Available online at www.scholarsresearchlibrary.com

Scholars Research Library

Annals of Biological Research, 2013, 4 (7):91-93
(http://scholarsresearchlibrary.com/archive.html)

ISSN 0976-1233
CODEN (USA): ABRNBW

Evaluation the affects of heat stress on performance and newcastle disease in broiler breeder farm

Adel Feizi*, Babak Farkhad Hoshyar, Sasan Hamzei, Javad Khanzade, Hasan Arab and Peyman Bijanzad

Department of Clinical Sciences, Faculty of Veterinary Medicine, Tabriz Branch, Islamic Azad University, Tabriz, Iran

---

ABSTRACT

The birds are sensitive to stressor factors. Heat stress is important among stressor agents. By increase of temperature more that the conformity of the bird with temperature is imbalanced and it leads to reduction of bird performance. The purpose of this study was to investigate the effects of heat stress on Newcastle disease and performance of broiler breeders. In this research two sections of a big broiler hen farm were studied. All nutritional and management factors like vaccination, nutrition, density and water resource and environment were the same and only the temperature was different during production. In farm 1 the temperature was high and uncontrolled, but in farm 2 the temperature was controlled by temperature control system. In farm 1 the clinical and autopsy symptoms of Newcastle disease and falling of production until 18% and 4.5% death were observed as a result of increase of temperature after 20 days. In farm 2, the outcomes of heat stress was not seen, because of controlling temperature in houses by ventilation and cooling system. It should be pointed that the average of Newcastle vaccines titer was 7 and its CV average was 18 before production in both farm. There was a significant difference among blood and biochemical factors like lymphocyte, and heterophil in both farm (p<0/05).

Keywords: heat stress; broiler breeder; performance; Newcastle disease

---

INTRODUCTION

Heat stress occurs when birds have difficulty in balancing body heat production and body heat loss. At high environmental temperatures, birds rely on different mechanisms to regulate their body temperature within a zone of comfort, the thermo neutral zone (12). High temperature seems to be the main factor which lowers the productive performance of layer (8). Ambient temperature and diet can influence the acid base balance in poultry. However, birds are equipped to regulate body fluid pH during metabolism. Most final metabolites are acids, and unless regulated, these can accumulate in the body and alter acid-base balance from its normal status. Acids can be removed from the body by the kidneys and lungs. Excess acids in the blood (H+) can combine with bicarbonate ions (HCO3 −) to form H2CO3 (carbonic acid), which is converted into CO2 and H2O by the action of carbonic anhydrase. The CO2 resulting from this reaction is removed by the lungs, and the H+ ions are excreted by the kidneys with the HCO3 − retained to maintain the acid-base ratio. Therefore, to maintain the acid-base balance, birds have to regulate acid uptake and excretion (11). Alkalosis is a pathologic condition resulting from accumulation of base by uptake, or loss of acid from the body by excretion, and is characterized by a decrease in hydrogen ion (H+) concentration or increase in pH. Respiratory alkalosis is a state due to excess loss of CO2 from the body. Metabolic alkalosis is a disturbance in which the acid-base status of the body shifts toward the alkaline side because of changes in the fixed (nonvolatile) acids and bases (4).

Blood heterophils are phagocytic cells designed to defend the organism against infections by bacteria, viruses, or foreign particles. They are present in abundance at infection sites to which they are attracted by chemotactic compounds from injured cells. Lymphocytes play an important physiological role in immunity, particularly for the production of antibodies. One of the physiological responses of exposure to stress is the release of glucocorticoids, causing dissolution of lymphocytes in lymphoid tissues and leading to lymphopenia. However, there is an increase in heterophil release by the bone marrow, thus increasing their number in circulation, but their phagocytic and bactericidal activities are decreased (1, 13). In a review, Zulkifli and Siegel (1995) indicated that stress can reduce the number of circulating lymphocytes and increase the number of heterophils in chickens (14). Thus birds could easily infected with viral disease specially Newcastle disease.

The aim of this study was to investigate heat stress effects on Newcastle disease and performance of broiler breeder farms.

MATERIALS AND METHODS

In this research two sections of a big broiler breeder farm were studied. All nutritional and management factors like vaccination, nutrition, density, water resource and environment were the same and only the temperature was different during production. In section one temperature increased to $38^{\circ c}$, but in section two, temperature was lowered to $28^{\circ c}$ with good performance of cooling system and temperature control system. During heat stress in both sections production rate, clinical signs were recorded, and also from two farm sections blood was obtained and heterophil and lymphocytes was studied and heterophil to lymphocyte ratio calculated.

RESULTS

In section 1 the temperature was up to $38^{\circ c}$ and because of heat stress newcastle diseases was seen in this section after 20 days, that the symptoms was falling of egg production up to 18% during stress period in comparison to section two, and clinical signs and lesions of newcastle disease was observed in section one. The mortality rate caused by Newcastle disease was about 4.5 percent. In section two because of controlling temperature and use of cooling systems that decrease temperature to $28^{\circ c}$ there was not any problem and production was normal. It should be pointed that the average of Newcastle vaccines titer was 7 and its CV average was 18 before production in both farm.

There was a significant difference among lymphocyte, heterophil count and Heterophil to Lymphocyte ratio (H/L) in both farm ($p<0.05$). Lymphocyte count was decreased significantly in heat stressed section and heterophil count was increased significantly in heat stressed section. H/L ratio in section one that was under heat stress increased significantly ($p<0.05$).

DISCUSSION

The results showed very clearly the significant ($p<0.05$) effect of increasing ambient temperatures on broiler breeder performance. The depression in the egg production might be due to many factors which include decreasing feed consumption (5), inefficient digestion (7), impaired metabolism (6), genetic makeup of birds (2).

Broiler breeder pullets are placed under feed restriction starting at about 14 days of age. Two methods of quantitative restriction are used for pullets. Birds can be fed either restricted amounts daily or on a skip-a-day program. For hot climates, the heat production of birds on every-day feeding is about 10% lower than those on a skip-a-day feeding (10). The use of vitamin supplements to improve performance of breeder flocks in hot climates has been studied by few workers. It is fairly well established that fertility in hot climates can be improved in breeder flocks by the addition of extra amounts of vitamin E if breeder rations contain the usual level of 15-20 mg/kg of diet. The addition of ascorbic acid to breeder rations of both chickens and turkeys has yielded positive responses in many cases. Supplementation of broiler breeder feeds with 300 ppm ascorbic acid during hot summers in the Eastern Mediterranean region improved performance (3). Researchers showed that the mean egg weight in layers supplemented with vitamin C was higher ($p<0.05$) than non-supplemented groups under heat stress condition. This indicates that vitamin C was effective in improving egg weight consistently in layers during summer (8). Also (9), who reported that vitamin C supplementation increased egg weight under high temperature and relative humidity. The mean shell thickness of eggs in layers supplemented with vitamin C was higher ($p<0.05$) than those of non-supplemented groups (8), and production rate was decreased in heat stressed group with any supplementation (8). Results of our study in case of production decrease was in agreement with khan et al., 2005, lazer et al., 1983, and Cier et al., 1992, that showed heat stress cause decrease in egg production.

One of the physiological responses of exposure to stress is the release of glucocorticoids, causing dissolution of lymphocytes in lymphoid tissues and leading to lymphopenia. However, there is an increase in heterophil release by the bone marrow, thus increasing their number in circulation, but their phagocytic and bactericidal activities are decreased (1, 13). In a review, Zulkifli and Siegel (1995) indicated that stress can reduce the number of circulating lymphocytes and increase the number of heterophil in chickens (14). Our results showed that after 20 days of heat stress lymphocyte counts was decreased and heterophil counts was increased and H/L ratio was increased significantly in heat stressed section and this results was in agreement with previous studies by Berne et al., 1998, and Swenson et al., 1996 and Zulkifli et al., 1995 that they mentioned similar results. Lymphopenia that caused by stress due to release of glucocorticoids cause chickens vulnerable to viral disease as seen in our studies the section that was under heat stress infected with newcastle disease despite the higher titer of antibodies and desirable CV against newcastle disease before production onset.

Based on the findings of the present study, it may be stated that prevention of heat stress by use of right cooling system is effective in improving performance of broiler breeder farms under heat stress conditions and it could be prevent of chickens to be infected by viral disease.

REFERENCES

[1] R. W. Berne, M. N. Levy: Fisiologia, Guanabara, **1998**.
[2] A. Cahaner, Y. Pinchasov, I. Nir, *Poultry Science*, **1995**, 74968-975.
[3] D. Cier, I. Rimsky, N. Rand, O. Polishuk, N. Gur, A. Benshahan, Y. Frish, A. Benmoshe, 19th World's Poultry Congress, **1992**. 620-621.
[4] N. W. Dorland: Dorland's Illustrated Medical Dictionary, W. B. Saunders, **1965**.
[5] G. C. Emmans, D. R. Charles: Climatic environment and poultry feeding in practice, Ancher Press Ltd, **1989**.
[6] D. J. Farrell, S. Swain, *british Poultry Science*, **1978**, 18735-748.
[7] L. Har, D. Rong, Z. A. Zhang, *Animal Physiology*, **2000**, 8361-75.
[8] S. H. Khan, R. Sardar, *Pakistan Vet. J.*, **2005**, 25(4)**:** 163-166.
[9] J. Lazar, L. Slepcova, D. Magic, T. Kovalcik, F. Jencik, L. Baran, L. Bindas, *Folia Veterinaria*, **1983**, 281-92.
[10] S. Leeson, J. D. Summers: Commercial Poultry Nutrition, University Books, **1991**.
[11] B. Ruiz-Lopez, R. E. Austic, *Poultry Science*, **1993**, 721054-1062.
[12] M. S. Simon, *World Poultry*, **2003**, 19(3)**:** 16-17.
[13] M. J. Swenson, W. O. Reece: Dukes, Fisiologia dos animais dome´sticos, Guanabara, **1996**.
[14] I. Zulkifli, P. B. Siegel, *Worlds Poultry Science Journal*, **1995**, 5163-76.