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INFLUENCE OF EARLY THERMAL CONDITION ON INTESTINE DEVELOPMENT, PLASMA T3 AND THE IMMUNE RESPONSE IN TWO LOCAL STRAINS CHICKENS

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ABSTRACT

This experiment was conducted to study the effect of heat stress at early age on small intestine development, plasma diiodothyronine T_3 and immune response of Matrooh and Inshas chicks. Seven hundred and twenty, one day old chicks (360 Inshas chicks and 360 Matrooh chicks). The experiment included three treatments; birds from each strain were divided into three groups. The first no treated group (control T1), the birds in second group were exposed to (42°c - 43°c ± 1°c) for 4 hrs at three days of age (T2) while birds of the third group were exposed to the same thermal treatment of the second group but at four weeks of age (T3). After these treatments birds of (T1, T2 and T3) were raised under regular conditions. At 8 weeks of age 90 chicks from each strain (30 bird from each group) were subjected to heat stress (42°c - 43°c ± 1°c) for 4 hrs. blood samples were taken from (5 chicks) before and after this heat stress. At the end of experiment (18 weeks of age) 5 birds from each treatment groups were slaughtered and the jejunum was removed for histological procedure. The important results obtained are : 1) Early heat stress acclimated bird showed significant effect on small intestine morphology, there was significant difference in villus (height, width, volume) than control. 2) Early heat acclimated birds (at 3 days) had the lowest plasma T_3 content compare to control and (T3). 3) Early heat acclimated birds (3 d or 4 weeks of age) had significantly higher antibody titer than control. Conclusively, the obtained results showed that morphological change observed in the small intestine illustrated one possible mechanism for the loss of bird production induced by heat stress. It appears, therefore, that heat exposure in early age can improve thermo tolerance at maturity improving the ability to reduce T_3 concentration and consequence reduces heat production.

Keywords*:* Early heat stress, Intestine, T_3, Immune response.

INTRODUCTION

In chickens, the small intestine of the newly hatched chick undergoes maturation and dramatic morphological, biochemical, and molecular changes during the first 10 days post hatched (Uni *et al.,* 1996 & 1998). The proliferation of intestinal epithelial cells in chickens is not restricted to the crypt but also occurs along the villus during the first week post hatch. The epithelium of the small intestine is composed of a continuously renewable population of cells. The stem cells, located in crypt region, produce enterocytes that migrate up the villi and are extruded at the villus tip into the intestinal lumen (Potten and loeffler, 1987). It is well documented that thermal conditioning at an early age modulates responses of chickens to heat stress during their life span (Arjona et al., 1988 &1990). Early-age thermal conditioning thus resulted in a significant and sustained reduction in plasma T_3 levels (Yahauv and Plawnik, 1999). Plasma concentration of T_3 is also inversely related to environmental temperature (Hillman et al., 1985). The

association between ambient temperature and plasma T_3 levels may be associated with changes in the structure and function of the intestinal tract as suggested by Mitchell and Carlisle (1992). Several studies have been conducted on the effect of high temperature on the immune responses of chickens, with variable results. Thaxton et al. (1968) demonstrated that high environmental temperatures (44.4°c to 47.8°c) affect the development of specific immune responses in young chickens. Heat stress was also reported to cause a reduction in antibody production in young chickens (Zulkifli et al., 2000). On the other hand, Donker et al. (1990) found that heat exposure did not reduce antibody production to SRBC. Heller et al. (1979) found significantly increased in antibody titers to SRBC following heat exposure. The difference in these findings could be associated with age and type of bird used or due to the experimental methodology that applied. Regnier et al. (1980) suggested that heat-induced immuno suppression may depend on breed of bird. Kelley (1983) reported that effects on immune responses may depend on the length and intensity of the heat exposure. Few studies are available that address the effect of heat stress on small intestine growth and function by measuring villus size and immune parameters. Therefore, the present study was conducted to determine the effect of heat stress on small intestine growth and function and, also the association between the effects of heat stress and the immune response of chickens.

MATERIALS AND METHODS

The fieldwork of the present study was carried out at Inshas poultry research farm Animal Production Research Insitute, Dokki, Giza, Egypt.

A total of seven hundred and twenty (720) one day old chicks (360 chicks Inshas and 360 chicks Matrooh) were individually weighed to the nearst gram, wig banded and raised under management practice. The brooding temperature was maintained between 34◦c and 37◦c at the beginning of the growing period and gradually decreased every 2 to 3 days to reach 24◦c ± 1 untill the end of the growing period. The birds were raised on the wood shaving litter in floor brooding pens. Chicks received ration containing 20% protein and 2900 k cal ME/1 kg diet. Water and ration were provided ad-libitum.

The experiment included three treatments; birds from each strain were divided into three groups. The first no treated group (control T1), the birds in second group were exposed to (42°c - 43°c ± 1°c) for 4 hrs at three days of age (T2) while birds of the third group were exposed to the same thermal treatment of the second group but at four weeks of age (T3). After these treatments birds of (T1, T2 and T3) were raised under regular conditions. At 8 weeks of age, a total of 90 chickens from each strain (30 birds from each group) were subjected to heat stress (42°c - 43°c ± 1°c) for 4 hrs. Blood samples were taken from the brachial vein (5 chickens from each group), in heparinized tube just and immediately after heat exposure and centrifuged at 3000 rpm for 10 min. The plasma was stored at -20°c for analysis. At the end of the experiment (18 weeks of age) five birds were randomly taken from each treatment group and slaughtered. From each bird, the jejunum (from the

end of the duodenum to Michel's diverticulum) was removed segments (1 cm. long) were taken from the middle of the section, fixed in 5% formal saline solution for histology and staining.Radio immunoassay (RIA) of T_3 was carried out on plasma samples using a diagnostic Kit with anintra assay variation of 5.0 to 5.9% as described by Bar and Hurwitz (1979). Jejunal section was fixed in 5% formal saline solution for 24 hrs. Samples were then washed, dehydrated in grades of ethyl alcohol and cleared in xylene. Embedding was carried out in paraffin wax.

All histological studies were performed on 5 µm section using standard procedures with uniform conditions of fixation and staining with hematoxylin and eosin. The morph metric variables included villus height (from the tip of villus to the crypt junction), Villus width (the width at half height and width of muscular layer. Villus volume was calculated as a cylinder from villus height and width at half height. The spaces between villi were determined. These data were then pooled to calculated the mean value of each variable and expressed as mean ± SE. Villus height and crypt depth were measured according to Wu *et al.* (1996).

Immunological parameters:

Antibody response against SRBC was measured from 5 birds in each treatment at 56 and 84 days of age. Birds were injected with 0.2 ml of 9% SRBC in 9% saline. Serum samples were collected at 7 days after each injection to determine anti-SRBC primary or secondary antibody titer, respectively. Antibody production was measured by agglutination test using the micro liter technique (Trout et al., 1996). At 1 and 3 weeks of age hemagglutination –inhibition test was applied for determination of antibodies response in plasma samples according to OIE Manual (2005). After 2 weeks of immunization of the flock by vaccine against New Castle Virus (NDV) and At 2, 9 weeks of age hemagglutination - inhibition test was applied for determination of antibodies response in plasma samples according to OIE Manual (2005). After 2 weeks of immunization of the flock by lasota vaccine against Influenza Disease Virus (AIDV). Commercial ELISA kits were used for selection of antibodies against nucleoprotein and matrix against of NDV and AIDV (Biockok. B.V., Gouda and Holland).

Statistical analysis:

Data of Matrooh and Inshas chickens were analyzed separately using package of SAS sotware version 9.1.

Statistical Model:

Model 1 was used to analyze data of intestinal morphology (villus length, width, volume and distance between villi) and immunological parameters as follow:

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

Where : Y_{ij}= the observation of the ij^{th} chick; µ=overall mean; T_i = the fixed of i^{th} treatments (1…3); e_{ij} = random error.

Model 2 was used to analyze data of Triiodothyronine (T_3) as follow :

$$Y_{ijk} = \mu + T_i + E_j + (TE)_{ij} + e_{ijk}$$

Where: T_i = the fixed of i^{th} treatments (1…3); E_j = the fixed effect of j^{th} time of exposure to heat stress (before, 1; after, 2).

RESULTS AND DISCUSSION

1-Effect of early heat stress on intestinal morphology:

The results of the morph metric measurements are presented in Table 1, These results showed that villus length in jejunum was significantly higher (p≤ 0.001) with (T2) compared to (T1) or (T3) in Matrooh chickens while in Inshas chickens the villus length was significantly (p≤ 0.001) higher with (T3) than the other two treatments. In Matrooh chickens villus width was significantly (p≤ 0.001) higher with (T2) than with (T1) and (T3). While no significant differences were observed in the villus wide for Inshas chickens.

Table 1. Effect of early heat stress on the intestinal morphology of Matrooh and Inshas chickens at 18 weeks of age

Item	Matrooh	Inshas
Villus length (m)		
T1 (control)	0.638±0.054^{b}	0.583±0.040^{a}
T2	0.828±0.036^{a}	0.456±0.039^{b}
T3	0.393±0.042^{c}	0.586±0.036^{a}
Significance	***	***
Villus width:		
T1 (control)	0.097±0.009^{a}	0.073±0.067
T2	0.105±0.008^{a}	0.074±0.067
T3	0.016±0.008^{b}	0.078±0.067
Significance	***	N.S
Villus volume:		
T1 (control)	0.004±0.0004^{b}	0.003±0.0002^{a}
T2	0.007±0.0002^{a}	0.002±0.0002^{b}
T3	0.003±0.0003^{c}	0.003±0.0002^{a}
Significance	***	***
Villus distance		
T1 (control)	0.035 ±0.005	0.049±0.006
T2)	0.034±0.005	0.053±0.006
T3	0.024±0.006	0.0052±0.005
Significance	N.S	N.S

a, b, c means within the same column with different superscript are significantly different; N.S =non-significant; *= significant (p≤ 0.001).**

Villus volume in Matrooh chickens was significantly higher with (T2) than (T1) and (T3). Villus volume in Inshas chickens were actual for (T1) and (T3) and significantly higher than (T2).The results showed no significant differences observed in the villus distance due to early heat stress in both the two strains. The small intestine of the newly hatched chicks undergoes maturation and dramatic morphological biochemical and molecular changes during the first 10 days post- hatch (Uni *et al.,* 1996 & 1998). It is well documented that thermal conditioning at an early age modulate responses of chickens to heat stress during their life span (Arjona, *et al.,* 1988 &1990, Yahauv and Hurwitz, 1996, Yahauv *et al.,* 1997). The chicks immediate response to thermal conditioning consists of a 25% reduction in prolifenting cell in the crypts and decrease in villus volume. These reduction were completely reversed during the recovery stage (48 hrs post – thermal conditioning). At 72 hrs postthermal treatment, the increased number of cells

in the villus underwent hyper trophy which led to elongation of the villus and resulted in differences in the villus volume between heat stress treatments and control group. Bao *et al.,* 2004; Sun *et al.,* 2007). The intestine is susceptible to heat stress, hypoxin and other environmental factors which result in mucosal change. Ning *et al.* (2003) reported that there were severe effects of heat stress on pathological damage of the duodenum, jejunum and ileum, which mainly involved mucosal epithelial cell exfoliation and villi fracture. One of the characteristics of the intestinal villus epithelium is a short proliferation cycle and rapid growth (Sigdostad and Lesher, 1970; Chang and Leblond, 1974). The vegenenative cycle is about 48 hrs, and the intestinal villus epithelium has a good self-repair capacity (Morini et al; 2000;Wang et al., 2002). The intestinal villus has a profuse blood capillary network, which enable the counter current exchange of nutrient substances between the ascending and descending blood vessels in the intestinal villus, maintaining high absorptive function (Yao, 2001). The microcirculation of small intestinal villus is like a hairpin with the tip located at the top of the villus with reduced blood supply. In addition, the capillaries branch at a right angle from the mother branches, easily resulting in the dysfunction of the red blood cells. The jejunum villus is morphologically like slender lobes, which are susceptible to form epithelium lesion following a decrease in blood oxygen supply (Guo and Su 2005). Studies have shown that under the severe heat stress, the systemic blood flow was redistributed (Ooue *et al.,* 2007). To dissipate heat the primary physiological autonomic responses increase blood flow to the body surface. From our findings it can be speculated that environmental heat stress at 3 d post hatching alters T_3 levels which affects the small intestine capacity to proliferate growth. These changes modulate the intestinal tract for compensatory growth 48 hrs. post treatment.

2- Effect of early heat stress on plasma triiodothyronineT_3

The effect of early heat stress on plasma triiodothyronine T_3 of chickens at 8 weeks of age are presented in Table 2. Plasma (T_3) concentration of 8 weeks old chickens subjected early in life (at 3 day and 4 weeks) to heat stress was significantly lower than birds in control group.

After heat stress at (42°c - 43°c ± 1°c) for 4 hours, plasma T_3 concentration significantly ($p \leq 0.001$) decreased in all treatments (Table 2). These findings proved the inhibitory effect of heat stress on thyroid activity. This is supported by the findings of Brwen *et al.,* 1984) on the direct relationship between thyroid function and heat tolerance in chickens. The heat adaptation process allowing the adjustment of the metabolic rates in favour of the body heat balance. The overall heat stress depression in T_3 is in agreement with other authors, who reported a decreased thyroid activity in chickens expressed either to acute or chronic type of heat stresses (Brigman *et al*. 1991; Yahauv and Plawnik, 1999; Tuo et al., 2006). Mitchell and Carlisle (1992) and Geraet et al. (1996) found a dramatic decline of plasma T_3 in broiler chickens reared at 32°c to 35°c environmental temperature, respectively. It has been reported that thyroid hormone administration stimulation heat production with increased metabolic rate resulting in reduced thermo-tolerance (Bowen and Washar, 1985). Also, early investigations, gave an evidence that thyroid gland of birds decreased in size and activity when birds become acclimated

to high environmental temperatures (Huston and Carm, 1962; Shafic et al , 1979 and Sinurat et al., 1987). Arjona et al. (1990) showed that exposure of chickens to high temperature at 42 d of age associated with reduction in plasma T_3 concentration regardless of previous high temperature exposure. In contrast, the present study showed that an early age heat exposure resulted in a significant reduction in plasma T_3 levels. Moreover, the conditioned birds exhibited lower T_3 levels than the control during the high temperature Challenge. It appears, therefore, that heat exposure in early age can improve thermo tolerance at maturity improving the ability to reduce T_3 concentration and, consequences reduce heat production.

Table 2. Effect of heat stress ((42c - 43◦c ± 1◦c) for 4 hrs on plasma T3 of Matrooh and Inshas chickens exposed to early heat stress

		Matrooh	Inshas
Treatments (T):			
T1 (control)		181.646±7.718[a]	160.361±7.670[a]
T2 three days of age		140.546±7.718[b]	128.773±7.670[b]
T3 four weeks of age		162.564±7.718[ab]	136.513±7.670[b]
Significance		***	***
Exposure:			
Before (1)		174.792±6.302[a]	152.797±6.263[a]
After (2)		148.379±6.302[b]	130.967±6.263[b]
Significance		***	***
T * Exposure			
1	1	184.296±10.915	181.297±10.848
1	2	178.997±10.915	139.429±10.848
2	1	161.082±10.915	133.255±10.848
2	2	120.010±10.915	124.291±10.848
3	1	178.997±10.915	143.840±10.848
3	2	146.311±10.915	129.185±10.848
Significant		N.S	N.S

a, b means within the same column with different superscript are significantly different; N.S =non-significant; *= significant (p≤ 0.001); Before = before heat stress; After = after heat stress.**

2-Effect of early heat stress on immunological parameters:

Immune response against Sheep red blood cells (SRBC), New Castle Disease virus (NDV) and Avian Influenza Disease Virus (AIDV) are presented in tables 3,4and 5. Data presented in table (3) indicated that heat stress caused increased in primary and secondry immune response against SRBC than control in Matrooh and Inshas chickens, but the differences were not significant.

Table 3. Effect of heat stress ((42c - 43◦c ± 1◦c) for 4 hrs on antibody titers against (SRBC) of Matrooh and Inshas chickens exposed to early heat stress

Item	Matrooh		Inshas	
	Primary antibody titers	Secondary antibody titers	Primary antibody titers	Secondary antibody titers
T1 (control)	4.200±0.902	4.00±1.254	6.20±0.766	5.80±1.322
T2	6.600±0.902	7.60±1.254	7.40±0.766	7.60±1.322
T3	5.800±0.902	6.60±1.254	6.40±0.766	7.20±1.322
Significance	N.S	N.S	N.S	N.S

N.S = not significant.

The results presented in table 4 showed that heat stress increased significantly p≤ 0.01) primary and secondary immune response against (NDV) than control in Inshas chickens, while in Matrooh chickens significant p≤ 0.01) increase only in secondary immune response against NDV.

Table 4 Effect of heat stress ((42◦c - 43◦c ± 1◦c) for 4 hrs on antibody titers against (NDV) of Matrooh and Inshas chickens exposed to early heat stress

Item	Matrooh		Inshas	
	Primary antibody titers	Secondary antibody titers	Primary antibody titers	Secondary antibody titers
T1 (control)	0.600±0.294	0.800±0.632^{b}	0.400±0.183^{ab}	0.400±0.548^{b}
T2	0.800±0.294	4.000±0.632^{a}	0.500±0.183^{b}	7.60±0.548^{a}
T3	0.600±0.294	3.600±0.632^{a}	0.800±0.183^{a}	7.20±0.548^{a}
Significance	N.S	***	**	***

a, b means within the same column with different superscript are significantly different; N.S =non significant; *= significant (p≤ 0.001); **= significant (p≤ 0.01).**

Data in table 5 showed that there were no significant difference in immune response against (AIDV) in Matrooh chickens under heat stress but, heat stress caused significant p≤ 0.0 5) increased in primary and secondary immune response against (AIDV) than control in Inshas chickens.

Table 5 Effect of heat stress ((42 - 43◦c ± 1◦c) for 4 hrs on antibody titers against (AIDV) of Matrooh and Inshas chickens exposed to early heat stress

Item	Matrooh		Inshas	
	Primary antibody titers	Secondary antibody titers	Primary antibody titers	Secondary antibody titers
T1 (control)	1.200±0.497	8.200±0.497	0.80±0.606^{b}	6.80±0.643^{b}
T2	2.000±0.497	9.800±0.497	3.40±0.606^{a}	9.60±0.643^{a}
T3	2.000±0.497	9.600±0.497	3.00±0.606^{a}	9.20±0.643^{a}
Significance	N.S	N.S	*	***

a, b means within the same column with different superscript are significantly different; N.S =non significant; *= significant (p≤ 0.05).

Similar results, Al-Bisher (1998) studied the effect of short term heat stress on antibody production of Baladi and leghorn chickens, they found that, heat stress stimulated antibody production during primary immunization and suppressed it during secondary immunization. On the other hand, Guo and Su 2005 found that heat stress significantly inhibited the normal development of lymphoid organs and impaired the immunological competence. Mohamed (2006) and Megahid (2007), they found that there were no significant of heat stress on antibody production against (SBRC). Also, Donker *et al.* (1990) found that heat exposure did not reduce antibody production to (SRBC). Heller et al. (1979) found significantly increased antibody titers to (SRBC) following heat stress exposure. The difference in these findings could be associated with age and type of birds used or due to the experimental methodology that applied. Regnier *et al* (1980) suggested that heat induced immunosuppression may depend on breed of bird. While, Kelley (1983) reported that effects on immune responses may depend on the length and intensity of heat exposure. Heat stress was also, reported to

cause a reduction in antibody production in young chickens, this reduction could be indirectly due to increase in inflammatory cytokines under stress (Zulkifli et al., 2000), (Oglo et al., 1990) which stimulates the lypothalamic production of corticotrophin releasing factor (Sapolsky et al., 1987). Corticotrophin releasing factor is known to increase adrenocorticotropic hormone (ACTH) from the pituitary; (ACTH) then stimulates corticosteron production from adrenal gland-Corticosteron ihibits antibody production (Gross, 1992). Furthermore, heat stress is known to decrease T-helper 2 cyteokines (Wang et al.,2001) which are important for antibody production (Lebman and Coffman, 1988).

CONCLUSION

The obtained results showed that morphological change observed in the small intestine illustrated one possible mechanism for the loss of bird production induced by heat stress. It appears, therefore, that heat exposure in early age can improve thermo tolerance at maturity improving the ability to reduce T_3 concentration and, consequence reduce heat production.

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تأثير التعرض الحراري المبكر على تطور الأمعاء الدقيقة وثيروكسين البلازما والاستجابة المناعية في سلالتين من الدجاج المحلى.

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أ

جريت هذه التجربة لدراسة تأثير التعرض الحراري المبكر على تطور الأمعاء الدقيقة وT3 في البلازما وكذا الاستجابة المناعية لكتاكيت سلالتي مطروح و انشاص. تم استخدام 720 كتكوت عمر يوم (360 كتكوت من سلالة مطروح و 360 كتكوت من سلالة انشاص). اشتملت التجربة على ثلاثة معاملات كل معاملة 120 كتكوت الأولى معاملة المقارنة (كنترول) وعرضت المعاملة الثانية لإجهاد حراري مبكر على درجة حرارة 42-43±1درجة مئوية لمدة 4 ساعات وعرضت المعاملة الثالثة لنفس المعاملة الحرارية السابقة ولكن عند عمر 4 اسابيع . بعد المعاملات الحرارية اعيدت الكتاكيت الى برنامج حرارى عادى . عند عمر 8 اسابيع تم تعريض 90 طائر من كل سلالة الى درجة حرارة 42-43±1درجة مئوية . تم اخذ عينات الدم قبل وبعد هذه المعاملة مباشرة وتم طردها مركزيا وحفظت للتحليل .وفى نهاية التجربة عند عمر 18 اسبوع تم ذبح 5 طيور من كل معاملة و أجريت الدراسة الهستولوجية للامعاء الدقيقة.

و كانت أهم النتائج المتحصل عليها :

1 - أدى التعرض الحرارى المبكر للكتاكيت الى زيادة معنوية فى الشكل الظاهري للأمعاء الدقيقة (طول وعرض وحجم الخملة) بالمقارنة بالمعاملة الكنترول.

2 - أدى التعرض الحرارى المبكر فى عمر 3 أيام الى انخفاض محتوى T3 من البلازما بالمقارنة بمجموعة الكنترول والمجموعة 3.

3 - أدى التعرض الحرارى المبكر فى عمر 3 أيام و4 اسابيع من العمر الى زيادة معنوية فى الأجسام المضادة مقارنة بمجموعة الكنترول.

ونخلص من ذلك ان التغيرات الحادثة فى تطور الامعاء هى المسبب لنقص الانتاج تحت تأثير الاجهاد الحرارى وان انخفاض محتوى البلازما من T3 للطيور المعرضة للمعاملة الحرارية المبكرة يؤدى الى خفض انتاج الحرارة.