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A COMPARATIVE STUDIES AMONG GOLDEN MONTAZAH, EL-SALAM AND FAYOUMI CHICKENS. 1- RESPONSE TO ACUTE HEAT STRESS AS EARLY HEAT CONDITIONING PROCEDURE.

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ABSTRACT: This study aimed to compare the response of three strains of domestic chickens to acute heat stress as early heat conditioning procedure and its consequences affects on growth, egg production and incubation performance. In this study 1200 one-day old female chicks from Golden Montazah (GM.), Fayoumi (Fy.) and El- Salam (Sm.)), 400 chicks from each strain were used. Chicks of each strain were equally divided into two groups (200 chicks each) with 4replicate of 50 chicks each. The first group served as control and reared without any treatments. The second group exposed to acute heat stress (40°C for 8hrrs) one time at five-day old as procedure to early heat conditioning. Respiration rate and body temperature were measured after acute heat stress (8 chicks /strain) from treatment and another (8 chicks /strain) were measured at the same time from control. Eight blood samples were collected from each strain in treatment group after acute heat stress and another eight were collected from each strain in control group to determine blood parameters.

Heated and control chickens were weighed at two, four month of age and at sexual maturity. For all replicates eggs counted, weighed daily, and average egg weight was calculated during the first six month of egg production. At 6th month of egg production three hatches were conducted, where (400 eggs/hatch) collected from each strain (200 eggs from each treatment) weekly at 50, 51 and 52 wk of age to study incubation response. At 6 month of egg production random samples of eggs representing controls and treatments (10eggs /replicate) for each strain were taken to estimate egg quality parameters. The

Key Words: Poultry, acute heat stress, early heat condition, egg production.

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following results were obtained:-

1- Treated chickens with acute heat stress increased significantly body weight at 4month of age and at sexual maturity than control. Responses of body weight to acute heat stress were upbeat for Fy. and GM. and was downbeat for Sm.
2- Treating GM. and Fy. with acute heat stress decreased age at sexual maturity, in contrast treating Sa. increased age at sexual maturity.
3- Treating chicks with acute heat stress increased significantly egg production percentage.
4- Acute heat stress didn't influence on incubation performance.
5- Acute heat stress did not affect the quality of the eggs, on the other hand albumin parameters of GM. and Sa. were higher significantly than Fy
6- Application of acute heat stress procedure led to increase in survivability chicken. Golden Montazah had significantly the highest survival percent, while Sa. had significantly the lowest once.
7- Acute heat stress increased significantly respiration rate and body temperature.
8- Lymphocytes% decreased significantly and heterophils % and H/L ratio increased significantly by application of acute heat stress procedure.
9- Plasma protein concentration, Alanine transaminase, Aspartate transaminase and Glutathione peroxidase GSHPx increased significantly by acute heat stress.
10- Triiodothyronine (T_3) and thyroxine (T_4) hormones are decreased significantly by acute heat stress, on contrast Corticosterone (Cs.) increased significantly.

INTRODUCION

Environmental factors can greatly decrease genetic progress. Clearly random environmental factors are a hindrance to breeding superior genetic stock. A high ambient temperature in Egypt during summer season generates a status of the stress. In hot climates, poultry production is generally reduced, due to the rise in environmental temperature. Heat stress causes many biochemical and physiological changes in the bird such as: Shift in acid-base balance, hyperthermia, increased production of free radical and corticosterone (Mebta and Sbingari, 1999). The expression of heat stress in poultry production field can be described as 'acute' or 'chronic'. Acute heat stress refers to sudden and short periods of extremely high temperature, whereas chronic heat stress refers to extended periods of elevated temperature (Abu-Dieyeh, 2006).

Early heat conditioning seems to be one of the most promising methods of enhancing the heat resistance of chickens. Early heat conditioning refers to the practice exposing chicks to high temperature at 3 to 5 d of age. This procedure induces the heat tolerance of broiler chickens. Now conditions of heat stress take more consideration in poultry production due to the rapid development of poultry industry in hot climate countries and to the reduced performance of poultry during summer months in temperate countries (Lin et al.,2006a). El-Moniary et al. (2010) reported that subjecting broiler chicks to early age heat conditioning at 5 days old improved their productivity of under summer stress conditions. They added that average body weight, weight gain, feed intake and feed conversion ratio were significantly improved at 42 days of old. Yahav and Mc Murtry (2001) reported

that early thermal conditioning (3 and 5days) of chicks resulted improvements in performance and thermotolerance in marketing age moreover, the highest body weight was coincided with low feed intake and significantly higher feed efficiency. Basilio et al. (2001) stated that the effects of early age thermal conditioning treatment resulted in improved body weight gain (P <0.03). Soleimani et al., (2011) reported that genetic differences in body size and age per se may not determine breed or strain variations in response to heat stress.

Several studies were conducted to study the effect of high temperature on the immune responses of chickens. High environmental temperatures affect the development of specific immune responses in young chickens. These effects include the suppression of circulating WBC. and an increase in the heterophil/lymphocyte ratio (H/L ratio), which is an indicator of stress (Mashaly et al.,2004). Heat stress was also reported to cause a reduction in antibody production in young chickens (Zulkifi et al.,2000). They added that heat-induced immunosuppression may depend on breed of bird and its effects on immune responses may depends on the length and intensity of the heat exposure.

Oxidative stress was observed in exposure to acute heat stress (Mujahid et al.,2005a; Mujahid et al.,2006; Lin et al.,2006b and Mujahid et al., 2007). Superoxide is a reactive molecule, but it can be converted to hydrogen peroxide by superoxide dismutase and then to oxygen and water by glutathione peroxidase GSHPx (Mujahid et al.,2005b).

Heat stress physiological responses can be assessed by recording panting and body temperature as well as hematological variables. When birds were challenged by heat stress, their body temperatures increased (Borges et al.,2004).

MATERIALS AND METHODS

This study aimed to compare the response of three strains of domestic chickens to acute heat stress. In this study 1200 one-day old female chicks from Golden Montazah (GM.), Fayoumi (Fy.) and El- Salam (Sm.)), 400 chicks from each strain were chosen and wing banded. Female chicks of each strain were randomly divided into two groups (200 chicks each) with 4replicate of 50 chicks each..The first group served as control and reared without any treatments. The second group exposed to acute heat stress (40°C for 8hrrs) one time at five-day old. During brooding and rearing period, experimental birds were maintained under the same managerial conditions of feeding, lighting and health programs and housing. Chicks received a commercial starter ration (1-6wk of age) and grower ration (7-18wk of age) and then switched to layer ration according to NRC (1994). The experimental lasted for 12 months, where the plan of the experiment and average monthly high and low temperatures (^{o}C) and relative humidity (%) inside stockyard were summarized in (Table,1). Directly after acute heat stress, 2 blood samples were taken from each replicate within each strain in treated groups another 2 blood samples were taken from each replicate within each strain in control groups at the same time. Blood samples were collected from wing vein in heparinized and non-heparinized test tubes. Fresh blood samples was taken to determine hemoglobin (Hb), hematocrit (Ht), total count of red blood cells (RBCs), total count of white blood cells (WBCs) and their differentiations (Heterophils%, lymphocytes%, H/L ratio, monocytes%, eosinophils% and basophils%). The reset of blood samples were immediately centrifuged at 3000 rpm for 15 min. Plasma and serum separated and stored at -20 ^{o}c. Glutathione peroxidase enzyme activity (GSHPX) was determined by enzymatic

methods, adjusted for poultry blood using available commercial kits SCLAVO INC., 5 Mansard Count., Wayne NJ 07470, USA. Triiodothyronine (T_3) and thyroxine (T_4) were determined by using commercial kits. Total plasma corticosterone was measured using a commercially available 125I corticosterone RIA kit (catalog no. 07-120122, MP Biomedicals, Solon,OH). Plasma total protein and Albumin concentration were quantitatively measured based on colorimetric determination. Globulin concentration was estimated by subtraction of albumin concentration from serum total protein value.

Respiration rate (R.R.) and body temperature (B.T.) were measured directly after acute heat stress (8chicks/strain) from treated chicks and another (8chicks/strain) from control. Body temperature was measured as a rectal temperature (°C) by inserting thermometers approximately 1 cm into each chick (8/strain) via the rectum. Respiration rate (breaths/min) was obtained by counting the wave cycles associated with respiratory movement.

Pullets were weighed at 2 month, 4 month of age and at sexual maturity and body weight gain were then calculated. Age at sexual maturity was recorded when the egg production percent reached 10% per replicate. Eggs were weighed daily, egg numbers were recorded and egg mass per hen per day were calculated. Egg mass production per hen per day was calculated as laying percentage multiplied by average egg weight of the hen (daily egg mass production, g/hen per d) according to Bonekamp et al., 2010. Feed intake was determined per replicate. Feed conversion ratio was calculated as gram feed consumption per hen per day divided by gram egg mass per hen per day according to EL-Husseiny et al.,2008.

At 6^{th} month of egg production three hatches were conducted, where 400 eggs collected from each strain (200 eggs from each treatment) weekly at 50, 51 and 52 wk of age and stored at 18°C and then set in a chick master incubator at 37.5°C and 55% RH. Eggs were candled at day 7 and 14 of incubation and transferred for hatching on d 18 of incubation. During candling, eggs were characterized as being infertile or containing early dead embryos (less than 7 d) or late dead embryos (more than 7d and less than 14 d). At the end of incubation period un-hatched eggs were broken to determine deformed (Abnormalities) embryos.At 6 month of egg production random samples of eggs representing controls and treatments (10eggs /replicate) for each strain were taken to estimate egg quality parameters as albumin weight %, yolk weight% and shell weight%. Albumin height was measured in millimeters by using tripod micrometers. Haugh unit was calculated according to (Haught, 1937). Yolk index and yolk color were estimated. The shell thickness was measured to 0.01mm accuracy with a micrometer. The following equation was used to calculate shell surface area (Su) according to Carter (1975).

$$Sa = 0.9109 \times (\text{egg length} \times 0.289) \times (\text{egg breadth} \times 0.3164) \times (\text{egg weight} \times 0.488)$$

Shell weight per unit of surface area (Sw/Sa)was calculated by divided shell weight/surface area according to Hamilton (1978).

Statistical analyses:

General linear model: general factorial design procedure of SPSS. 16 (2007) procedure was used to test variance mains. Along with Duncan's multiple range test to separate means (Duncan, 1955). All statements of significance were based on a probability level of (0.05).

RESULTS AND DISCUSSION

Body weight and body weight gain:

Acute heat stress increased body weight significantly at 4 month of age and at sexual maturity, On the contrary body weight at two month of age and body gain didn't differ significantly (Table, 2). Response of body weight at 2nd month of age to acute heat stress were +5.33%, -3.17% and +5.36% for GM., Sa. and Fy. respectively. The response were +0.06%, -0.06%, and +0.10% at 4th for GM., Sa. and Fy. Respectively. Simply, response of body weight at sexual maturity to acute heat stress were +0.06%, -0.03%, +0.05% for GM., Sa. and Fy. Respectively. The results indicated that response of El salam was negative, on contrast response of Fayoumi and golden Montazah were positive. This may be due to strain responses. In this respects El-Gendy et al. (2007) reported that the effect of heat on growth was breed-dependent. Moreover Yalcin et al. (2001) and El-Gendy et al. (2006) indicated that breed differences in resistance to high temperature were associated with the breed differences in body weight.

Egg production parameters:

Table (2) shows that treating GM. and Fy. with acute heat stress insignificantly decreased age at sexual maturity. Treating chicks with acute heat stress increased significantly egg production percentage. Heat treated Sa. and Fy. hens had significantly higher values than their control. Average egg weight and total egg mass had approximately similar trend. Regarding the effect of strains figure 1 showed that GM. hens had the highest monthly egg production during summer months than Fy. and Sa. Treated hens had higher monthly egg production percent (figure, 1) than control.

Treating chicks with acute heat stress didn't effect significantly on feed intake and feed conversion. In contrast feed intake and feed conversion were affected significantly by type of strain. The results in agreement with Bollengier-Leeet al. (1998) who reported that food intake were unaffected by heat treatment. Improving in egg production were 2%, 4% and %8 for GM., Sa. and Fy hen respectively, in generally improving egg production of Fayoumi strain may be due to slower growing strains of native or indigenous breeds of chickens in tropical countries are better able to withstand high ambient temperatures than faster growing strains (Cahaner, et al., 2008).

Survival percent:

Application of acute heat stress led to increase in survivability chicken (Table, 2). The results in agreement with (El-Moniary et al., 2010) who reported that exposure of 5-day-old broiler chicks to elevated temperature improved survivability. Golden had significantly the highest survival percent, while Sa. had significantly the lowest. Decreasing survival of Sa. may be due to Sa. was declined from meat breeder strains that couldn't resist higher ambient temperature. Increasing survival of GM. compared with some native and other development strains were reported by (Amin, 2008a; Amin, 2008b; El Full, et al.,2005; and Osman , et al.,2003).

Fertility, hatchability, dead and abnormalities percent:

Table (3) showed that acute heat stress not influenced on measures of hatching. Regarding breed effect Fy. had numerically the highest fertility % and G.M. had the lowest one. In contrast Fy. had numerically the lowest hatchability per total eggs and hatchability per fertile eggs % but G.M. had the highest one. The result agree with El Full et al., (2005) who found that Fy. had higher fertility than G.M. and

Sa. In contrast Kosba et al., (2008) reported that G.M. had higher fertility and lower hatchability than Fy.

Egg quality:

Acute heat stress didn't influence significantly on egg quality (Table, 4). Albumin quality of GM. and Sa. were higher significantly than Fy. Increasing hough unit and albumin percent of developed chickens significantly agree with El Full et al., (2005) and Osman et al., (2003) who demonstrated that developed chickens had higher hough unit and albumin percent than other native strains.

Fayoumi had higher yolk quality than GM. Increased yolk color and yolk weight % for Fy. agree with El Full et al., (2005).

Respiration rate and body temperature:

Employed acute heat stress significantly increased respiration rate (Table, 5). This may be due to evaporative heat-dissipation mechanism that maintains normal body temperature (Odom et al., 1986). Moreover, Kalamah (2001); El-Sheikh et al., (2004) and Faisal1 et al., (2008) postulated that the increased respiration rate (Panting Phenomenon) is desirable in hot weather to loss heat via evaporative cooling from respiratory passage.

Acute heat stress significantly increased B.T.. This result is in agreement with Donkoh, (1989) who found significant increases in B.T. occur when environmental temperatures significantly increased. The lack of response of the blood system to acute temperature changes may be at least partially responsible for the chickens' failure to control body temperature (Yahav et al., 1997). El-Salam strain had significantly the lowest B.T. and Fy. had significantly the highest respiration rate. This difference in thermoregulation ability of heat production or heat loss may be under genetic control (Uneo and Komiyama, 1987).

Complete blood count:

1- Hemoglobin, hematocrit and red blood cell:

Acute heat stress numerically decreased hemoglobin, hematocrit and R.B.C. (Table, 6) for Fy. and Sa. chickens. Which in line with those of Islam et al., (2004) and Ajakaiye et al., (2010) who reported that exposure to heat stress decreased hematocrit, hemoglobin and total R.B.C.. This may be due to, terminally differentiating avian red blood cells are capable of responding to heat stress by rapid changes in their highly restricted "program" of gene expression (Atkinson, et al., 1986).

2- White blood cells:

Results in table (5) showed that acute heat stress caused insignificant increase in WBC count, and caused significant increase in heterophils% and H/L ratio. Increasing heterophils% may be due to increasing Corticosterone (Cs). where , Post et al., (2003) reported that directly after Cs-treatment a rapid increase of heterophils frequencies in the blood was observed. Lymphocytes% significantly decreased after acute heat stress. This may be due to stress-induced reductions in circulating lymphocyte numbers that are not due to large-scale destruction of cells, but rather to glucocorticoid-induced alterations in the 'trafficking', or redistribution, of lymphocytes from the blood to other body compartments (Dhabhar 2002). In response to glucocorticoids, circulating lymphocytes adhere to the endothelial cells that line the walls of blood vessels, and subsequently undergo transmigration from circulation into other tissues, for example lymph nodes, spleen, bone marrow and skin, where they are sequestered. These changes

are thought to ensure that the different types of cells are routed to where they are needed during the stress response ((Dhabhar 2002).

Biochemical blood parameters:

1- Protein, Globulins and albumin:

Plasma protein concentration decreased significantly after acute heat stress, which in agreement with Geraert, et al., (1996) who reported that at high ambient temperatures decreased protein synthesis. Probably due to reduced plasma amino acid concentration and lower energy supply, as observed in broiler chicken muscle tissue (Temim et al., 2000). In addition, heat stress decreases plasma T_3 concentration and increased plasma Cs., both changes known to reduce protein deposition through alterations in protein turnover in birds and other species (Yunianto et al., 1997.). Concerning globulins decreasing the results were in harmony with those reported by (Zulkifi et al., 2000) and (Mashaly et al., 2004) who showed that heat stress caused a reduction in antibody production. This reduction could be indirectly due to an increase in inflammatory cytokines under stress. Cytokines stimulate the hypothalamic production of hypothalamic corticotrophin-releasing factor (CRF) (Ogle et al., 1997). Gross (1992) stated that CRF is known to increase ACTH from the pituitary; ACTH then stimulates Cs. production from the adrenal gland, Cs. in turn inhibits antibody production and heat stress is known to decrease cytokines (Wang et al., 2001), which are important for antibody production (Lebman and Coffman, 1988).

2- Enzymes:

Alanine transaminase (ALT) and Aspartate transaminase (AST):

Golden montazah strain had significantly the lowest ALT and numerically the lowest AST values (Table,6). The enzymes were increased by application of acute heat stress. These results are in harmony with those obtained by Faisal et al.,2008 who reported that activities of plasma ALT were significantly increased in heat treated groups.

Glutathione peroxidase GSHPx:

Table 6 cleared that acute heat stress significantly increased GSHPx. This may be due to acute exposure to high temperatures may depress the activity of the mitochondrial respiratory chain. This activation results in overproduction of Reactive Oxygen Species, which ultimately results in oxidative injury (Tan, et al.,2010) and Oxidative stress results from an imbalance between free radical generation and antioxidant defense systems (Sandhu and Kaur, 2002). Antioxidant enzymes such as Superoxide Dismutase, Catalase , and GSHPx. are the first line defense antioxidants (Ray and Husain, 2002).

3- Hormones:

Triiodothyronine (T_3) and Thyroxine (T_4):

The T_3 and T_4 hormones (Table, 6) are significantly decreased by acute heat stress. This may be due to T_3 and T_4 hormones were involved in the thermoregulatory process (Decuypere, and Kiihn, 1988) and these results agree with (Geraert, et al.,1996) who reported that heat stressed chickens had reduced plasma T_3 and increased plasma Cs. Brenner et al., (1998) revealed that heat stress significantly reduced the serum concentrations of T_3 and T_4. Moreover, Garriga et al., (2006) reported that heat stress significantly reduced T_3 and T_4 by (52 and 37%, respectively). The T_3 and T_4 can be used as an indicator of the physiological adaptation (Sohail et al.,2010).

Corticosterone (Cs.):

Acute heat stress significantly increased Cs. (Table, 6). This findings are correspond with Garriga et al., (2006) who reported that heat stress induces a significant increase in plasma Cs. (a90% increase compared with birds kept in thermo-neutral conditions). After acute heat stress Sa. had significantly the lowest Cs. The differences in results may be due to there are strain differences in the regulation of the adrenal function and cell-mediated immunity (Fahey and Cheng., 2008). Acute heat stress increased Cs. by simultaneously CRF and vasopressin secretion stimulate release of adrenocorticotropic hormone (ACTH), and resulting glucocorticoid secretion from the adrenal cortex (Dohms and Metz, 1991). These compounds can exert direct effects on the immune system. Catecholamines are released directly into lymphoid tissue as a result of direct innervation by sympathetic and parasympathetic nervous systems. The lymphocytes have high-affinity receptors for ACTH and glucocorticoids that are up regulated by immune stimulation (Mumma et al.,2006). Lymphocytes can also regulate the stress response since they can synthesize Cs. and other neuroendocrine mediators (Mashaly et al., 1993).

CONCLUSION

Application of acute heat stress at 5 days of age (early heat conditioning) increased growth for Fy and GM., while treated Sa. with acute heat stress had adverse effect. Early heat condition improved egg production by 2%, 4% and %8 for GM., Sa. and Fy. hen respectively.

Table (1): Experimental design.

Date	Chickens age (month)	Procedure	House temperature		Relative humidity
			Maximum	Minimum	
15th December 2011-14h January 2012	1	Acute heat stress 5th day of age	29.4	27.7	35
15th January 2012-14th February2012	2	Body weight 2nd month	25.9	22.3	45
15th February 2012-14th March2012	3	. . . . .	21.0	13.7	40
15th March 2012-14th April2012	4	Body weight 4th month	24.6	16.8	35
15th April 2012-14th May 2012	5	. . . . .	29.0	18.2	30
15th May 2012-14th June 2012	6	Body weight sexual maturity-1st month of egg production	35.0	20.6	20
15th June 2012-14th July 2012	7	1st – 2nd month of egg production	40.5	26.1	20
15th July 2012-14th August 2012	8	2nd – 3rd month of egg production	42.1	29.9	35
15th August 2012-14th September 2012	9	3rd – 4th month of egg production	41.4	29.1	30
15th September 2012-14th October 2012	10	4th – 5th month of egg production	38.2	25.4	30
15th October 2012-14th November 2012	11	5th – 6th month of egg production	32.1	18.7	20
15th November 2012-14th January 2013	12	6th month of egg production -incubation -egg quality	19.4	11.7	20

Table (2): Effect of acute heat stress on body weight and egg production performances.

Parameters	Body weight			Body gain		
	2nd month	4th month	S. M.	4th month - 2ndmonth	S. M.- 2ndmonth	S. M - 4thmonth
Heat stress						
Control	646.67	1086.66^{b}	1303.33^{b}	440.00	656.67^{b}	216.67
Acute	663.33	1116.66^{a}	1336.66^{a}	453.33	673.33^{a}	220.00
SE.	±8.16	±9.354	±8.28	±12.64	±10.13	±11.48
Pv.	0.17	0.04	0.01	0.47	0.02	0.84
Breed						
GM.	770.00^{a}	1255.00^{a}	1450.00^{a}	485.00^{a}	680.00^{a}	195.00^{b}
Sa.	620.00^{b}	1114.99^{b}	1287.50^{b}	495.00^{a}	667.50^{b}	172.50^{b}
Fa.	575.00^{c}	935.00^{c}	1222.50^{c}	360.00^{b}	647.50^{c}	287.50^{a}
SE.	±10.00	±11.46	±10.14	±15.48	±12.40	±14.06
Pv.	0.00	0.00	0.00	0.00	0.02	0.00
Treatments						
Control * GM.	750.00^{a}	1220.00^{b}	1410.00^{b}	470.00^{a}	660.00^{ab}	190.00^{b}
Control * Sa.	630.00^{b}	1149.99^{c}	1305.00^{c}	520.00^{a}	675.00^{ab}	155.00^{b}
Control * Fa.	560.00^{c}	890.00^{f}	1195.00^{e}	330.00^{b}	635.00^{b}	305.00^{a}
Acute * GM.	790.00^{a}	1290.00^{a}	1490.00^{a}	500.00^{a}	700.00^{a}	200.00^{b}
Acute * Sa.	610.00^{b}	1079.99^{d}	1270.00^{cd}	470.00^{a}	660.00^{ab}	190.00^{b}
Acute* Fa.	590.00^{bc}	980.00^{e}	1250.00^{d}	390.00^{b}	660.00^{ab}	270.00^{a}
SE.	±14.14	±16.20	±14.34	±21.89	±17.54	±19.88
Pv.	0.010	0.00	0.00	0.04	0.029	0.023

a,b, c… means with different letter in each column for every trait are differ significantly ($P \leq 0.05$).

S. M.= sexual maturity Egg prod%= egg production percent

Feed conv.= feed conversion

Cont. Table (2):

Parameters	Egg production						Survival
	Age at S. M.	Egg prod%	Average egg weight	Egg mass	Feed intake	Feed conv.	
Heat stress							
Control	161.79	39.79^{b}	43.22	18.38^{b}	93.86	5.10	90.56
Acute	161.32	41.70^{a}	43.72	19.43^{a}	93.83	4.82	89.44
SE.	±.03	± 0.79	± 0.29	± 0.41	±0.63	±0.53	±1.67
Pv.	0.86	0.001	0.24	0.04	0.95	0.12	0.64
Breed							
GM.	159.44	52.09^{a}	48.08^{a}	26.26^{a}	98.83^{a}	3.76^{b}	90.00
Sa.	169.91	35.29^{b}	44.58^{b}	16.58^{b}	93.84^{b}	5.67^{a}	91.67
Fa.	155.31	34.85^{b}	37.75^{c}	13.94^{c}	88.85^{c}	6.37^{a}	88.34
SE.	±0.04	± 0. 97	± 0.36	± 0.51	±0.77	±0.63	±2.04
Pv.	0.24	0.01	0.01	0.01	0.01	0.02	0.53
Treatments							
Control * GM.	160.42	51.55^{a}	47.84	25.85^{a}	98.84^{a}	3.82^{c}	91.67
Control * Sa.	168.24	34.52^{c}	44.34	16.07^{bc}	93.84^{b}	5.83^{b}	93.33
Control * Fa.	156.72	33.30^{c}	37.50	13.25^{d}	88.84^{c}	6.70^{a}	86.67
Acute * GM.	158.46	52.64^{a}	48.33	26.68^{a}	98.83^{a}	3.70^{c}	88.33
Acute * Sa.	171.59	36.06^{b}	44.83	17.05^{b}	93.83^{b}	5.51^{b}	90.00
Acute* Fa.	153.91	36.39^{b}	38.02	14.64^{cd}	88.82^{c}	6.06^{a}	90.00
SE.	±0.06	± 1.37	± 0.51	± 0.72	±1.09	±0.91	±2.89
Pv.	0.16	0.02	0.51	0.03	0.04	0.04	0.43

a,b, c… means with different letter in each column for every trait are differ significantly ($P \leq 0.05$).

S. M.= sexual maturity Egg prod%= egg production percent

Feed conv.= feed conversion

Table (3): Effect of acute heat stress on incubation parameters.

Parameters	Fertility	Hatch-ablity$_1$	Hatch-ability$_2$	Early dead	Late dead	Abno-rmalities
Heat stress						
Control	91.23	82.58	90.52	5.80	1.74	1.10
Acute	90.11	80.72	89.58	6.06	1.88	1.45
SE	± 0.42	± 1.06	± 0.84	± 0.64	± 0.14	± 0.15
Pv	0.11	0.26	0.46	0.78	0.52	0.16
Breed						
GM	90.24	81.85	90.69	5.60	1.75	1.05
Sa	90.53	81.70	90.25	5.70	1.79	1.34
Fa	91.24	81.41	89.21	6.50	1.89	1.43
SE	± 0.52	± 1.30	± 1.03	± 0.78	± 0.17	± 0.19
Pv	0.43	0.97	0.60	0.69	0.82	0.39
Treatments						
Control * GM	90.43	83.23	92.04	4.90	1.66	0.65
Control * Sa	90.35	81.90	90.66	5.30	1.85	1.29
Control * Fa	92.90	82.61	88.88	7.22	1.72	1.35
Acute * GM	90.05	80.47	89.35	6.30	1.83	1.45
Acute * Sa	90.70	81.49	89.85	6.10	1.73	1.39
Acute* Fa	89.58	80.21	89.53	5.79	2.07	1.52
SE	± 0.73	± 1.83	± 1.46	± 1.11	± 0.24	± 0.27
Pv	0.10	0.80	0.55	0.45	0.64	0.40

a,b, c… means with different letter in each column for every trait are differ significantly ($P \leq 0.05$).

Hatchablity1=Hatchability per fertile eggs

Hatchability2 =Hatchability per total eggs

Table (4): Effect of acute heat stress on egg quality parameters.

Parameters	Albumin quality		Yolk quality			Shell quality			
	Alb%	**Hu. U**	**Yk%**	**Yk.In.**	**Yk.C.**	**Sh.%**	**Sh.th**	**Su**	**Sw/Su**
Heat stress									
Control	59.31	85.09	30.748	37.42	7.84	9.94	4.93	62.49	79.05
Acute	59.30	85.61	31.013	37.47	8.04	9.69	4.71	61.82	76.38
SE	±.425	±1.23	±.346	±0.419	±.22	±.212	±.108	±0.46	±1.66
Pv	0.978	0.768	0.590	0.937	0.526	0.410	0.150	0.302	0.26
Breed									
GM	61.43^{a}	90.31^{a}	29.593^{b}	35.84^{b}	7.43^{c}	8.98^{b}	4.47^{b}	62.60^{b}	71.26^{c}
Sa	59.43^{b}	87.86^{a}	31.004^{a}	38.86^{a}	8.17^{a}	9.57^{b}	5.17^{a}	66.16^{a}	77.91^{b}
Fa	57.06^{c}	77.87^{b}	32.044^{a}	37.64^{a}	8.23^{a}	10.89^{a}	4.83^{ab}	57.71^{c}	83.96^{a}
SE	±.520	±1.51	±.423	±0.51	±.27	±.259	±.133	±0.56	±2.03
Pv	0.000	0.000	0.000	0.000	0.035	0.000	0.002	0.000	0.001
Treatments									
Control * GM	61.28	92.54^{a}	29.593	36.65^{bc}	7.27	9.13	4.60	63.07^{bc}	72.91
Control * Sa	59.24	88.64^{a}	30.936	39.68^{a}	8.27	9.83	5.47	67.57^{a}	80.63
Control * Fa	57.43	74.09^{c}	31.716	35.93^{bc}	8.00	10.86	4.73	56.83^{d}	83.60
Acute * GM	61.57	88.08^{a}	29.592	35.03^{c}	7.60	8.84	4.33	62.13^{c}	69.62
Acute * Sa	59.62	87.09^{ab}	31.073	38.03^{ab}	8.07	9.31	4.87	64.76^{b}	75.19
Acute* Fa	56.70	81.65^{b}	32.373	39.34^{a}	8.47	10.93	4.93	58.58^{d}	84.33
SE	±.736	±2.127	±.599	±0.726	±.384	±.366	±.187	±0.791	±2.87
Pv	0.705	0.016	0.845	0.001	0.658	0.722	0.107	0.019	0.553

a,b, c… means with different letter in each column for every trait are differ significantly ($P \leq 0.05$).

Alb%	=	Albumin weight percent	Yk%	=	yolk weight percent
Sh.%	=	Shell weight percent	Hu.U.	=	Hough unit
Yk.In.	=	Yolk index	Yk.C.	=	Yolk color
Sh.th	=	Shell thickness	Su	=	Surface aria
Sw/Su	=	Shell weight/Su			

Table (5): Effect of acute heat stress on thermoregulation, red blood cell profile and cellular immunity of three local chickens.

Parameters	Thermoregulation		Red blood cell profile					
	R. R.	B. T.	Hb (GM/dl)	Ht%	R.B.C. × 10^6	MCV	MCH	MCHC
Heat stress								
Control	49.40^{b}	42.51^{b}	13.32	36.00	3.18	138.23	41.58	30.07
Acute	81.67^{a}	42.69^{a}	10.55	32.67	2.73	136.50	39.27	37.32
SE	±1.50	±0.03	±0.80	±1.35	±0.19	±3.21	±1.05	±6.46
Pv	.00	0.02	0.09	0.13	0.15	0.72	0.17	0.46
Breed								
GM	64.60^{b}	42.68^{a}	10.13	32.50	2.65	134.25	38.35	28.58
Sa	62.00^{b}	42.39^{b}	13.08	35.75	3.17	132.73	41.65	43.95
Fa	70.00^{a}	42.72^{a}	12.60	34.75	3.05	145.13	41.28	28.55
SE	±1.84	±0.04	±0.98	±1.66	±0.24	±3.93	±1.29	±7.92
Pv	0.01	0.004	0.15	0.42	0.33	0.13	0.22	0.35
Treatments								
Control * GM	48.60^{c}	42.60^{bc}	10.10	33.50	2.55	132.90	39.40	29.60
Control * Sa	46.60^{c}	42.30^{d}	14.65	36.50	3.40	141.00	43.05	30.55
Control * Fa	53.00^{c}	42.62^{bc}	15.20	38.00	3.60	140.80	42.30	30.05
Acute * GM	80.60^{ab}	42.76^{ab}	10.15	31.50	2.75	135.60	37.30	27.55
Acute * Sa	77.40^{b}	42.48^{c}	11.50	35.00	2.95	124.45	40.25	57.35
Acute* Fa	87.00^{a}	42.82^{a}	10.00	31.50	2.51	149.45	40.25	27.05
SE	±2.60	±0.05	±1.38	±2.35	±0.34	±5.56	±1.82	±11.19
Pv	.0.00	0.03	0.24	0.54	0.24	0.14	0.97	0.38

a,b, c… means with different letter in each column for every trait are differ significantly ($P \leq 0.05$).

R. R = Respiration rate
B. T. = Body temperature
Hb = hemoglobin concentration
Ht% = hematocrit percent
R.B.C. = red blood cells count
MCV = Mean corpuscular volume
MCH = mean corpuscular hemoglobin
MCHC = mean corpuscular hemoglobin concentration
W.B.C. = White blood cells count
H % = heterophils%
L % = Lymphocytes%
H/L = heterophils/ Lymphocytes ratio
Mn% = Monocytes%
Es% = Eosinophils%
Bs% = .=Basophils%

Cont. Table (5):

Parameters	Cellular immunity						
	W.B.C. × 10^3	**H %**	**L %**	**H/L ratio**	**Mn.%**	**Es.%**	**Bs.%**
Heat stress							
Control	13.38	25.58[b]	68.33[a]	0.38[b]	3.65	1.34	1.10
Acute	13.43	29.58[a]	65.00[b]	0.46[a]	3.25	1.19	0.98
SE	±0.59	±1.15	±1.34	±0.03	±0.21	±0.08	±0.06
Pv	0.95	0.03	0. 01	0.05	0.20	0.22	0.25
Breed							
GM	14.00	27.50	66.75	0.42	3.45	1.27	1.04
Sa	12.32	27.13	67.13	0.41	3.45	1.27	1.04
Fa	13.90	28.13	66.13	0.43	3.45	1.27	1.04
SE	±0.73	±1.41	±1.64	±0.03	±0.26	±0.10	±0.08
Pv	0.26	0.88	0.91	0.86	0.75	0.85	0.81
Treatments							
Control * GM	14.15	26.75	67.00	0.41	3.75	1.38	1.13
Control * Sa	11.69	24.00	70.00	0.34	3.60	1.32	1.08
Control * Fa	14.30	26.00	68.00	0.39	3.60	1.32	1.08
Acute * GM	13.85	28.25	66.50	0.43	3.15	1.16	0.95
Acute * Sa	12.95	30.25	64.25	0.47	3.30	1.21	0.99
Acute* Fa	13.50	30.25	64.25	0.48	3.30	1.21	0.99
SE	±1.03	±2.00	±2.32	±0.05	±0.37	±0.14	±0.11
Pv	0.60	0.50	0.53	0.45	0.71	0.75	0.77

a,b, c… means with different letter in each column for every trait are differ significantly ($P \leq 0.05$).

R. R = Respiration rate B. T. = Body temperature
Hb = hemoglobin concentration Ht% = hematocrit percent
R.B.C. = red blood cells count MCV = Mean corpuscular volume
MCH = mean corpuscular hemoglobin
MCHC = mean corpuscular hemoglobin concentration
W.B.C. = White blood cells count H % = heterophils%
L % = Lymphocytes% H/L = heterophils/ Lymphocytes ratio
Mn% = Monocytes% Es% = Eosinophils%
Bs% = .=Basophils%

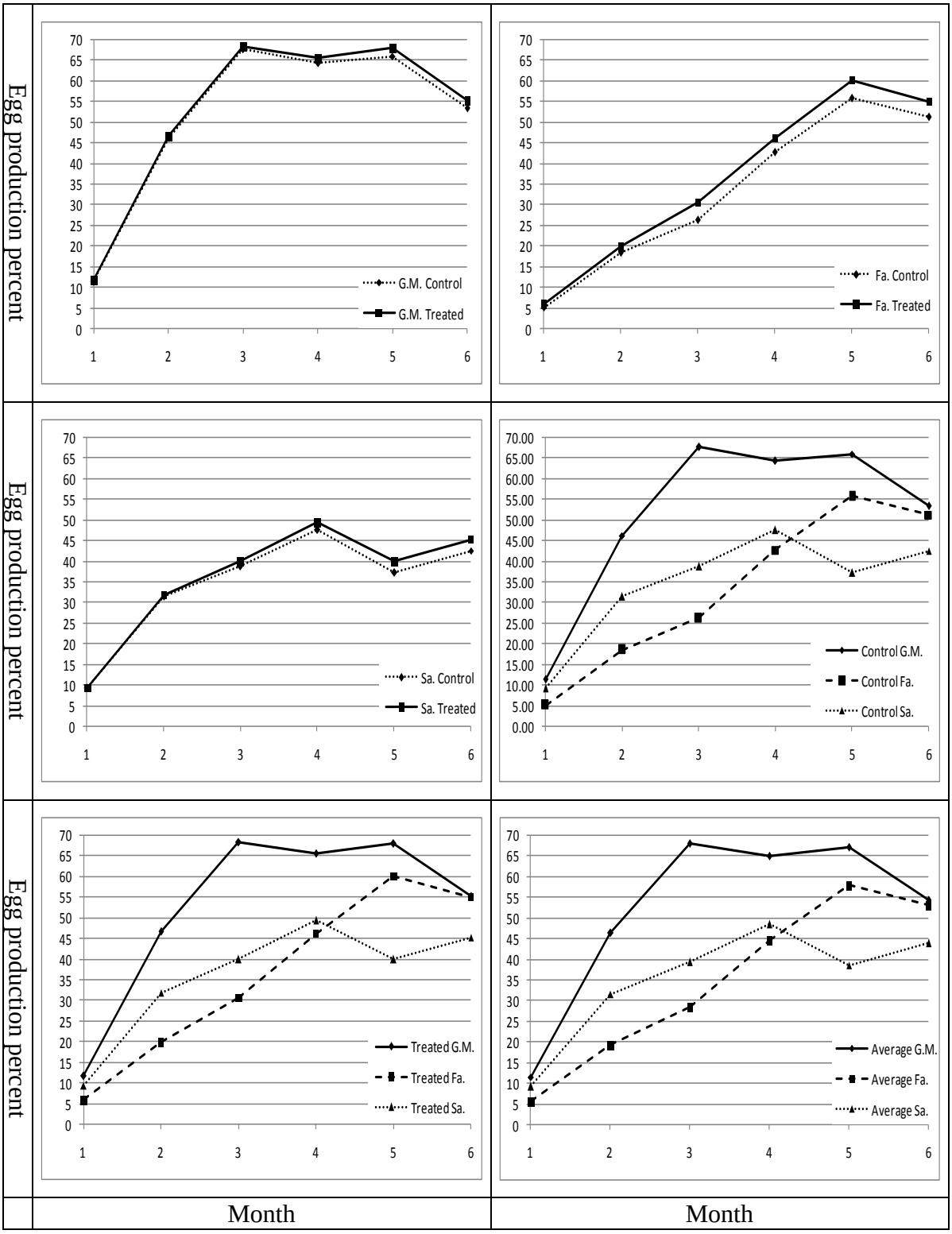
Table (6): Effect of acute heat stress on plasma proteins, enzymes and hormones of three local chickens.

	Plasma proteins					Plasma enzymes		Plasma Hormones			
Parameters	**T.P. g/dl**	**Alb. g/dl**	**Glb. g/dl**	**Alb/ Glb. Ratio**	**ALT U/L**	**AST U/L**	**GSHPx pu/GM**	**T_3 ng/ml**	**T_4 ng/ml**	**T_3/T_4 ratio**	**Cs. ng/mL**
Heat stress											
Control	4.96[a]	2.25	2.71	0.86	23.08	258.03	8.77[b]	2.25[a]	10.43[a]	0.22	0.78[b]
Acute	4.55[b]	2.13	2.42	0.92	34.08	257.21	13.08[a]	1.92[b]	9.30[b]	0.21	1.26[a]
SE	± .10	±	±	±	± 4.86	± 4.17	± .14	± .09	± .27	±	± 0.02
Pv	.01	.35	.09	.59	.13	.89	.00	.02	.01	.40	.00
Breed											
GM	4.51[b]	1.99	2.53	0.83	11.00[b]	251.21	10.98	2.03	10.18	0.20[b]	0.94
Sa	5.05[a]	2.24	2.81	0.80	31.50[a]	260.14	10.73	2.00	9.90	0.20[b]	1.07
Fa	4.70[b]	2.35	2.35	1.00	43.25[a]	261.50	11.06	2.23	9.51	0.23[a]	1.05
SE	± .13	±	±	±	± 5.95	± 5.11	± .17	± .11	± .33	± .01	± 0.02
Pv	.02	.07	.09	.13	.01	.33	.36	.33	.38	.04	.45
Treatments											
Control * GM	4.83	2.08	2.75	0.79	7.75[e]	253.98	8.68	2.13	10.75	0.20	0.73
Control * Sa	5.23	2.30	2.93	0.79	22.50[c]	259.93	8.50	2.20	10.25	0.22	0.80
Control * Fa	4.83	2.38	2.45	1.00	39.00[b]	260.18	9.13	2.43	10.28	0.24	0.82
Acute * GM	4.20	1.90	2.30	0.86	14.25[d]	248.45	13.28	1.93	9.60	0.20	1.15
Acute * Sa	4.88	2.18	2.70	0.82	40.50[ab]	260.35	12.95	1.80	9.55	0.19	1.33
Acute* Fa	4.58	2.33	2.25	1.07	47.50[a]	262.82	13.00	2.03	8.75	0.23	1.29
SE	± .18	±	±	± .12	± 8.41	± 7.22	± .24	± .16	± .47	± .01	± 0.03
Pv	.56	.91	.79	.98	.007	.84	.29	.77	.68	.51	.75

a,b, c… means with different letter in each column for every trait are differ significantly ($P \leq 0.05$).

T.P. = Protein Alb. = albumin AST = Aspartate transaminase (AST)
ALT = Alanine transaminase (ALT) Glb. = Globulin GSHPx = Glutathione peroxidase
T_3 = Triiodothyronine T_4 = Thyroxine Cs. = Corticosterone

Figure (1): Monthly egg production (breeds and treats differences).



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الملخص العربي

دراسة مقارنة بين سلالات المنتزه الذهبي والسلام والفيومي

١- الاستجابة للاجهاد الحراري الحاد كوسيلة للتكيف الحرراري المبكر.

صبّاح فاروق يوسف- دعاء محمد ياسين- نادية معوض الباهي- احمد عبد التواب فضل
معهد بحوث الإنتاج الحيواني- مركز البحوث الزراعية- وزارة الزراعة- الدقي- مصر

تهدف هذه الدراسة لمقارنة استجابة ثلاث سلالات من الدجاج المحلي للاجهاد الحراري الحاد كوسيلة للتكيف الحراري المبكر على أداء النمو وانتاج البيض والتفريخ. استخدم في هذه الدراسة ١٢٠٠ كتكوت انثى عمر يوم من سلالات المنتزه الذهبي و الفيومي والسلام، (٤٠٠ كتكوت انثى من كل سلالة). قسمت الكتاكيت الى مجموعتين (٢٠٠كتكوت لكل مجموعة) قسمت الى٤ مكررات يحتوي كل مكرر على ٥٠ كتكوت. تم استخدام المجموعة الاولى كمجموعة مقارنة دون اي معاملة بينما عرضت المجموعة الثانية للاجهاد الحراري الحاد (٤٠°م لمدة ٨ ساعات) لمرة واحدة على عمر ٥ ايام وذلك كوسيلة لاحداث تكييف حراري مبكر. تم قياس درجة حرارة الجسم ومعدل التنفس (٨ كتاكيت) من كل سلالة بعد اجراء الاجهاد الحراري الحادوكذلك تم قياس درجة حرارة الجسم ومعدل التنفس(٨ كتاكيت) من كل سلالة من مجموعات المقارنة في نفس الوقت. تم سحب ٨ عينات دم بعد اجراء الاجهاد الحراري الحاد وكذلك تم سحب ٨ عينات من مجموعات المقارنة في نفس الوقت. تم وزن كتاكيت المعاملة والمقارنة على عمر شهرين واربعة اشهر والنضج الجنسي. تم عد البيض ووزنه يومياً وتم حساب متوسط وزن البيضة خلال الستة شهور الاولى من الانتاج. تم اجراء ٣ تفريخات حيث تم جمع ٤٠٠ بيضة/تفريخه من كل سلالة (٢٠٠/تفريخة من كل معاملة) وذلك على عمر ٥٠ و٥١ و ٥٢ اسبوع. في نهاية التجربة تم تقدير صفات جودة البيض٢٠ (بيضة/معاملة/سلالة). هذا وققد تم الحصول على النتائج التالية:-

١- زاد وزن الجسم على عمر ٤ شهورمعنويا بالمعاملة الحرارية وكذلك عند عمر النضج الجنسي. وكان تاثير المعاملة الحرارية ايجابي للمنتزه الذهبي والفيومي وسلبي للسلام.
٢- معاملة دجاج المنتزه الذهبي والفيومي حرارياً نقصت عمر النضج الجنسي مقارنة بالسلام المعامل.
٣- ادت المعاملة الحرارية لزيادة معنويه في نسبة انتاج البيض وكان مقدار الزياده اعلى في اللفيومي والسلام عن المنتزه الذهبي. ولم تتأثر قياسات التفريخ بالمعاملة الحرارية.
٤- لم تؤثر المعاملة الحرارية على صفات جودة البيض. وكانت صفات جودة اللبيومين البيض اعلى دجاج الذهبي والسلام عن الفيومي.
٥- زودت المعاملة الحرارية نسبة الدجاج الحي مقارنة بالدجاج الغير معامل. وكان دجاج المنتزه الذهبي الاعلى معنوياً في نسبة الدجاج الحي بنما كان دجاج السلام الاقل معنوياً.
٦- زاد معدل التنفس ودرجة الحرارة بعد المعاملة الحرارية مباشرة عن مجموعات المقارنة.
٧- انخفضت النسبة المئوية لخلايا الليمفوسيت معنوياً في حين زادت نسبة الهيتروفيلس والنسبة بين الليمفوسيت و الهيتروفيلس معنوياً بعد المعاملة الحرارة مباشرة عن تلك النسب بمجموعات المقارنة.
٨- زاد تركيز كل من بروتينات البلازما الكلية، وانزيمات (ALT,AST, GSHPx) مباشرة بعد اجراء الاجهاد الحراري مقارنة بمجموعات المقارنة.
٩- نقص تركيز هرموني الغده الدرقية معنوياً في حين زاد تركيز هرمون الكورتيكوستيرون معنوياً مباشرة بعد اجراء الاجهاد الحراري مقارنة بمجموعات المقارنة.

هذا ويمكن التوصية باجراء المعاملة الحرارية الحادة على عمر ٥ ايام لتقليل الاثار الناجمة عن الإجهاد الحراري خلال فصل الصيف ولتحسين الاداء الانتاجي للسلالات المحلية.