manipulated chickens, such as Cobb 500, live at a higher metabolic limit. An increase in their growth rate probably would increase the mortality of these broilers because of caloric stress [14]. Third, administering hormones as growth promoters is difficult because these must be injected into the birds, a practice that is not practical on farms with thousands of broilers. Fourth, steroid hormones are expensive and their sale is restricted by several companies.

Finally, the lipid profile, represented in this case by the total cholesterol and triglyceride analyses, is commonly used as a nutritional status parameter. The differences observed in serum total cholesterol and triglyceride levels between broilers from the different broiler companies and those from the control group suggest that each producer has its own nutritional strategy.

Although serum is a good representative matrix of the different tissues of the organism, the meat would have been a more appropriate tissue in which to investigate hormones as growth promoters. This limitation of our study must be considered when interpreting the data. Further surveys are needed to assess steroid hormone concentrations and lipid content in the breast muscle of broilers used for human nutrition.

CONCLUSIONS AND APPLICATIONS

1. To our knowledge, this is the first study in Costa Rica to quantify serum steroid

hormones in broiler chicken hybrids destined for human consumption.

- 2. We observed no statistical differences in mean serum concentrations of steroid hormones in broilers between the commercial and control groups.
- 3. Similar BW of the birds were recorded in both groups at 40 d of age.
- 4. We conclude, based on these findings, that the high BW and growth reached by these broilers by approximately 6 wk of age were not due to administration of steroid hormones.

REFERENCES AND NOTES

1. Scheuermann, G. N., S. F. Bilgili, J. B. Hess, and D. R. Mulvaney. 2003. Breast muscle development in commercial broiler chickens. Poult. Sci. 82:1648–1658.

2. Van Horne, P. L. M. 2002. Production cost development of broiler meat. Arch. Geflugelkd. 66:26–27.

3. Barton, N. F. 1994. Breeding meat type poultry for the future targets for selection, limits to performance and market requirements for chicken. Pages 33–38 in Proc. 9th Eur. Poult. Conf. World's Poult. Sci. Assoc., UK Branch, Glasgow, UK.

4. Mead, G. C. 1998. The safety of poultry production: Present trends and future developments. Temperton Fellowship Rep. No. 7. Harper Adams Agric. College, Newport UK.

5. Aksglaede, L., A. Juul, H. Leffers, N. E. Skakkebæk, and A. M. Andersson. 2006. The sensitivity of the child to sex steroids: Possible impact of exogenous estrogens. Hum. Reprod. Update 12:341–349.

6. Andersson, A. M., and N. E. Skakkeback. 1999. Exposure to exogenous estrogens in food: Possible impact on human development and health. Eur. J. Endocrinol. 140:477–485.

7. Gandhi, R., and S. Snedeker. 2005. Consumer concerns about hormones in food. Pages 1–5 in Cornell University Program on Breast Cancer and Environmental Risk Factors. Cornell Univ., Ithaca, NY.

8. Wong, A., L. S. Millan, J. L. C. B. Aldunate, and C. A. Sanaron. 2004. The toxicity of growth promoters in food animals and its implications on human health in Brazil. J. Toxicol. Clin. Toxicol. 42:493. (Abstr.)

9. US Food and Drug Administration. 2005. Guidance for Industry #3: General Principles for Evaluating the Safety of Compounds Used in Food-Producing Animals. US Food and Drug Admin., Rockville, MD. http://www.fda.gov/ohrms/dockets/98fr/2005d-0219-gdl0001.pdf Accessed Oct. 28, 2009.

10. Joint FAO/WHO Expert Committee on Food Additives. 1988. Residues of Some Veterinary Drugs in Animals and Food. WHO Tech. Rep. Ser. 763. World Health Org., Geneva, Switzerland.

11. Joint FAO/WHO Expert Committee on Food Additives. 1988. Residues of Some Veterinary Drugs in Animals and Food. Food Nutr. Paper 41. Food Agric. Org. United Nations, Rome, Italy. 12. Cordle, M. 1988. USDA regulation of residues in meat and poultry products. J. Anim. Sci. 66:413–433.

13. Rojas, J. L. 2009. Laboratorio Nacional de Servicios Veterinarios, Ministerio de Agricultura y Ganadería, Heredia, Costa Rica. Personal communication.

14. Cobb-Vantress. 2006. Cobb-500[™] Product Profile. Cobb-Vantress, Siloam Springs, AR. http://www.cobb-vantress.com Accessed Oct. 23, 2009.

15. Corporación PIPASA S.A., Alajuela, Costa Rica. http://www.pipasa.net. Accessed June 21, 2010.

16. Vineland Laboratories, Vineland, NJ.

17. Becton Dickinson, Rutherford, NJ.

18. Growth hormone and testosterone, Diagnostic Systems Laboratories Inc., Webster, TX.

19. Progesterone and estradiol, International Immuno-Diagnostic, Foster City, CA.

 $20.\ Model ACE, Alfa Wassermann, Woerden, the Netherlands.$

21. SPSS. 2006. SPSS, Version 15.0 for Windows. SPSS Inc., Chicago, IL.

22. Fanatico, A. 2005. Poultry Genetics for Pastured Production. ATTRA-Natl. Sustainable Agric. Inf. Serv., Natl. Cent. Appropriate Technol., Fayetteville, AR.

23. Radu-Rusu, R. M., I. Vacaru-Opris, and C. G. Radu-Rusu. 2008. Quantitative and qualitative features of meat production in Cobb-500 chicken hybrid. Zooteh. Biotehnol. 41:690–697.

24. Tanabe, Y., T. Nakamura, K. Fujioka, and O. Doi. 1979. Production and secretion of sex steroid hormones by the testes, the ovary, and the adrenal glands of embryonic and young chicken (*Gallus domesticus*). Gen. Comp. Endocrinol. 39:26–33.

25. Bloom, M. A., W. Bloom, and F. C. McLean. 1941. The role of androgen in the production of medullary bone in pigeons by the administration of sex hormones. Am. J. Physiol. 133:216P–222P

26. Galli, F. E., O. Irusta, and G. F. Wassermann. 1973. Androgen production by testes of *Gallus domesticus* during postembryonic development. Gen. Comp. Endocrinol. 21:262–266.

27. Schanbacher, B. D., W. R. Gomes, and N. L. VanDemark. 1974. Diurnal rhythm in serum testosterone levels and thymidine uptake by testes in the domestic fowl. J. Anim. Sci. 38:1245–1248.

28. Gulati, D. P., T. Nakamura, and Y. Tanabe. 1981. Diurnal variations in plasma LH, progesterone, testosterone, estradiol and estrone in the Japanese quail. Poult. Sci. 60:668–673.

29. Wilson, E. K., J. C. Rogler, and R. E. Erb. 1979. Effect of sexual experience, location, malnutrition, and repeated sampling on concentrations of testosterone in blood plasma of *Gallus domesticus* roosters. Poult. Sci. 58:178–186.

30. Ottinger, M. A. 1983. Short-term variation in serum luteinizing hormone and testosterone in the male Japanese quail. Poult. Sci. 62:908–913.

31. Johnson, A. L., and A. van Tienhoven. 1981. Pharmacokinetics of estradiol-17 β in the laying hen. Poult. Sci. 60:2720–2723.

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