Effects of Corticosterone and Propylthiouracil on Growth and Hepatic Lipid and Abdominal Fat Deposition in Broiler and Egg-Type Chickens

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Abstract Six experiments were conducted to study the effects of corticosterone and/or antithyroid agent (PTU) on growth and lipid deposition in liver and adipose tissue of broiler and egg-type chickens aged 10-32 days. Corticosterone was intramuscularly injected daily at graded dose levels (300-1200 $\mu\text{g/day/bird}$) in broiler and egg-type chickens. Chickens starved for 2 days were subjected for 4 days refeeding with corticosterone injection. A silastic tube filled with corticosterone was implanted in egg-type chickens. Corticosterone injection extensively retarded growth rate and increased linearly abdominal fat content as increasing the dose levels. Constant and slow release of corticosterone at a rate of 22 or 43 $\mu\text{g/kg}$ body weight/day from the implanted tube increased abdominal fat content with no growth retardation. PTU feeding (0.025% in diet) elevated liver lipid and abdominal fat contents. Concomitant administration of corticosterone and PTU resulted in marked increases of liver lipid and abdominal fat contents. In terms of abdominal fat deposition, broiler chicks appear more susceptible than egg-type chicks of corticosterone administered. It is, therefore, suggested that an increased rate of corticosterone secretion and hypothyroid state are concerned in the fatness of broiler and egg-type chickens.

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Key words : corticosterone, hypothyroid, PTU, chickens, liver lipid, abdominal fat

Injection of broiler chicks with corticosterone retards growth and increases liver lipid and abdominal fat contents^{7,8,10,11,16)}. AKIBA *et al.*⁷⁾ reported that in broiler chickens corticosterone released at a low concentration from silastic tubes implanted under the neck skin significantly increased abdominal fat content with no retardation of growth. In addition, AKIBA *et al.*⁶⁾ observed a significant correlation between plasma corticosterone concentration

and abdominal fat content in broilers. Hence, it is suggested that a higher corticosterone concentration in the plasma is possibly concerned in the excess fat deposition which is frequently found in broiler production. There is, however, little information about what extent corticosterone affects lipid deposition in egg-type growing chicks.

Thyroid status affects hepatic lipid deposition. An administration of antithyroid sub-

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stances induces excessive deposition in livers of immature chicks^{2,5,12,17)} whereas thyroactive substances decrease liver lipids^{12,18)}. These reports led authors to expect that the antithyroid agent increases lipid deposition in the carcass and liver of broiler and egg-type chickens.

The present work was undertaken to study the effect of corticosterone and/or antithyroid agent on growth and lipid deposition in liver and adipose tissue of growing chicks.

Materials and Methods

Animals and diet : Male broiler chickens of Arbor Acre strain and male White Leghorn chickens of Shaver strain were used in 6 experiments. They were reared in cages under a constant lighting program and temperature ($22 \pm 2^\circ\text{C}$). The experimental diet⁷⁾ (ME 3105 kcal/kg, CP 20%) mainly consisted of corn and soybean meal and water were provided on free access. At the start of each experiment, the birds were weighed and allocated into the specified groups according to their body weight to equalize the mean body weight in each group.

Experiments 1 and 2 : White Leghorn chicks (10 days old, 105 g in mean body weight) and broiler chicks (18 days old, 375 g in mean body weight) were allocated into 5 or 6 groups with 8 chicks each. The birds were treated with or without corticosterone at graded dose levels and/or propylthiouracil (PTU, 0.025% in diet) for 14 days.

Experiment 3 : White Leghorn chicks (16 days old, 170 g in mean body weight) were allocated into 5 groups with 8 birds each. They were injected with or without corticosterone at graded dose levels or implanted with corticosterone-tube for 10 days.

Experiments 4 and 5 : Broiler chicks (27 days old, 635 g in mean body weight) and White Leghorn chicks (32 days old, 273 g in mean body weight) were fed the diet for 4 days following 2 days starvation in experiments 4 and 5, respectively. A half of the birds in

each group received daily injection of corticosterone during the 4 days feeding period.

Experiment 6 : White Leghorn chicks (14 days old, 120 g in mean body weight) were implanted with tube containing corticosterone and reared for 21 days. Estimated release rates from the tube were 22 and 43 $\mu\text{g/kg}$ body weight/day.

Administration of corticosterone : Corticosterone (Sigma Chemical Company, St. Luis, MO, U.S.A.) was administered through two different ways. For the injection, corticosterone dissolved in 0.2 ml soybean oil was injected intramuscularly into the pectoral muscle daily on 10 AM throughout the experimental period. For the perparation of corticosterone tube, polymethylsiloxane (Silastic, Dow Chemical Corp., Midland, MI, U.S.A.) and Silastic Medical Adhesive Silicone Type A were used by the methods of SMITH *et al.*¹⁹⁾ and AKIBA *et al.*⁴⁾ Different length of corticosterone tubes were implanted under the neck skin to ensure the constant release as being described by AKIBA *et al.*⁷⁾ The release rates of corticosterone from implanted tubing were estimated based on the report of KINCL *et al.*¹⁴⁾

Analytical procedure : At the end of each experiment, the birds were killed by cervical dislocation, livers and abdominal fat pads were removed and weighed. Lipid in the liver was extracted by the method of FOLCH *et al.*¹³⁾ and quantified by gravimetry. The data were analyzed using the General Linear Model Procedure of STATISTICAL ANALYSIS SYSTEM²⁰⁾ with the Duncan separation of means option.

Results

Experiments 1 and 2 : Corticosterone (1200 $\mu\text{g/day}$) injection and PTU feeding significantly reduced body weight gains but did not affect food intake (Table 1). PTU feeding increased both liver weight and abdominal fat content, whereas corticosterone injection at 1200 $\mu\text{g/day}$ increased liver lipid and abdominal fat contents. Implantation of corticosterone tube (20

Table 1. Effects of corticosterone administration and PTU feeding on performance and liver and abdominal fat contents in broiler and egg-type chicks (Experiments 1, 2 and 3)

Treatment		Body weight gain	Food intake	Liver weight	Liver lipid content	Abdominal fat content
Corticosterone ($\mu\text{g/day}$)	PTU (% diet)	(g)	(g)	(g/100 g BW)	(% wet weight)	(g/100 g BW)
Experiment 1 ¹ (White Leghorn male chicks)						
0	0	242 $\pm$ 10 ^{a6}	377	3.4 $\pm$ 0.2 ^b	4.7 $\pm$ 0.4 ^{bc}	0.34 $\pm$ 0.09 ^d
600 ⁵	0	233 $\pm$ 20 ^a	377	3.3 $\pm$ 0.2 ^b	4.3 $\pm$ 0.1 ^c	0.64 $\pm$ 0.24 ^c
1200	0	190 $\pm$ 12 ^b	370	3.7 $\pm$ 0.2 ^b	4.9 $\pm$ 0.3 ^b	1.00 $\pm$ 0.15 ^b
600	0.025	191 $\pm$ 8 ^b	363	6.2 $\pm$ 0.3 ^a	5.5 $\pm$ 0.4 ^a	2.20 $\pm$ 0.10 ^a
1200	0.025	185 $\pm$ 15 ^b	363	5.7 $\pm$ 0.5 ^a	5.4 $\pm$ 0.4 ^a	2.24 $\pm$ 0.36 ^a
Experiment 2 ² (Broiler male chicks)						
0	0	545 $\pm$ 38 ^{a6}	1102 $\pm$ 22 ^a	2.2 $\pm$ 0.3 ^b	4.4 $\pm$ 0.3 ^b	0.60 $\pm$ 0.28 ^c
20 ⁴	0	554 $\pm$ 19 ^a	1085 $\pm$ 12 ^{ab}	2.3 $\pm$ 0.3 ^b	4.6 $\pm$ 0.3 ^b	1.00 $\pm$ 0.38 ^{bc}
600 ⁵	0	499 $\pm$ 14 ^b	1092 $\pm$ 10 ^{ab}	2.4 $\pm$ 0.1 ^b	5.4 $\pm$ 0.3 ^a	1.47 $\pm$ 0.50 ^b
0	0.025	478 $\pm$ 38 ^b	1084 $\pm$ 8 ^{ab}	5.0 $\pm$ 0.5 ^a	4.7 $\pm$ 0.5 ^b	1.75 $\pm$ 0.73 ^b
20 ⁴	0.025	491 $\pm$ 14 ^b	1093 $\pm$ 10 ^{ab}	5.5 $\pm$ 0.5 ^a	4.5 $\pm$ 0.1 ^b	1.89 $\pm$ 0.42 ^b
600	0.025	423 $\pm$ 19 ^c	1064 $\pm$ 29 ^b	4.9 $\pm$ 0.7 ^a	5.4 $\pm$ 0.4 ^a	2.83 $\pm$ 0.44 ^a
Experiment 3 ³ (White Leghorn male chicks)						
0	0	94 $\pm$ 4 ^{b6}	289	3.1 $\pm$ 0.1 ^a	4.4 $\pm$ 0.4 ^b	0.34 $\pm$ 0.09 ^c
12.4 ⁴	0	110 $\pm$ 7 ^a	313	3.3 $\pm$ 0.5 ^a	4.5 $\pm$ 0.4 ^b	0.64 $\pm$ 0.24 ^b
300 ⁵	0	92 $\pm$ 7 ^b	284	3.1 $\pm$ 0.2 ^a	4.5 $\pm$ 0.3 ^b	0.55 $\pm$ 0.08 ^c
600	0	84 $\pm$ 10 ^b	297	3.1 $\pm$ 0.2 ^a	4.5 $\pm$ 0.5 ^b	0.65 $\pm$ 0.14 ^{ab}
1200	0	78 $\pm$ 8 ^b	267	3.2 $\pm$ 0.3 ^a	5.5 $\pm$ 0.8 ^a	0.94 $\pm$ 0.39 ^a

¹ White Leghorn male chicks (10 days old, 105 g in mean body weight) were treated with corticosterone and/or PTU for 14 days.

² Broiler male chicks (18 days old, 375 g in mean body weight) were treated with corticosterone and/or PTU for 14 days.

³ White Leghorn male chicks (16 days old, 170 g in mean body weight) were treated with corticosterone for 10 days.

⁴ Silastic tube filled with corticosterone was implanted under the neck skin.

⁵ Corticosterone dissolved in soybean oil was injected daily into the pectoral muscle.

⁶ Mean $\pm$ SD with 8 observations in Experiments 1 and 3. Mean $\pm$ SD with 5 observations with 3 chicks each in Experiment 2. Means with different superscript letters in columns are significantly different ($P<0.05$).

$\mu\text{g/day}$ as the release rate) tended to increase abdominal fat content with no retardation in body weight gain in broiler chicks. Concomitant administration of corticosterone and PTU resulted in the marked increase in abdominal fat content in both experiments.

Experiment 3: Injecting corticosterone daily at the rate over 600 $\mu\text{g/day}$ tended to reduce body weight gains, but the injection at 300 $\mu\text{g/day}$ and tube implants did not affect those (Table 1). There were no significant changes

in liver weight and liver lipid content except an increase of liver lipid content in chicks received corticosterone at 1200 $\mu\text{g/day}$. Abdominal fat content increased linearly as an increase of corticosterone doses. Abdominal fat content in chicks implanted with corticosterone-tube (20 $\mu\text{g/day}$) was almost comparable with that in chicks injected at the level of 600 $\mu\text{g/day}$.

Experiments 4 and 5: Corticosterone injection tended to increase liver weight, liver lipid content and abdominal fat content in starved-

Corticosterone and Lipid Deposition

Table 2. Effects of corticosterone administration on performance and liver lipid and abdominal fat contents in starved and refed chicks (Experiments 4 and 5)

Treatment Corticosterone ³ ($\mu\text{g/kg BW/day}$)	Body weight (g)	Food intake (g)	Liver weight (g/100g BW)	Liver lipid content (% wet weight)	Abdominal fat content (g/100 g BW)
Experiment 4 ¹ (Broiler male chicks)					
0	959 $\pm$ 33 ^{a1}	415	2.8 $\pm$ 0.2 ^b	4.9 $\pm$ 0.5 ^a	0.90 $\pm$ 0.23 ^b
940	906 $\pm$ 56 ^a	453	3.2 $\pm$ 0.1 ^a	5.4 $\pm$ 0.6 ^a	1.32 $\pm$ 0.23 ^a
Experiment 5 ² (White Leghorn male chicks)					
0	391 $\pm$ 16 ^a	190	3.2 $\pm$ 0.2 ^a	4.9 $\pm$ 0.2 ^a	0.48 $\pm$ 0.09 ^a
930	379 $\pm$ 14 ^a	197	3.6 $\pm$ 0.3 ^a	5.2 $\pm$ 0.3 ^a	0.55 $\pm$ 0.18 ^a

¹Broiler male chicks (27 days old, 635 g in mean body weight) were fed the diet for 4 days following 2 days starvation.

²White Leghorn male chicks (32 days old, 273 g in mean body weight) were fed the diet for 4 days following 2 days starvation.

³Corticosterone dissolved in soybean oil was injected daily into the pectoral muscle during 4 days refeeding period.

⁴Mean $\pm$ SD with 5 observations with 2 chicks each. Means with different superscript letters in columns are significantly different ($P < 0.05$).

Table 3. Effects of corticosterone-tube implantation on performance and liver lipid and abdominal fat contents in egg-type chicks¹ (Experiment 6)

Treatment Corticosterone ($\mu\text{g/kg BW/day}$)	Body weight (g)	Food intake (g)	Liver weight (g/100g BW)	Liver lipid content (% wet weight)	Abdominal fat content (g/100 g BW)
0	280 $\pm$ 8 ^a	642	3.5 $\pm$ 0.2 ^a	4.3 $\pm$ 0.4 ^a	0.45 $\pm$ 0.16 ^b
22	281 $\pm$ 21 ^a	712	3.5 $\pm$ 0.2 ^a	4.3 $\pm$ 0.3 ^a	0.64 $\pm$ 0.24 ^{ab}
43	281 $\pm$ 22 ^a	723	3.5 $\pm$ 0.2 ^a	4.3 $\pm$ 0.4 ^a	0.66 $\pm$ 0.12 ^a

¹White Leghorn male chicks (14 days old, 120 g in mean body weight) were implanted with silastic tube filled with corticosterone and reared for 21 days.

²Mean $\pm$ SD with 8 observations. Means with different superscript letters in columns are significantly different ($P < 0.05$).

refed broiler and egg-type chickens, though the significant increase was evident in the abdominal fat content of broiler chickens (Table 2).

Experiment 6: Corticosterone released at 43 $\mu\text{g/day}$ from implanted tubes significantly increased abdominal fat content whereas it affected neither body weight gain, food intake, liver weight and liver lipid content (Table 3). Similar trend was demonstrated in chicks implanted corticosterone (22 $\mu\text{g/day}$).

Discussion

It has been documented that injecting cortico-

sterone reduces body weight gain and increases lipid deposition in chicks^{3,7-11}. The present data show a growth retardation and lipid deposition in the abdominal area by injection of corticosterone in both broiler and egg-type chickens, complementing the previous studies. Furthermore, in experiment 2 we examined the effects of corticosterone when chicks accompanied much lipid deposition and increased lipogenesis. Refeeding for several days following starvation induces overeating and increased lipid deposition^{4,15}. Our data demonstrate that corticosterone injection accelerates

moreover the lipid deposition which was induced by the refeeding following starvation.

Abdominal fat content was increased by daily injection of 600 μg corticosterone to approximately 3 times the content in chicks received no corticosterone in both broiler and egg-type chickens (experiments 1 and 2). Considering the dose level of corticosterone per unit body weight, the dose level injected in egg-type chickens was 3 times higher than that in broiler chickens because body weights of the egg-type's were almost one-third those of the broilers. The significant increase of abdominal fat content in the starved-refed chickens was only evident in broiler chicks, but not in egg-type chickens, even when they were injected with the same dose of corticosterone in experiments 4 and 5. In addition, no significant increase of liver lipid content by injecting 600 μg corticosterone was observed in egg-type chickens, despite they received 3 times higher corticosterone than broiler chickens in terms of unit body weight basis. Taken together, it appears that in terms of lipid deposition egg-type chickens are less susceptible to corticosterone injected intramuscularly in comparison with broiler chickens. It is, however, inappropriate to rule out the possibility that the differences in susceptibility to corticosterone might be, in part, due to differences in ages of chickens used.

Implanting tubes ensures a constant and slow release of hormones¹⁴⁾. The estimated release rates (12.4 μg –43 $\mu\text{g}/\text{kg}$ body weight/day) of corticosterone from tubes are far below the pharmacological level and within almost physiological level as being discussed in our previous paper. This is supported by the evidence that the growth rate was not depressed by the implantation of tube filled with corticosterone, while body weight gain was reduced by the daily injection in the present experiment. In line with our previous experiment using broiler chickens, this study shows that implanting tube to ensure a slow release of cortico-

sterone significantly increased abdominal fat deposition in egg-type chickens (experiment 6). The degree of increase in abdominal fat content by corticosterone tube (12.4 $\mu\text{g}/\text{day}$ as the release rate) was almost comparable with that found in egg type chickens injected with 600 μg corticosterone (experiment 3), being in line with our previous report⁷⁾ and emphasizing that tube implantation is an effective mean to increase fatness in chickens with no growth retardation.

The corticosterone tube (22 and 43 $\mu\text{g}/\text{kg}$ body weight/day as the release rates) elevated abdominal fat content to 1.5 times that in chicks with no corticosterone in the egg-type chickens, whereas in the broiler chickens corticosterone tube (12.4 $\mu\text{g}/\text{kg}$ body weight/day) elevated the abdominal fat up to 1.9 times of chicks with no corticosterone (experiments 3 and 6). Previously, we showed that in broiler chicks implanting corticosterone (53 $\mu\text{g}/\text{kg}$ body weight/day as the release rate) increased abdominal fat content to 2 times of the control⁷⁾. Thus, in terms of carcass fat deposition broiler chickens are more susceptible than egg-type chickens of corticosterone administered at physiologically low level, although it is unclear whether the reason is ascribed to the difference in plasma corticosterone concentrations and/or the difference in responsiveness of the tissues to exogenous corticosterone. This is coincident with the findings, as being stated above, that broiler chickens respond more susceptively to corticosterone injected and thereby accumulates carcass fats when compared to egg-type chickens. This might be a further reflection of findings that broiler chickens accompanies with much deposition of carcass fat in the modern broiler production. AKIBA *et al.*⁶⁾ demonstrated that a higher corticosterone concentration in plasma is a causative factor promoting excess fat deposition in broilers.

It has been shown in immature egg-type chickens that an administration of PTU induc-

ed hypothyroidism¹⁾ and thereby stimulating lipid deposition in liver^{2,17)}. In the present study, however, PTU feeding at a level of 0.025 % had no influence on liver lipid content, while PTU increased the liver weight in broiler chickens (experiments 1 and 2). Obviously shown in the present experiment is that PTU feeding at a low dose level increased extensively abdominal fat deposition in both broiler and egg-type chickens. It is, therefore, suggested that hypothyroid state is concerned, at a certain extent, in the excess fat deposition in chickens.

Since body weight gains were reduced by PTU but not by corticosterone tube, it seems likely that the properties of abdominal fat deposition induced by PTU is not comparable with those induced by the corticosterone implantation. A concomitant administration of PTU and corticosterone markedly increased liver lipid and abdominal fat contents as being shown in Table 1. These effects appear rather additive than synergistic. AKIBA *et al.*²⁾ observed that liver lipid contents were elevated by synergistic actions of PTU and estrogen. Accordingly, it would possibly be better to speculate that mechanism by which corticosterone increases carcass lipid deposition in chickens is not homologous with the mechanism by which estrogen does.

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ブロイラーおよび卵用種ヒナの成長と脂肪蓄積に対する コルチコステロンと抗甲状腺物質投与の影響

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ブロイラーにおいて血漿中コルチコステロン濃度と腹腔内脂肪量の間に正の相関関係があること (AKIBA *et al.*, 1987), そして, コルチコステロン微量投与により脂肪蓄積が増加すること (AKIBA *et al.*, 1992) が明らかにされている。そこで, コルチコステロンを数段階のレベルで注射した場合とコルチコステロンチューブ埋込みにより微量投与した場合, そして抗甲状腺物質 (PTU) を投与した場合の成長と脂肪蓄積の変化をブロイラーおよび卵用種ヒナを用いて 6 回の試験を行なって検討した。ブロイラーおよび卵用種ヒナにコルチコステロン (300-1200 μg /羽/日) を毎日 14 日間筋肉注射すると共に, PTU を飼料中に 0.05% 混合して給与する試験区も設定した。また, 両種のヒナを用いて, 2 日間の絶食の後に 4 日間飼料を再給餌し, その間にコルチコステロンを注射する試験を行なった。コルチコステロンを生理的微量で定常的に投与するために 14 日齢の卵用種ヒナにコルチコステロンを充填した Silastic チューブを皮下に埋込み, 21 日間の試験を行なった。コルチコステロン注射によりブロイラーおよび卵用種ヒナの成長が有意に低下し, 肝臓脂質含量と腹内脂肪含量はコルチコステロン投与レベルの増加に伴って著しく増加した。卵用種ヒナへのコルチコステロンチューブ埋込みによる微量投与 (22 μg および 43 $\mu\text{g}/\text{kg}$ 体重/日) の場合には成長の低下と肝臓脂質含量の増加は認められず, 腹腔内脂肪含量の有意の増加のみが見られた。絶食-再給餌した卵用種ヒナでもコルチコステロン注射による腹腔内脂肪の増加が認められた。PTU 投与により肝臓重量と腹腔内脂肪が増加し, PTU とコルチコステロン注射の組合わせにより腹内脂肪は著しく増加した。以上の結果から, コルチコステロンの分泌上昇と甲状腺機能低下は鶏における体脂肪蓄積に関与することが示唆された。

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