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# THE RELATIONSHIP BETWEEN IMMUNE RESPONSE AND HEAT SHOCK PROTEIN 70 (Hsp 70) UNDER COLD AND HEAT STRESSES IN NORFA CHICKENS

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**ABSTRACT:** *This study was carried out during the period from 2014 to 2015 in the Poultry Research Farm, Department of Poultry Production, Faculty of Agriculture, Menufia University, Shibin El- Kom, Egypt. The aim of this research was to study the relation between immune response and Hsp70 under different (cold (10 ˚C / 76% humidity) and heat (37 ˚C / 57% humidity)) stresses in order to predict the ability of chicks to tolerate these stresses in older ages. Total number of 322 males and females of Norfa chicken were used in 12 wks of age. Birds were divided into three groups according its immunity level (high, low and Control), the previous groups were subdivided into 6 subgroups (3 under heat stress and 3 under cold stress)by using a climatic chamber. These subgroups were subjected to different periods of exposure to stress (acute and chronic).*

*Results indicated that, in acute heat stress experiment, birds with high immunity levels which exposed to cold stress for 6 hours had the highest values of Hsp70 (0.056±0.0045) with no significant differences with the other levels except for the birds with high immunity levels which exposed to high temperature stress for 3 hours. There were no significant differences among all exposure periods for the birds of low immunity level.*

*In chronic heat stress experiment, birds with high immunity levels, which exposed to high temperature for 6 days, had the highest values of Hsp70 (0.086±0.0031) with significant differences* (P≤0.05) *compared with the other groups. Regarding the birds of the low immunity levels which exposed to cold temperature stress for 6 days had high value of Hsp70 more than the birds which exposed to 3 days.*

*As a conclusion, immunity level can be used as an indicator for heat tolerance for the birds exposed to cold or heat stress.*

***Key words:*** *Immune response, Hsp 70, cold stress, heat stress, chickens.*

## INTRODUCTION

Stress represents the reaction of the animal organism (i.e., a biological response) to stimuli that disturb its normal physiological equilibrium or homeostasis.

When living organisms are exposed to thermal or non thermal stressors, the synthesis of most proteins is retarded; however, a group of highly conserved proteins known as HSPs are rapidly synthesized (Baqchi *et al.,* 2001; Ogura *et al.,* 2007; Park *et al.,* 2007). These proteins are essential for organisms living at the edge of their thermal range. It is well documented that one of the most important functions of Hsps is to protect organisms from the toxic effects of heating (Arrigo, 2000). HSPs may play important roles in protein assembly and disassembly (Bukau *et al.,* 2000; Hartl and Hayer-Hartl, 2002).

One relevant feature of Hsps is that overexpression of one or more Hsp genes confers protection against subsequent stress (McCormick *et al.,* 2003; Zhang *et al.,* 2007; Luh *et al.,* 2007).

The protein acts as a molecular chaperone by binding to other cellular proteins, assisting intracellular transport, and folding into the proper secondary structures, thus preventing aggregation of protein

during stress (Chirico *et al.,* 1988; Hartl, 1996).

Heat shock proteins (Hsp) have been suggested to play a role in cellular protection under high ambient temperature, with a proposed relationship between the development of thermo tolerance and Hsp synthesis, especially Hsp70 (Hsp70; Lindquist and Craig, 1988).

The hormone-binding ability can be restored after a transient loss using an in vitro 5 protein system containing Hsp40, Hsp70, Hsp organizing protein, Hsp 90, and adenosine triphosphate (Hernandez *et al.,* 2002).

According to the homology and molecular weights, Hsp can be classified into 3 main families: Hsp90 (~85–90 kDa), Hsp70 (~68–73 kDa), and low molecular weight Hsp (~16–47 kDa; (Basu *et al.,* 2002). Among the Hsp, Hsp70 is one of the most conserved and important protein families and has been studied extensively (Deane and Woo, 2005; Ming *et al.,* 2010).

Discrepancies in susceptibility to different pathogens may be attributed to the effects of corticosteroids and heat shock proteins either on the particular pathology involved or on the immunological defense mechanisms (Siegel, 1980, 1985; Wampler *et al.,* 2004; Malago *et al.,* 2005; Nemeth *et al.,* 2006).

These Hsp play an important role in the survival of stressed cells and the stabilization of the internal environment (Gabai *et al.,* 1997).

Most research on Hsp in poultry has emphasized its association with body temperature (Wang and Edens, 1993; Yahav *et al.,* 1997; Givisiez *et al.,* 1999).

## MATERIALS AND METHODS

A total number of 322 males and females of Norfa strain were used in this experiment. At 12 wks. of age, birds were selected divergently for high (HIR) and low (LIR) secondary immune response 7 d after i.m. immunization with 0.1 mL of a 0.25% suspension of SRBC and the treatments were continued up to 19 wks. The birds were divided into three groups as follows:-

**a)** High immune response group: the highest 154 chicks in antibody titers were taken from the base stock to form the high immune response group more than $(\bar{X} + S.E)$.

**b)** Low immune response group: the lowest 154 chicks in antibody titers $(\bar{X} - S.E)$ also, taken to form the low immune response group.

**c)** Control group: 14 both sexes birds were taken at random from the base stock (322 individuals) to serve as control group.

Birds of each group were kept in two climate chamber rooms where temperature, relative humidity and lightning were artificially controlled following the recommendations of the manufacturer. The measurements of each room are 6 m length, 3 m width and 3 m height. The rooms are equipped with an adjustable heating system with temperature ranged from -5:+50 °C. The rooms also have an adjustable humidity system to get relative humidity ranged from 10-90 %.

**Room 1:-** Where cold stress was applied, the birds were transferred to the climatic champers and kept at 10 ˚C / 76% humidity. The birds exposed to acute cold stress for 3, 6, and 12 h and chronic cold stress for 3 and 6 days.

**Room 2:-** Where heat stress was applied, the chicks were transferred to the climatic champers and kept at 37 ˚C / 57% humidity. The birds exposed to acute heat stress for 3, 6, and 12 hours and chronic heat stress for 3 and 6 days.

**Room 3:-** Served as control and exposed to normal air temperature. Conventional diet and fresh clean water were available at all the experimental period.

Blood samples were collected randomly from each group end of the experiment using serum separator tube (SST) until to allow samples to clot for two hours at room temperature or overnight at 4 $^{o}$C before centrifugation for 15 minutes at 1000 x g. Serum removed and assay immediately or aliquot and samples stored at -20 $^{o}$C. Avoiding repeated freeze thaw cycles. Antibody presence in serum was evaluated by ELISA by using a commercial kit.

## Assay procedure

All reagents and samples were brought to room temperature before use. Centrifuged the samples again after thawing before the assay. It was recommended that all samples and standards be assayed in duplicates.

1. All reagents and samples are prepared as directed in the previous sections.
2. The number of wells is determined to be used and put any remaining wells and the desiccant back into the pouch and seal the ziploc, store unused wells at 4°C.
3. A blank well without any solution is setted.
4. 50µl of standard or sample per well are added. Standard need test in duplicate.
5. 50µl of HRP-conjugate are added to each well (not to Blank well), then 50µl antibody are added to each well. They are mixed well and then incubated for 1 hour at 37°C.
6. Aspirated each well and wash, this process is repeated two times for a total of three washes. Washing is by filling each well with wash buffer (200µl) using a squirt bottle, multi-channel pipette, manifold dispenser, or auto washer, and it is let stand for 10 seconds, liquid is removed completely at each step and this is essential to good performance. After the last wash, any remaining wash buffer is removed by aspirating ordecanting. The plate is Inverted and blotted against clean paper towels.
7. 50µl of substrate A and 50µl of substrate B are add to each well, mixed well. It is incubated for 15 minutes at 37°C. The plate is kept away from drafts and other temperature fluctuations in the dark.
8. 50µl stop solution is added to each well, gently tap the plate to ensure thorough mixing.
9. The optical density of each well is determined within 10 minutes, using a microplate reader set to 450 nm.

This ELISA kit uses Sandwich-ELISA as the method. The Microelisa stripplate provided in this kit has been pre-coated with an antibody specific to HSP-70. Standards or samples are added to the appropriate Microelisa stripplate wells and combined to the specific antibody. Then a Horseradish Peroxidase (HRP)- conjugated antibody specific for HSP-70 is added to each Microelisa stripplate well and incubated. Free components are washed away. The TMB substrate solution is added to each well. Only those wells that contain HSP-70 and HRP conjugated HSP-70 antibody will appear blue in color and then turn yellow after the addition of the stop solution. The optical density (OD) is measured spectrophotometrically at a wavelength of 450 nm. The OD value is proportional to the concentration of HSP-70. You can calculate the concentration of HSP-70 in the samples by comparing the OD of the samples to the standard curve by (MyBiosource.com).

## Statistical analysis:

Data were analyzed by the least square analysis of variance using the General Linear Model Procedure (SAS, 2004). Significant differences among treatment means were tested using Duncan multiple range test (Duncan, 1955).

## RESULTS AND DISCUSSION

**In experiment (1):** The Hsp70 protein was an important part of the cell's machinery for protein folding and can act to protect cells from thermal or oxidative stress.

The effect of immunity levels, acute heat stress and its exposure periods ( $\bar{X} \pm SE$ ) on heat shock protein (Hsp70) were shown in (Table 1). Differences between birds of the high levels of immunity (0.051±0.002) and the birds of the low group (0.041±0.002) were significant (P≤0.05). There were no significant differences among different exposure periods. This may be due to stimulation of increasing in Hsp70 protein and its mRNA expression after heat stress. These results were in agreement with that obtained by Franco-Jimenez and Beck (2007) who stated that, heat stress to 35°C for 1 h did not induce changes in Hsp70 liver expression.

The levels of Hsp70 increases as a consequence of increasing the periods of exposure for heat stress. This Hsp70 may be used as a good indicator of thermo tolerance.

**Table (1): Effect of immunity levels, acute thermal stress and its periods of exposure on Hsp70 in Norfa chickens ( $\overline{X} \pm$ S.E).**

| Immunity and thermal stress treatments | Hsp70 |
|---|---|
| **Immunity levels** | |
| **High** | 0.051±0.002 [a] |
| **Low** | 0.041±0.002 [b] |
| **Control** | 0.043±0.005 [ab] |
| **Thermal stress** | |
| **Cold** | 0.047±0.002 [a] |
| **Heat** | 0.045± 0.002 [a] |
| **Control** | 0.047±0.004 [a] |
| **Periods of exposure** | |
| **3 hours** | 0.041±0.003 [a] |
| **6 hours** | 0.048±0.003 [a] |
| **12 hours** | 0.049±0.003 [a] |
| **Control** | 0.048±0.004 [a] |

a, b, c Means within the same column and the same treatment factors carry different small superscripts are significant at levels $P \leq 0.05$
Hsp70 (ng/mg total protein) Heat Shock Protein.

Interaction among immunity levels, acute heat stress and its exposure periods were shown in (Table 2). Significant differences (P≤0.05) between birds of high and low immunity levels which exposed to cold stress were found. This may be due to confer protection from oxidative stress induced by cold stress by improving antioxidant capacity of immune organs. These results were in agreement with that obtained by Tamzil *et al.,* (2013) who found that , exposure to acute heat stress (40°C) for 0.5, 1.0 and 1.5 h increased Hsp70 expression in commercial chickens.

**Table (2): The effect of interaction between immunity levels, acute thermal stress and its periods of exposure on heat shock protein (Hsp70) levels in Norfa chickens ($\overline{X}$ ± S.E).**

| Immunity and thermal stress treatments | | $Hsp_{70}$ |
|---|---|---|
| **Immunity levels × Thermal stress** | | |
| **Control** | | 0.043±0.005 $^{a\ b}$ |
| **High immunity** | **Cold** | 0.054±0.003 $^{a}$ |
| | **Heat** | 0.047±0.003 $^{a\ b}$ |
| **Control high immunity** | | 0.054±0.005 $^{a}$ |
| **Low immunity** | **Cold** | 0.041±0.003 $^{b}$ |
| | **Heat** | 0.042±0.003 $^{b}$ |
| **Contol low immunity** | | 0.041±0.005 $^{b}$ |
| **Immunity levels ×Periods of exposure** | | |
| **Control** | | 0.043±0.005 $^{bcd}$ |
| **High Immunity** | **3 hours** | 0.054±0.005 $^{a\ b}$ |
| | **6 hours** | 0.046±0.003 $^{a\ bcd}$ |
| | **12 hours** | 0.055±0.003 $^{a}$ |
| **Control high immunity** | | 0.051±0.003 $^{a\ bc}$ |
| **Low immunity** | **3 hours** | 0.041±0.005 $^{cd}$ |
| | **6 hours** | 0.036±0.003 $^{d}$ |
| | **12 hours** | 0.041±0.003 $^{cd}$ |
| **Control low immunity** | | 0.047±0.003 $^{abcd}$ |
| **Thermal stress ×Periods of exposure** | | |
| **General Control** | | 0.043±0.005 $^{a\ b}$ |
| **Cold** | **3 hours** | 0.045±0.004 $^{a\ b}$ |
| | **6 hours** | 0.047±0.004 $^{a\ b}$ |
| | **12 hours** | 0.050±0.004 $^{a}$ |
| **Heat** | **3 hours** | 0.037±0.004 $^{b}$ |
| | **6 hours** | 0.049±0.004 $^{a\ b}$ |
| | **12 hours** | 0.049±0.004 $^{a\ b}$ |

a, b, c Means within the same column and the same treatment factors carry different small superscripts are significant at levels $P \leq 0.05$

Hsp70 (ng/mg total protein) Heat Shock Protein.

In contrast, the differences between birds of high and low immunity levels under high temperature acute stress were not significant. Heat shock proteins have been suggested to be involved in cellular protection in adverse situations, and these proteins may improve thermo tolerance of the bird. These results were in agreement with that obtained by Hao *et al.,* (2012) who noticed that, no effects of over expression of Hsp70 in the jejuna mucosa under 36 ± 1°C for 0, 2, 3, 5, and 10 h in broilers.

Birds with high immunity levels which exposed to 3 hours had higher levels of Hsp70 (0.054±0.005) when compared with that of low immunity. The same result was found in case of 12 hours exposure periods. Moreover, the differences among the three exposure periods were not significant regardless of the levels of immunity. Hsp expression may be due to protective protein to regulate the immune function of chicks in cold stress conditions.

Members of the Hsp70 family were very strongly up regulated by heat stress. With regard to the interaction between acute heat stress and its exposure periods, there were significant differences (P≤0.05) in the levels of Hsp70 between the birds that exposed to cold stress for 12 hours (0.050±0.004) and the other group which exposed to high temperature stress (0.037±0.004) for 3 hours. This lower response in the pre exposed HS group might be due to HS acclimation and could be indicative of acquired thermotolerance.

Heat shock proteins were extremely potent molecules, the importance of which to physiological and immunological processes was indicated by the high degree to which their structure and function were phylogenetically conserved.

The interactions among the three factors (immunity levels, acute heat stress and its exposure periods) were shown in (Table 3, Fig 1). Birds with high immunity levels which exposed to cold stress for 6 hours had the highest values of Hsp70 (0.056±0.0045) with no significant differences with the other levels except for the birds with high immunity levels which exposed to high temperature stress for 3 hours. There were no significant differences among all exposure periods for the birds of low immunity level. This may be due to provide the link between innate and adaptive immune systems which may indicate that the time of exposure was not long enough or certain adaptation had already occurred in those pre exposed birds, reducing the amount of HSP70 needed for protection when the bird was exposed to HS. The results obtained by Zhao *et al.,* (2014) who showed that, significantly decreased (P<0.05) in the *m*RNA levels of the Hsp70 gene of spleen and thymus under acute cold stress (12±1) °C and kept for 1, 3, 6, 12, and 24 h, but significantly increased (P<0.05) the *m*RNA levels of the Hsp70 gene of bursa of fabricius in all treatment groups.

**In experiment (2):** The induction of Hsp70 in response to stress serves to protect against the initial insult, augment recovery, and produce a state of resistance to subsequent stress.

The effect of immunity levels, chronic heat stress and its exposure periods $(\bar{X} \pm SE)$ on heat shock protein (Hsp70) were shown in (Table 4). Differences among the high levels of immunity and both of low and control groups were significant (P≤0.05). The birds with high immunity level had the highest value of Hsp70 (0.064±0.003) followed by the low immunity group (0.044±0.003) and the lowest value for the control group (0.043±0.006).

Heat stress induced remarkable changes in heat shock protein levels. There were no significant differences between the cold, heat and control groups for the effect of heat stress, taking into account that the heat stress group had the highest levels of Hsp70 (0.057±0.0043). These results were in agreement with that obtained by Liew *et al.,*

(2003) who found that, the Hsp70 expression was not significant different during the heat exposure periods.

As the periods of exposure to heat stress increases, the levels of Hsp70 increases also. Hsp70 was increased by about 24% when the periods of exposure increased from 3 to 6 days. The control group had the lowest levels of Hsp70. Moreover, the differences among the exposure periods were not significant. The greater Hsp70 expression may be due to the proteins are involved in the stress caused by heat shock exposure in chickens.

The effect of cold and heat stress on immune response may depend on the exposure periods after the stress or at which immune response was determined. Interaction among immunity levels, chronic heat stress and its exposure periods were shown in (Table 5). Birds with high immunity levels which exposed to high temperature stress had the highest value of Hsp70 (0.068±0.005), the same trend as in (Table 4), there were significant differences between heat and cold stress for the birds of low immunity. These results were in agreement with that obtained by Mohamed, Hanan (2006), Zhen *et al.,* (2006) and Badri *et al.,* (2008) who indicated that, quails under heat stress 39°C significantly higher Hsp 70 density in liver tissue.

**Table (3): The interaction effect between immunity levels, acute thermal stress, its periods of exposure on (Hsp70) of Norfa chickens ( $\overline{X}$ ± S.E).**

| Levels | Thermal stress | Periods of exposure | Hsp 70 |
|---|---|---|---|
| **General control** | | | $0.0427\pm0.0045^{abcde}$ |
| **High immunity** | **Cold** | **3 h** | $0.0517\pm0.0045^{abcd}$ |
| | | **6 h** | $0.0560\pm0.0045^{a}$ |
| | | **12 h** | $0.0540\pm0.0045^{ab}$ |
| | **Heat** | **3 h** | $0.0393\pm0.0045^{bcde}$ |
| | | **6 h** | $0.0543\pm0.0045^{ab}$ |
| | | **12 h** | $0.0487\pm0.0045^{abcde}$ |
| **Control high immunity** | | | $0.0537\pm0.0045^{abc}$ |
| **Low immunity** | **Cold** | **3 h** | $0.0387\pm0.0045^{cde}$ |
| | | **6 h** | $0.0383\pm0.0045^{de}$ |
| | | **12 h** | $0.0460\pm0.0045^{abcde}$ |
| | **Heat** | **3 h** | $0.0337\pm0.0045^{cd}$ |
| | | **6 h** | $0.0430\pm0.0045^{abcde}$ |
| | | **12 h** | $0.0487\pm0.0045^{abcde}$ |
| **Control low immunity** | | | $0.0413\pm0.0045^{abcde}$ |

a, b, c, d, e, f Means within the same column and the same treatment factors carry different small superscripts are significant at levels $P \leq 0.05$,

Hsp70 (ng/mg total protein) Heat Shock Protein.

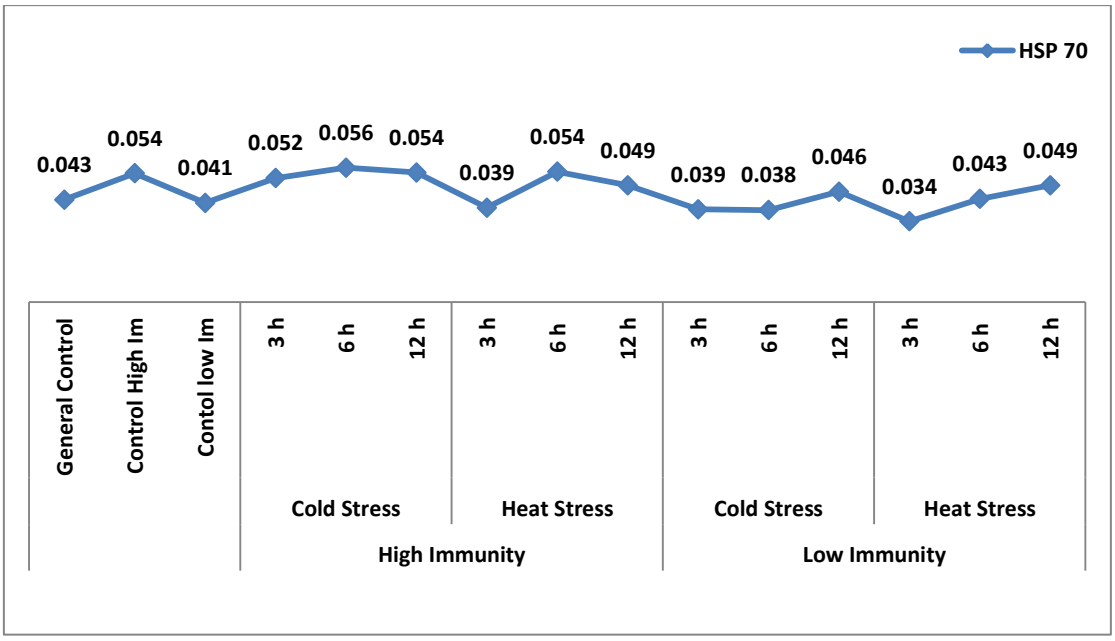


**Fig. (1): The interaction between immunity levels, acute thermal stress and periods of exposure on heat shock protein (Hsp70) of Norfa chickens.**

**Table (4): Effect of immunity levels, chronic Thermal stress and its periods of exposure on Hsp70 in Norfa chickens ($\overline{X}$ ± S.E).**

| Immunity and Thermal stress treatments | Hsp70 |
|---|---|
| **Immunity levels** | |
| **High** | 0.064±0.003 [a] |
| **Low** | 0.044±0.003 [b] |
| **Control** | 0.043±0.006 [b] |
| **Thermal stress** | |
| **Cold** | 0.055±0.0043 [a] |
| **Heat** | 0.057±0.0043 [a] |
| **Control** | 0.048±0.0061 [a] |
| **Periods of exposure** | |
| **3 days** | 0.050±0.004 [ab] |
| **6 days** | 0.062±0.004 [a] |
| **Control** | 0.048±0.006 [ab] |

a, b, c Means within the same column and the same treatments factors carry different small superscripts are significant at levels $P \leq 0.05$

Hsp70 (ng/mg total protein) Heat Shock Protein;

**Table (5): The effect of interaction between immunity levels, chronic thermal stress and its periods of exposure and heat shock protein levels in Norfa chickens ($\overline{X}$ ± S.E).**

| Immunity and heat stress treatments | | Hsp70 |
|---|---|---|
| **Immunity levels × Thermal Stress** | | |
| **Control** | | 0.043±0.007 [b] |
| **High immunity** | **Cold** | 0.066±0.005 [a] |
| | **Heat** | 0.068±0.005 [a] |
| **Control high immunity** | | 0.054±0.007 [ab] |
| **Low immunity** | **Cold** | 0.045±0.005 [b] |
| | **Heat** | 0.046±0.005 [b] |
| **Contol low immunity** | | 0.041±0.007 [b] |
| **Immunity levels× Periods of exposure** | | |
| **Control** | | 0.043±0.006 [c] |
| **High Immunity** | **3 days** | 0.062±0.004 [bc] |
| | **6 days** | 0.073±0.004 [ab] |
| **Control high immunity** | | 0.054±0.006 [a] |
| **Low immunity** | **3 days** | 0.039±0.004 [c] |
| | **6 days** | 0.051±0.004 [c] |
| **Control low immunity** | | 0.041±0.006 [bc] |
| **Thermal Stress × Periods of exposure** | | |
| **General Control** | | 0.043±0.008 [b] |
| **Cold** | **3 days** | 0.055±0.005 [ab] |
| | **6 days** | 0.056±0.005 [ab] |
| **Heat** | **3 days** | 0.046±0.005 [b] |
| | **6 days** | 0.068±0.005 [a] |

a, b, c Means within the same column and the same treatments factors carry different small superscripts are significant at levels $P \leq 0.05$,

Cold: Cold stress (10ºC / RH: 76%); Heat: Heat stress (37ºC / RH: 57%),

Hsp70 (ng/mg total protein) Heat Shock Protein.

Birds with high immunity levels which exposed to stress for 6 days had the highest value of Hsp70 (0.073±0.004), the same trend as in (Table 4), significant differences were observed with the birds of low immunity levels which exposed to stress for 3 days or 6 days.

Regarding the interaction between chronic heat stress and its periods of exposure, birds which exposed to high temperature stress for 6 days had the highest value of Hsp70 (0.068±0.005), significant differences were noted with the birds which exposed to high temperature stress for 3 days.

The interactions among the three factors (immunity levels, heat stress and its exposure periods) were shown in (Table 6, Fig 2). Birds with high immunity levels which exposed to high temperature stress for 6 days had the highest values of Hsp70 (0.086±0.0031) with significant differences compared with the other groups. Regarding the birds of the low immunity levels which exposed to cold temperature stress for 6 days had high value of Hsp70 more than the birds which exposed to the same stress for only 3 days. These results were in agreement with that obtained by Givisiez *et al.,* (2001) and Soleimani *et al.,* (2012) who noticed that, Hsp70 levels increased significantly during heat stress in the birds.

**Conclusion:** This study revealed that there is a strong relation between chicken with high immunity level and Hsp 70 in heat resistance. Birds with high immunity level had a better heat shock protein 70 as compared to birds with low immunity level.

**Table (6): The interaction between immunity levels, chronic thermal stress, its periods of exposure on (Hsp70) of Norfa chickens ( $\overline{X}$ ± S.E).**

| Levels | Thermal stress | Periods of exposure | Hsp 70 |
|---|---|---|---|
| **General control** | | | 0.043±0.0031 $^{def}$ |
| **High immunity** | **Cold** | **3 d** | 0.073±0.0031 $^{b}$ |
| | | **6 d** | 0.059±0.0031 $^{c}$ |
| | **Heat** | **3 d** | 0.050±0.0031 $^{cde}$ |
| | | **6 d** | 0.086±0.0031 $^{a}$ |
| **Control high immunity** | | | 0.054±0.0031 $^{c}$ |
| **Low immunity** | **Cold** | **3 d** | 0.038±0.0031 $^{f}$ |
| | | **6 d** | 0.052±0.0031 $^{cd}$ |
| | **Heat** | **3 d** | 0.041±0.0031 $^{ef}$ |
| | | **6 d** | 0.050±0.0031 $^{cde}$ |
| **Control low immunity** | | | 0.041±0.0031 $^{ef}$ |

a, b, c, d, e, f Means within the same column and the same treatments factors carry different small superscripts are significant at levels P $\leq$ 0.05,

Hsp70 (ng/mg total protein) Heat Shock Protein.

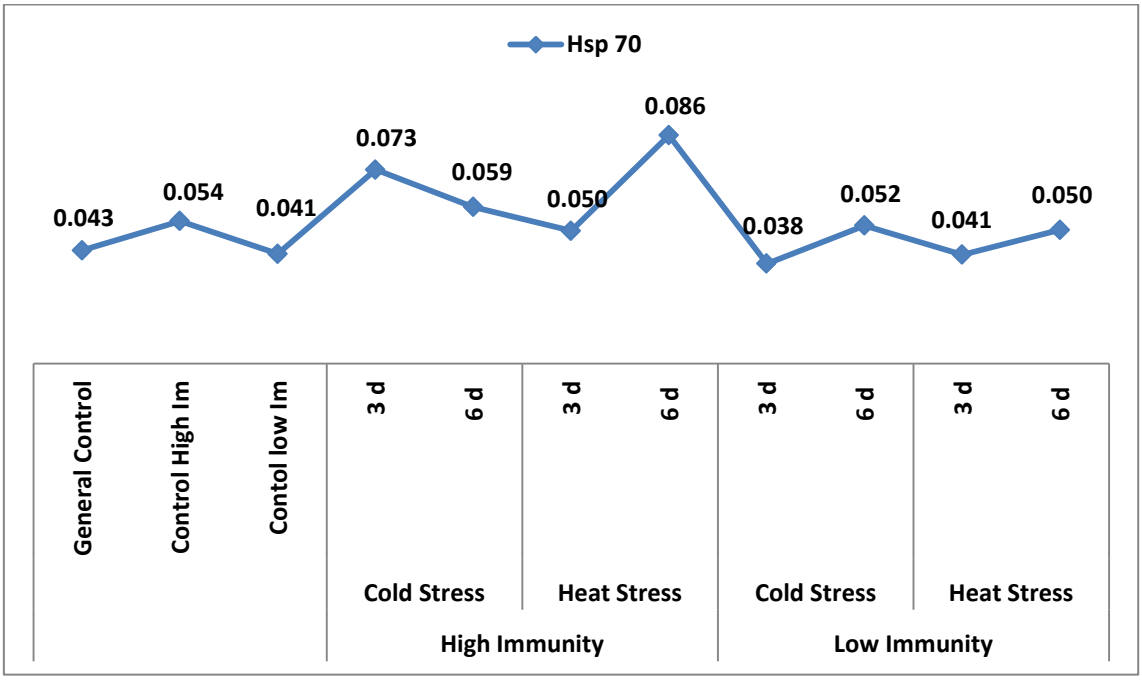


**Fig (2): The interaction between immunity levels, chronic thermal stress and periods of exposure on heat shock protein (Hsp70) of Norfa chickens.**

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# العلاقة بين الاستجابه المناعية و بروتين الصدمة الحرارية 70 تحت تأثير إجهاد البرودة والحرارة في دجاج النورفا

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**الملخص العربي**

أجريت هذه التجربة خلال الفترة من 2014 إلى 2015م بمزرعة بحوث الدواجن التابعة لقسم إنتاج الدواجن،كلية الزراعة، جامعة المنوفية، شبين الكوم، مصر. والهدف من البحث دراسة العلاقة بين الاستجابة المناعية و بروتين الصدمة الحرارية تحت تأثير تعرض الطيور للإجهاد بالبرودة (10 درجة مئوية / 76 % رطوبة نسبية) والإجهاد بالحرارة (37 درجة مئوية / 57 % رطوبة نسبية) للتنبؤ الطيور الصغيرة علي تحمل الإجهاد في الأعمار الكبيرة . والعدد الكلي للطيور المستخدم في التجربة 322 ذكور و إناث لسلاله النورفا عند عمر 12 أسبوع. قسمت الطيور لثلاث مجموعات من المناعة (عالية – منخفضة – كنترول) باستخدام غرف الأقلمة، وقسمت المجموعات السابقة الي 6 تحت مجموعات ( 3 معرضه لإجهاد الحرارة ، و 3 معرضه لإجهاد البرودة). وهذه المجموعات تحت نظامي تعريض(حاد و مزمن).

تم تلخيص النتائج المتحصل عليها كما يلي:-

بالنسبة للتعرض للإجهاد الحاد، سجلت الطيور ذات المناعة العالية والتى تعرضت للإجهاد بالبرودة لمدة 6 ساعات أعلى قيم لبروتين الصدمة الحرارية (0.0045±0.056) وكانت الفروق معنوية بينها وبين المجموعات الأخرى من الطيور عدا الطيور عالية المناعة والتى تعرضت للإجهاد بالحرارة لمدة 3 ساعات. بالنسبة للطيور منخفضة المناعة كانت الفروق بين كل المستويات غير معنوية.

بالنسبة للتعرض للإجهاد المزمن، الطيور عالية المناعة والتى تعرضت للإجهاد بالحرارة العالية لمدة 6 أيام قد سجلت أعلى مستويات لبروتين الصدمة الحرارية (0.0031±0.086) مع فروق معنوية بينها وبين المجموعات الأخرى. بالنسبة للطيور منخفضة المناعة والتى تعرضت للإجهاد بالبرودة لمدة 6 أيام قد سجلت مستويات عالية من بروتين الصدمة الحرارية أكثر من الطيور التى تعرضت لنفس الإجهاد ولكن لمدة 3 أيام فقط.

توضح التجربة إمكانية استخدام مستوي المناعة في الأعمار الصغيرة للكتاكيت للتنبؤ بمدي تحمل الدجاج في الأعمار الكبيرة للإجهاد الحراري عن طريق إفراز بروتينات الصدمة الحرارية 70 للطيور المعرضة لإجهادي الحرارة والبرودة.