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EFFECT OF INDUCED STRESS BY DEXAMETHASONE ADMINISTRATION ON PERFORMANCE, EGG QUALITY AND SOME BLOOD PARAMETERS OF LAYING HENS

By

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Abstract: *Thirty four laying hens of a synthetic egg-type strain at their 46th week of age were housed individually in cages in an open sided building under a 16 hr light: 8 hr dark lighting schedule. Commercial feed and water were provided ad libitum. Birds were divided into two groups; the treated-group was given intramuscular injections of dexamethasone (4 mg/hen/day) for seven successive days and the control group was equally injected with physiological saline solution (0.9% NaCl). Body weight, egg production, feed consumption and egg quality characteristics were recorded. Total erythrocytes, total leukocytes, leukocytes differential counts and plasma glucose, cholesterol and calcium concentrations were measured.*

The main results obtained could be summarized as follows:

1. *Induced stress by injection of dexamethasone (DEX) for 7 days caused a significant decline in feed intake, feed conversion, egg production and egg quality characteristics.*

2. *Stressed hens produced thinner shells (0.260 mm) as compared to control ones (0.320 mm).*

3. *DEX administration caused a significant decrease in the proportion of lymphocytes and monocytes and a significant increase in heterophils, basophiles, eosinophils and H / L ratio.*

4. *Dexamethasone treated birds had significantly higher plasma glucose, cholesterol and calcium levels, than the control birds.*

INTRODUCTION

Hen eggs have an important part of the human diet since the dawn of recorded history and they are one of the few foods that are used throughout

the world. Egg quality of laying hens is influenced by several factors, including hen's age, strain, nutrition, housing system and stress.

Stress is considered one of the main factors affecting egg quality. As a result of stress, feed consumption, feed efficiency, egg production and shell quality decline and survivability (Miller and Sunde, 1975; De Andrade *et al.,* 1977; Emery *et al.,* 1984). It is declared that corticosterone administration delayed the onset of egg laying and inhibited reproduction. Etches *et al.* (1984) established that DEX administration or infusions of corticosterone blocked ovulation. Huang and Shirley (2001) observed that treatment of the follicles with increasing concentrations of dexamethasone suppressed LH and progesterone production in a dose-dependent manner.

Salvante and Williams (2003) proved that corticosterone-treated females had high plasma levels of very-low density lipoprotein (VLDL), suggesting that corticosterone inhibited yolk precursor production and perhaps shifted lipid metabolism away from production of yolk VLDL and towards production of generic (non-yolk) VLDL.

Recently, El-Lethey *et al.* (2001; 2003) proved that corticosterone feeding, induced stress as revealed by higher heterophil/ lymphocyte (H/L) ratios and reduced body weight. Huff *et al.* (2001) revealed that body weights of turkey poults were decreased by dexamethasone (DEX) treatment.

Treatment with dexamethasone (DEX) resulted in increases in WBC's counts, heterophils and monocytes and H / L ratio (Huff *et al.,* 2001). Gross and Siegel (1985) realized that H/L ratio could be used as an effective measure of stress in different strains of fowls. Moreover, corticosterone infusion induced higher H/L ratio than infusion of avehicle only (Donker and Beuving, 1989). Regarding to the effect of stress on serum glucose, cholesterol and calcium levels, Sahin and Kucuk (2001) declared that stress increased serum glucose, albumin, triglyceride and cholesterol and calcium concentrations. Moreover, Siegel (1995) noted that hypercholesteremia is one of many symptoms associated with long term stress. Also, Huff *et al.* (2001) who concluded that serum glucose was decreased in dexamethasone-*E. coli-challenged* birds (178 mg/dl) than that of controls (257 mg/dl). Similarly, Joseph and Ramachandran (1992) proved that dexamethasone treatment increased tissue glycogen contents and hypoglycaemia.

Therefore, the present study was carried out to shed more light on the effect of DEX-induced stress on performance, egg quality, blood parameters and some plasma components of laying hens.

MATERIALS AND METHODS

Thirty four laying hens of a synthetic egg-type strain at their 46th week of age were housed individually in cages with standard food (commercial corn-soybean meal type laying diet containing 2830 ME Kcal/ kg, 16% crude protein, 3.5% calcium, and 0.75% phosphorus). Food and water were available *ad libitum.* The birds were kept under a 16 hr light: 8 hr dark lighting schedule. Birds were divided into two groups; the treated-group was given intramuscular injections of dexamethasone (4 mg/hen /day) for seven successive days while the control group was equally injected with physiological saline solution (0.9% NaCl). Dexamethasone ampoules (as sodium phosphate) produced by Amriya Pharmaceutical Industries Co., Alexandria, Egypt.

Egg production, feed consumption and feed conversion were recorded daily. On the seventh day (2 hr after the last injection), produced eggs were collected for egg quality analysis egg weight, shell thickness, shell percentage, egg shape index, albumen height, albumen percentage, albumen index, Haugh Unit, yolk height, yolk index and yolk percentage. Total yolk cholesterol was determined according to Washburn and Nix (1974). Birds were individually weighed and blood samples (10 sample/ group) were obtained from brachial vein using heparinised syringes. Immediately following each sample, blood pH was measured. Total erythrocytes (RBC) and total leukocytes (WBC) were measured according to (Natt and Herrick, 1952). Blood smears were made and stained for differential leukocytic counts (100 cells/ smear; Cook, 1959) and means were calculated for heterophils, lymphocytes, monocytes, basophiles, eosinophils and the heterophil: lymphocyte (H/L) ratios were determined by dividing the number of heterophils by that of lymphocytes. Hemoglobin value (Hb) was determined using Hllige-Sahli's hemoglobin meter, using the acid-hematin method (Benjamin, 1961). Another blood samples were centrifuged and the collected plasma was stored at -20ºC until assay. Glucose was estimated in plasma by the method of Lott and Turner (1975), by using “Glucose GOD-PAP kits” which produced by Spinreact, S. A., Spain. Plasma cholesterol was determined by method of Richmond (1973), by using “Cholesterol CHOD-PAP kits” which produced by Human,

Germany, and calcium levels in plasma were assayed by method of Gindler and King (1972), by using "Ca-kit" which produced by Bio merieux, France.

Statistical Analysis:

Data were statistically analyzed using the General linear Models Procedure "GLM" of the SAS program (1999). Differences between treatments were subjected to Duncan's Multiple Range-test (Duncan, 1955).

The following model was used to study the effect of DEX injections on the parameters investigated as follows:

$Y_{ij} = \mu + E_i + e_{ij}$

Where,

Y_{ij} = an observations

μ = overall mean

E_i = effect of the induced stress

e_{ij} = Residual "random error"

RESULTS AND DISCUSSION

Responses to intramuscular dexamethasone (DEX) administration for body weight, feed consumption, feed conversion and egg production are shown in Table (1). Administration of the synthetic glucocorticoids (dexamethasone) did not significantly alter body weight. These results are not coincident with Leili and Scanes (1998) and Isobe and Lillehoj (1993) who confirmed that chickens treated with DEX intramuscularly or orally showed lower body weight gain than the untreated group. In other words, there was a dramatic weight loss in corticosteroids-treated birds. This weight loss was attributed, in part, to a reduction in absorptive efficiency from the intestinal distention caused by increased water intake (Lepkovsky *et al.,* 1960). Similarly, El-Lethey *et al.* (2003) proved that induced stress by feeding corticosterone had reduced body weight. The lack of such effect in the present experiment could be due to the fact that hens used in our study

had already reached their adult weight and/or to the relatively short term of treatment (7 days).

There was a significant (P = .001) difference in feed consumption due to DEX administration (Table 1). Birds treated by DEX consumed less feed (131.13 g/bird/day) than others injected by saline (172.98 g/day) also the feed conversion for birds treated by DEX was higher (9.83) than others injected by saline (2.17). Data reported by Donoghue *et al.* (1990) supported these results. They suggested that DEX infusion leads to anorexia which reduced the feed intake and consequently reduced body weights in DEX-treated hens.

Induced stress by daily injection of DEX for 7 days caused a significant (P≤.001) decline in egg production (Table 1). Mean hen-day egg production was 20.14% or 54.17% for stressed or control hens, respectively. It was observed that egg production was reduced sharply on the 6^{th} and 7^{th} day of administration. These responses are in agreement with the results obtained by Freeman (1971), Siegel (1980), Hill (1983) and El-Lethey *et al.* (2001; 2003). They noted that increases of circulating levels of corticosterone cause a reduction in egg production and also delayed the onset of egg laying and inhibited the reproduction. A possible explanation for this was suggested by Etches *et al.* (1984) who established that DEX administration or infusions of corticosterone blocked ovulation. These results were confirmed by Huang and Shirley (2001) who observed that treatment of the follicles with increasing concentrations of DEX suppressed LH and progesterone production. Similarly, Deitemeyer *et al.* (1985) indicated that DEX markedly inhibited the synthesis of prostaglandin E2. Another interpretation was offered by Vanmontfort *et al.* (1997) who revealed that Dexamethasone markedly decreased inhibin production by granulosa cells *in vitro*, and consequently, decreased the plasma inhibin concentration. Inhibin, a dimeric glycoprotein, is involved in the regulation of follicular development by providing negative feedback for the secretion of pituitary follicle-stimulating hormone (FSH) (Burger, 1988). Since DEX is liposoluble, it may preferentially accumulate in the yolk-filled preovulatory follicles and exert a prolonged suppressive effect on the production of inhibin by the surrounding granulosa layer (Decuypere *et al.,* 1997). Furthermore, a substantial amount of inhibin is found in the adrenals and is secreted by cultured adrenal cells *in vitro*. The adrenal contribution to inhibin secretion has also been demonstrated by the suppression of adrenal function by the administration of DEX (Vanmontfort *et al.,* 1997).

As shown in Table (2), DEX infusion had a significant effect on egg weight where DEX-treated hens produced smaller eggs (45.19 g) than the control hens (50.41 g). This result is in agreement with Donoghue *et al.* (1990) who declared that hyperthermic hens had reduced egg weights. Dexamethasone administration had a significant effect on shell thickness and shell percentage. Stressed hens produced thinner shells (0.260 mm) and controlled hens produced thicker shells (0.320 mm). Moreover, shell percentage in DEX-treated birds is smaller than control birds. Therefore, it could be summarized that shell quality of birds treated by DEX was significantly decreased. These shell quality responses are in agreement with the results obtained by Miller and Sunde (1975), De Andrade *et al.* (1976, 1977), Wolfenson *et al.,* 1979; and Emery *et al.* (1984). These authors postulated that stress had adversely affected shell quality and shell thickness. Factors suggested to be responsible for this reduction in shell quality include reduced feed intake (Payne, 1966), reduced blood flow to the reproductive tract (De Andrade *et al.,* 1977), disturbed blood acid-base balance (Smith, 1974), and/or reduced blood ionized calcium concentration (Odom *et al.,* 1986).

With respect to albumen and yolk quality, there are significant differences in albumen height, albumen index and Haugh Units between hens injected with DEX and those injected with physiological saline. Haugh Units in DEX-treated hens and controlled hens were 54.15 and 74.42; respectively and this is because albumen height in DEX-treated hens is lower than controlled hens. There are no significant differences in yolk height and yolk index due to DEX administration. On the other hand yolk percentage in controlled hens is greater than DEX-treated hens. This means that DEX causes decreases in yolk formation. These findings confirm the results of Salvante and Williams (2003) who indicated that corticosterone-treated females had high plasma levels of very-low density lipoprotein (VLDL), suggesting that corticosterone inhibited yolk precursor production and perhaps shifted lipid metabolism away from production of yolk VLDL and towards production of generic (non-yolk) VLDL. Thus, DEX-treated hens produce eggs of smaller weight and size as compared to those of controlled hens, with thinner shells, lower Haugh Units values and a lower percentage of yolk.

The effect of induced stress by infusion of DEX on total erythrocytes (RBCs), total leucocytes (WBCs) and hemoglobin content (Hb) is shown in Table (3). DEX administration had no significant effect on total RBC's but

birds treated with DEX had significantly (P≤.01) lower total WBC's (19.53 $x10^3$) and higher hemoglobin content (10.2 g/100ml). Many studies have demonstrated the alterations in the proportions of circulating blood cells following the administration of ACTH. This treatment produced a transient lymphopenia and leucocytosis (Gilck, 1961, 1962; Wolford and Ringer, 1962; Siegel, 1968). Likewise, Davison and Flack (1981) revealed that there was a leucopenia after 1 hr and a marked leucocytosis between 4 and 12 hr after ACTH injection. Heat stress exposure at 42ºC for 1-2 hr was responsible for a significant decrease in leucocytes and an increase in plasma corticosterone in adult cockerels (Ben Nathon *et al.,* 1976). On the other hand Huff *et al.* (2001) noted that treatment with DEX resulted in increases in WBCs counts; heterophil and monocytes, as well as H/L ratio.

Effect of administration of synthetic glucocorticoids (i.e., DEX) once daily for 7 days on leucocytes differential count is shown in Table (4). Results showed that DEX administration caused a significant decrease in the proportion of lymphocytes and monocytes and a significant increase in heterophils, basophiles, eosinophils and H/L ratio. In Table (4), it can be seen that percentage of heterophiles was higher in DEX-treated chickens (32.40%) than in the controls (24.64%).This situation is consistent with the results of Gross and Siegel (1983) who postulated that the number of heterophils increased in response to stressors and to increasing levels of corticosterone in the chicken feed. These findings are in agreement with results of other workers. Maxwell *et al.* (1992) concluded that in some poultry, a heterophilia may be the response to mild to moderate stress. Similarly, Gray *et al.* (1989) proved that ACTH treatment induced significant heterophilia.

The results shown in Table (4) state that DEX-treated birds had lower percentage of lymphocytes (51.8%) than controlled birds (59.6%). Similar results were obtained by Isobe and Lillehoj (1992, 1993) who confirmed that the percentage of lymphocytes, of chickens treated with DEX was significantly lower than in controls in a dose-dependent manner. Also, there was a regression of the thymus, spleen and bursa of Fabricius in birds following ACTH or corticosteroids injections (Siegel and Beane, 1961; Donker and Beuving, 1989). The regression includes depletion of lymphocytes from germinal centers (Glick, 1967) and concomitant lymphopenia and heterophilia in several avian species (Siegel, 1968). Similarly, high temperature (44 to 48^{o}C) show regression of lymphatic

tissues and depresses the numbers of peripheral blood lymphocytes within 2 hr of exposure (Chancellor and Glick, 1960).

By reason of heterophil percentage increased concomitantly with a decrease in lymphocyte percentage, the H/L ratio was increased by DEX administration. The data in Table (4) show that DEX injection induced higher H/L ratio (0.62) than the vehicle only (0.41). These types of observations were repeatedly reported by Huff *et al.* (2001) who noted that treatment with DEX resulted in increases in H/L ratio. Similarly, El-Lethey *et al.* (2001; 2003) proved that birds fed on corticosterone had higher H/L ratios. In addition, McFarlane and Curtis (1989) reported that a variety of stressors, alone or in combination, including increased ambient ammonia, a single electric shock and heat (35 ºC) for 7 days increased H/L ratios. Gross and Siegel (1983, 1985) realized that ratio could be used as an effective measure of social stress in different strains of fowls. Moreover, Siegel (1995) suggested that end-organ responses, such as heterophil and lymphocytes responses or lymphoid organ regression may represent better indicators of chronic stress, while plasma hormone (corticosteroids or catecholamine) concentrations are effective indicators of acute response.

Basophiles and eosinophils percentage in DEX-treated birds were significantly greater than those in saline-treated birds (Table 4). The proportions of basophiles and eosinophils in DEX-treated birds were 4.4% and 5.8%, respectively, while in controls they were 2.4% and 2.6%, respectively. These results comply with numerous reports. **Gray *et al.* (1989)** proved that ACTH treatment induced significant heterophilia, monocytosis, eosinophilia, and basophilia. Significantly elevated leucocyte counts and lymphopenia were observed with the high dosage of ACTH.

Laying hens treated for 7 days with DEX (4 mg/bird/day) had significantly ($P \leq .001$) higher plasma glucose, cholesterol and calcium levels (Table 5). Plasma glucose levels were increased in DEX- treated birds (386.6 mg/dl) relative to control birds (205.2 mg/dl). These results are in agreement with Kobayashi *et al.* (1989) who found that DEX increased the level of plasma glucose. Moreover, Freeman *et al.* (1979) noted that hyperglycemia had developed after 2 hr in birds which were injected with corticotrophin. Furthermore, a single injection of corticotrophin (Siegel and Beane, 1961) or a series of three injections at daily intervals (Bell, 1961) leads to hyperglycemia which persists for at least 24 hr. However, after 7 doses at daily intervals the birds are normoglycaemic after 24 hr. This

elevation in plasma glucose levels is attributed to enhancing of glycogenolysis (Stamler *et al.,* 1954; Snedecor *et al.,* 1963).

Exogenous corticosterone in the chicken increases plasma glucose concentrations (Saadoun *et al.,* 1987; Joseph and Ramachandran, 1992; Thurston *et al.,* 1993). Corticosterone-induced hyperglycemia in chickens is, in part, supported by enhancing of tissue glycogenolysis and hepatic glucose-6-phosphate (Joseph and Ramachandran, 1992). Corticosterone also induced circulating insulin (Bisbis *et al.,* 1994) but this is counteracted by down-regulation of insulin receptors in the liver (Taouis *et al.,* 1993) and suppression of postreceptor (after kinase activity) events in muscles and kidneys (Taouis *et al.,* 1993; Bisbis *et al.,* 1994).

These effects of exogenous corticosterone on intermediary metabolism may be mediated directly by specific receptors in the relevant target tissue. In addition, the effects may be indirect in that exogenous corticosterone produces changes in other hormones of intermediary metabolism such as thyroid hormones, growth hormone, prolactin, somatomedin C and/or norepinephrine (John *et al.,* 1987; Saadoun *et al.,* 1987).

As shown in Table (5), plasma cholesterol levels were increased significantly (P≤.001) in DEX treated birds (380.8 mg/dl) relative to control birds (173.4 mg/dl). Similarly, Siegel (1995) noted that hypercholesteremia is one of many symptoms associated with long term stress. However, Thaxton and Siegel (1973) reported that consistent changes in serum cholesterol and corticosterone were not found when heat, ACTH or combination of these with metyrapone "an inhibitor of adrenal corticosterone synthesis" were administered and measurements made at times from two hours to one week after treatments. The authors suggested that changes in circulating levels of cholesterol or corticosterone may have occurred at times other than those at which measurements were made.

The data in Table (5) show that plasma total calcium concentrations were significantly (P≤.001) higher in DEX-treated birds (28.44 mg/dl) than control hens (19.92 mg/dl). Similar results were reported by Huff *et al.* (2001) who noted that serum calcium levels were increased in birds dexamethasone-*E. coli* challenged birds compared to those of controls. Moreover, Sahin and Kucuk (2001) declared that stress by darkening and feed withdrawal treatments increased serum calcium, glucose, albumin,

triglyceride and cholesterol concentrations. It could be argued that, as in mammals, increases in corticosteroids inhibit skeletal calcification in growing birds and induce osteoporosis in adults (Siegel, 1995). The loss of skeletal calcium is reflected in higher than normal blood calcium (Siegel, 1968).

No significant differences were detected in blood pH due to DEX administration (Table 5). Blood pH in DEX treated birds was 7.89 and it was 7.81 in control birds indicating that blood pH was not elevated in DEX-treated hens by 2 hr of administration of DEX.

It could be mentioned that, administration of synthetic glucocorticoids (i.e., DEX) once daily for 7 days did not alter body weight but reduced the feed intake which negatively affected egg production. Also it had a significant negative effect on shell, albumen and yolk quality characteristics. Dexamethasone infusion changes the number of circulating leukocytes (in particular heterophilia and lymphocytopenia) and increased plasma levels of glucose, cholesterol and calcium.

Table (1): Effect of dexamethasone administration on laying performance.

Group	Body Weight (kg)	Feed Consumption (g/bird/day)	Feed conversion	Egg Production (%)
Dexamethasone	2.105± 0.10	131.13^{b} ± 6.58	9.83±1.60	20.14^{b} ± 3.22
Control	2.205± 0.10	172.98^{a} ± 5.00	2.17±0.22	54.17^{a} ± 3.22
Significance	NS	***	***	***

^{a,b} Means followed by different letters in the same column are significantly different.
^{NS} Non significant. *** ($P \leq .001$).

Table (2): Effect of dexamethasone administration on egg quality characteristics.

Characteristic	Dexamethasone	Control	Significance
Egg weight (g)	$45.19^{b} \pm 3.38$	$50.41^{a} \pm 2.79$	*
Shell thickness (mm)	$0.260^{b} \pm 0.22$	$0.320^{a} \pm 0.24$	*
Shell (%)	$9.41^{b} \pm 0.44$	$10.93^{a} \pm 0.36$	*
Egg shape index (%)	64.48± 4.92	74.28± 4.07	NS
Albumen height (mm)	$4.50^{b} \pm 0.47$	$5.56^{a} \pm 0.39$	*
Albumen (%)	54.88± 3.56	51.07± 2.94	NS
Albumen index (%)	$5.20^{b} \pm 0.68$	$6.88^{a} \pm 0.56$	*
Haugh Units	$54.15^{b} \pm 5.39$	$74.42^{a} \pm 4.46$	**
Yolk height (mm)	16.81± 0.95	18.47± 0.79	NS
Yolk (%)	$35.71^{b} \pm 2.55$	$38.00^{a} \pm 2.11$	NS
Yolk index (%)	37.04± 2.59	42.32± 2.14	NS
Cholesterol (mg/g yolk)	16.66± 0.14	15.96± 0.15	NS

^{a,b} Means followed by different letters in the same row are significantly different.
NS = Non significant. * (P≤ 0.05). ** (P≤0.01).

Table (3): Effect of dexamethasone administration on blood picture.

Group	Hb (g/ 100 ml)	RBCs ($x10^{6}$)	WBCs ($x10^{3}$)
Dexamethasone	$10.20^{a} \pm 0.19$	2.052± 0.02	$19.53^{b} \pm 0.68$
Control	$9.04^{b} \pm 0.19$	2.020± 0.01	$23.78^{a} \pm 0.68$
Significance	**	NS	**

^{a,b} Means followed by different letters in the same column are significantly different.
^{NS} Non significant. ** (P≤0.01).

Table (4): Effect of dexamethasone administration on leukocytic differential count.

Group	H	L	M	B	E	H/L Ratio
Dexamethasone	32.40^{a}± 0.31	51.8^{b}± 0.71	$6.6^{b} \pm 0.51$	$4.4^{a} \pm 0.6$	5.8^{a}± 0.71	$0.62^{a} \pm 0.01$
Control	24.64^{b}± 0.31	59.6^{a}± 0.71	12.6^{a}± 0.51	$2.4^{b} \pm 0.6$	2.6^{b}± 0.71	$0.41^{b} \pm 0.01$
Significance	***	***	***	*	**	***

^{a,b} Means followed by different letters in the same column are significantly different.
* (P≤0 .05). ** (P≤0.01). *** (P≤0.001).

Table (5): Effect of dexamethasone administration on plasma levels of glucose, cholesterol, calcium and pH.

Group	Glucose (mg/dl)	Cholesterol (mg/dl)	Calcium (mg/dl)	pH
Dexamethasone	386.60^{a}± 22.32	$380.80^{a} \pm 24.81$	$28.44^{a} \pm 0.42$	7.89± 0.06
Control	205.20^{b}± 22.32	$173.40^{b} \pm 24.81$	$19.92^{b} \pm 0.41$	7.81± 0.06
Significance	***	***	***	NS

^{a,b} Means followed by different letters in the same column are significantly different.
^{NS} =Non significant. *** (P≤0.001).

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الملخص العربى

تأثير الاجهاد الاصطناعى بالحقن بالدكساميثازون على الكفاءة الانتاجية وخواص البيض وبعض خصائص الدم فى الدجاج البياض

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أجريت هذه التجربة بمزرعة الدواجن التابعة لقسم انتاج الدواجن بكلية الزراعة بكفرالشيخ خلال شهر مارس 2004 وذلك بهدف دراسة تأثير تعرض الدجاج البياض للاجهاد الاصطناعى بالحقن بمادة الدكساميثازون على الكفاءة الانتاجية وجودة البيض الناتج وبعض خصائص الدم.

استخدم فى هذه التجربة اربعة وثلاثون دجاجة بياضة عمر 46 اسبوع وتم تسكينهم فى اقفاص فردية داخل مبنى مفتوح وتم تقديم العلف حتى الشبع.

تم تقسيم الدجاجات الى مجموعتين كالاتى:

المجموعة الاولى تم حقنها فى العضل بالدكساميثازون بمعدل 4 جم/دجاجة /يوم.

المجموعة الثانية تم حقنها بمحلول فسيولوجى (0.9% كلوريد صوديوم) واستمر الحقن لمدة 7 ايام متتالية.

- تم قياس وزن الجسم واستهلاك العليقة والنسبة المئوية لانتاج البيض وجودة البيض الناتج ومستوى الكولسترول فى الصفار.
- وتم اخذ عينات دم وذلك لقياس مستوى الهيموجلوبين ، عدد كرات الدم الحمراء وعدد كرات الدم البيضاء وانواعها
- وايضا تم قياس مستوى الجلوكوز فى الدم ومستوى الكولسترول فى الدم ومستوى الكالسيوم فى الدم وتركيز ايون الايدروجين فى الدم.

وكانت النتائج كالاتى :

1- ادى تعرض الطيور للاجهاد الاصطناعى عن طريق حقن الدكساميثازون لمدة 7 ايام الى انخفاض الاستهلاك الغذائى وكذلك انخفاض انتاج البيض.

2- اثر الاجهاد الاصطناعى عكسيا على جودة القشرة حيث كانت القشرة اقل سمكا (0.260 مم) فى الدجاجات التى تعرضت للاجهاد عنها فى الدجاجات الغير مجهدة (0.320 مم).

3- انخفضت وحدات هاوف فى البيض الناتج من الدجاجات المعرضة للاجهاد (54.15) عنها فى الاخرى التى لم تتعرض للاجهاد (74.42) وكان ذلك بسبب انخفاض ارتفاع البياض فى المجموعة الاولى.

4- نسبة الصفار كانت اعلى فى دجاجات مجموعة المقارنة عنها فى المجموعة التى تعرضت للاجهاد الاصطناعى.

5- ادى تعرض الطيور للاجهاد الاصطناعى بالحقن بالدكساميثازون الى خفض اعداد كرات الدم البيضاء وكذلك انخفضت نسبة الكرات الليمفاوية والكرات لاحادية بينما ارتفعت نسبة الكرات المتعادلة والقاعدية والحامضية وارتفعت النسبة بين الكرات المتعادلة والكرات الليفماوية.

6- ادى تعرض الطيور للاجهاد الاصطناعى بالحقن بالدكساميثازون الى ارتفاع مستويات الجلوكوز والكولسترول والكالسيوم فى البلازما.

وبناء على ما تقدم يمكن القول بأن لاجهاد الاصطناعى بالحقن بالدكساميثازون ادى الى التأثير عكسيا على الغذاء المأكول وعلى انتاج البيض وكذلك على جودة كل من القشرة والبياض والصفار كما ادى الى تقليل العدد الكلى لكرات الدم البيضاء ورفع مستويات الجلوكوز والكولسترول والكالسيوم فى البلازما.