ISSN: 0975-8585

Research Journal of Pharmaceutical, Biological and Chemical Sciences

Heat Shock Protein In Birds : A Review.

Salman KAA*.

Department of Animal Production, College of Agricultural Engineering Sciences, University of Baghdad, Iraq

ABSTRACT

Living organisms respond to changes in environmental temperature by activation of physiological mechanisms involved in heat loss or production. Several cellular events are triggered when cells and organisms are exposed to stress injury. Nevertheless, the activation of these responses seems to be transient since they are blocked after a return to the non-stressing physiological state. If the animal or the cell is not able to react or adapt to these environmental changes, homeostasis may be compromised and even death may occur. The physiological and molecular mechanisms involved in the stress response have been extensively investigated in a wide variety of species. It has been well characterized that this emergency response is marked by a drastic reduction in cellular protein synthesis, except for a set of proteins named heat shock proteins (HSPs).

Keywords: Heat shcok protein, birds

Corresponding author

ISSN: 0975-8585

INTRODUCTION

Heat stress is one of the most challenging environmental conditions affecting on an animal's physiology and performance of poultry. Different mechanisms are utilized to resist the harmful effects of high temperature depending on duration of exposure. Short-term sub-lethal heat stress (acute heat stress) provokes heat shock response, and it causes the loss of revenue that ranges into millions of dollars each year (1). Exposure of chickens to heat stress causes significant behavioral and physiological responses (2). Thermal stress exerts negative influence on feed intake, BW gain (3), neuroendocrine, and immune function as well as on mortality rates (4). Heat stress seriously harms animal welfare and productivity, and therefore, becomes one of the major concerns for the poultry industry, especially in the hot regions of the world.

When living organisms are exposed to thermal stresses, the synthesis of most proteins is delayed, but a group of highly conserved proteins known as heat shock proteins or heat stress proteins (HSP) is rapidly synthesized (5). These HSP play an important role in the survival of stressed cells and the stabilization of the internal environment (6).

Heat shock proteins (HSPs) are widely distributed in nature and are among the most highly conserved molecules of the biosphere. hsp perform important functions in the folding and unfolding or translocation of proteins, as well as in the assembly and disassembly of protein complexes. Because of these helper functions, hsp have been termed molecular chaperones. The molecules involved in antigen recognition, i.e., immunoglobulins (Ig), T-cell receptors (TCR), and gene products of the major histocompatibility complex (MHC), are all multimeric complexes, and their assembly is promoted by distinct chaperones. Several lines evidence also favor an important role for members of the hsp family in intracellular antigenprocessing pathways. The first part of this review describes the biological roles of hsp as they relate to the assembly of protein complexes and participation in different processing and presentation steps of antigens (7).

Heat shock protein are a class of polypeptides powerfully induced by heat shock that mediate profound levels of stress resistance (8), (9) and co- workers first discovered that subjecting Drosophila melanogaster larvae to temperature shock induced specific gene activation, however, it was not until 1974 that the first products of these genes were identified and the term 'heat shock protein' was adopted (10), Subsequent work has demonstrated that heat shock proteins are present, and can be induced, in all species and that they are among the most phylogenetically conserved proteins.

HSPs synthesis is increased to protect prokaryotic or eukaryotic cells from various insults during periods of stress caused by infection, inflammation, or similar events or other stress conditions including exposure to cold (11), UV (12), wounded healing or reshaping tissues (13) and, as well as heavy metals and toxic organic, high temperature and other factors that affect the cells which responsible for the restructuring of protein and called stress proteins and occur in both prokaryotic or eukaryotic and the concentration of heat shock proteins in children or adults Increases rapidly when cells are exposed to stress, and this increase leads to significant changes in gene expression, leading to muscle and skeletal reconstruction (14).

HSPs are molecular chaperones (are proteins that assist the covalent folding or unfolding and the assembly or disassembly of other macromolecular), binding to (holding) and refolding other cellular polypeptides (clients) with aberrant conformations (11), by stabilizing new proteins to ensure proper folding of proteins or by helping to restore proteins that have been damaged as a result of cell stress (15), regulating the work of heat shock proteins is a major part of the response to heat shock, due to the presence of a heat shock factor (HSF) (16).

Heat shock proteins are ategorised into several families that are named on the basis of their approximate molecular mass (HSP 60, 70 and 90) depended on its molecular weight 60,70 and 90 kDa respectively (17), Whereas HSP 60 important for protein stability (18), and HSP 70 family are necessary for protein synthesis and transport (19) and HSP 90 is important in the formation of the steroid receptor complex 20).

Heat shock protein–peptide complexes (HSP.PC) can be used as vaccines to elicit antigen-specific cytotoxic lymphocytes (CTL) responses (21, 22, 23, 24, 25). In order for polypeptides bound to HSPs to activate adaptive immunity, associated antigens must be internalized by antigen presenting cells (APC) and inserted

into the antigen presentation pathways. Indeed, HSPs have been shown to be taken up by dendritic cells (DC), the most efficient professional APC (26). Antigen presentation occurs through a number of pathways. Intracellular proteins were shown to be processed by digestion through the multiple protease activities in the proteasome and antigens presented on the cell surface by major histocompatibility class I (MHCI) molecules found in all cells and thus displayed to CD8+ T cells to permit immunosurveillance (27). By contrast, exogenous antigens after internalization into immune cells are processed in lysosomes and presented on the cell surface by major histocompatibility class II (MHCII) molecules restricted to cells of the immune system (28). It was subsequently shown that another antigen presentation pathway exists permitting external antigens to enter the MHC class I pathway (29). This process, antigen cross presentationpermits external antigens to be presented by APC in the context of MHC class I and activate CD8+ CTL to kill virus infected or malignant cells (29). Antigen cross presentation was shown to be a complex process requiring external antigens to enter cells, penetrate sites for protein processing, and associate with MHC class I molecules in intracellular vesicular structures although many aspects of this process are incompletely understood. We aim to elucidate some of the mechanisms by which antigens bound to HSP interact with APC and mediate antigen cross presentation as compared with the pathways utilized for unchaperoned antigens.

In recent years, studies have found that there is some correlation between high-temperature stress on the chicken pathological lesion and the expression of HSP (30,31), the intestine is susceptible to heat stress, hypoxia, and other environmental factors, which result in mucosal damage. Studies found that a variety of stress factors in the intestinal tract, such as endotoxins, arsenite, ethanol, and ischemia may stimulate the production of HSP70 (32, 33). It is said that damage to the intestinal mucosal structure and digestion absorption function under heat exposure is the main factor for decreased feed intake and feed conversion (34). (35) 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. Studies of (36) and (37) found that heat stress caused marked damage to porcine intestinal epithelia, which included damage to the tips of the intestinal villi, inducing epithelial cell shedding, exposing the intestinal mucosa lamina propria, as well as shortening villus height and crypt depth.

Changes in digestive enzyme activity may be one reason for the change in digestive function under heat stress. However, studies focused on the effects of heat stress on the poultry digestive enzyme activity are few at present.therfore, the studies demonstrates the overexpression of HSPs may increase alkaline phosphatase activity. Intestinal alkaline phosphatase is the traditional marker of intestinal maturation and is located in the small intestinal epithelium villus brush border and regulated by endotoxins of intestinal normal flora (38). It is related to intestinal digestion and absorption and plays an important role in the maintenance of normal intestinal barrier function (39). Its change may reflect the change in intestinal digestion and absorption; however, the underlying mechanism is not clear.

The study of (40) showed that, after 4 wk of continuous heat exposure under 34.7°C, the total proteolytic enzyme, lipase, and amylase activity in broiler intestinal contents were significantly decreased compared with that of the control group at 49 d of age. On the contrary, (41) reported that pancreatic trypsin and lipase were not significantly different compared with those of the control group, but the pancreas amylase activity significantly decreased under acute heat stress. The amylase, lipase, and trypsin activity data presented here indicate that heat stress has a significant influence on the digestive enzyme activity.

CONCLUSION

From this study we conclude that heat shock proteins affected by increased temperature and effect on body physiological performance in broiler, so HSPs make intercelural adapted to exposed to heat stress, therefore, maintains the function of cells physiology, this led to maintains birds performance.

REFERENCES

[1] Mahmoud, K. Z.,Edens, F. W.,Eisen,E. J. and Havenstein,G. B.2003. Effect of ascorbic acid and acute heat exposure on heat shock protein 70 expression by young white leghorn chickens.Comp. Biochem. Physiol. C Toxicol. Pharmacol.136:329–335.

[2] Altan,O.,Pabuccuoglu,A.,Altan,A.,Konyalioglu,S. and Bayraktar,H.2003.Effect of heat stress on oxidative stress, lipid peroxidation, and some stress parameters in broilers.Br. Poult. Sci.44:545–550.

[3] Geraert,P. A.,Padilha,J. C. and Guillaumin,S.1996. Metabolic and endocrine changes induced by chronic heat exposure in broiler chickens: Growth perfo mance, body composition, and energy retention.Br. J. Nutr.75:195–204.
[4] Smith,M. O.1993. Parts yield of broilers reared under cycling high temperatures.Poult. Sci.72:1146 1150.
[5] Al-Aqil,A. and Zulkifli, I. 2009. Changes in heat shock protein 70 expression and blood characteristics in transported broiler chickens as affected by housing and early age feed restriction.Poult. Sci.88:13581364.
[6] Gabai, V. L.,Meriin,A. B.,Mosser,D. D.,Caron,A. W.,Rits,S.,Shifrin,V. I. and Sherman,M. Y.1997. HSP70 prevents activation of stress kinase-A novel pathway of cellular thermotolerance.J. Biol. Chem.272:18033–18037.
[7] Zugel,U. and Kaufmann,H.E.1999. Role of Heat Shock Proteins in Protection from andPathogenesis of Infectious Diseases. CLIN. MICROBIOL. REV.,12(1):19-39.
[8] Craig, E. A. 1985. The heat shock response. CRC Crit. Rev. Biochem. 18, 239.
[9] Ritossa, F.A. 1962. A new puffing pattern induced by temperature shock and DNP in Drosophila. Experientia 18, 571-573.
[10] Tissieres, A., Mitchell, H.K. and Tracy, U.M. 1974. Protein synthesis in salivary glands of Drosophila melanogaster: relation to chromosome puffs. J Mol Biol 84, 389-398.
[11] Ellis, R. J. 2007. Protein misassembly: macromolecular crowding and molecular chaperones. Adv. Exp. Med. Biol. 594, 1–1310.
[12] Cao, Y., Ohwatari, N., Matsumoto, T., Kosaka, M., Ohtsuru, A. and Yamashita, S. 1999. TGF-beta1 mediates 70-kDa heat shock protein induction due to ultraviolet irradiation in human skin fibroblasts. Pflügers Archiv. 438 (3): 239–44.
[13] Laplante, A.F., Moulin ,V., Auger, F.A., Landry, J., Li, H., Morrow, G., Tanguay, R.M. and Germain, L. 1998.Expression of heat shock proteins in mouse skin during wound healing. The Journal of Histochemistry and Cytochemistry. 46 (11): 1291–301.
[14] Tkáčová,J. and Angelovičová,M.2012. Heat Shock Proteins (HSPs): a Review. Animal Science and Biotechnologies, 45 (1),PP:349-353.
[15] De Maio,A.1999. Heat shock proteins: facts, thoughts, and dreams. Shock. 11(1): 1–12.
[16] Wu,C.1995. Heat shock transcription factors: structure and regulation. Annual Review of Cell and Developmental Biology. 11: 441–69.
[17] Murshid,A.,Gong,J. and Calderwood,S.K.2012. The Role of Heat Shock Proteins in Antigen Cross Presentation.
[18] Richter-Landsberg, C. and Goldbaum, O. 2003. Stress proteins in neural cells: functional roles in health and disease, Cellular and Molecular Life Sciences, , 60, 337-349.
[19] Geething, M.J., Sambrook, J. 1992. Protein folding in the cell, Nature. 355, 33-45.
[20] Pratt,W.B.1997.The role of the HSP90 based chaperone system in signal transduction by nuclear receptors and receptor signalling via map dinase, Annual Review of Pharmacology and Toxicology. 37, 297-326.
[21] Srivastava P. K. 2000. Immunotherapy of human cancer, lessons from mice. Nat. Immunol. 1, 363–366.
[22] Manjili M. H., Henderson R., Wang X. Y., Chen X., Li Y., Repasky E., Kazim L. and Subjeck J. R. 2002. Development of a recombinant HSP110-HER-2/neu vaccine using the chaperoning properties of HSP110. Cancer Res. 62, 1737–1742.
[23] Xang X. Y., Kazim L., Repasky E. A. and Subjeck J. R. 2003. Immunization with tumor-derived ER chaperone grp170 elicits tumor-specific CD8+ T-cell responses and reduces pulmonary metastatic disease. Int. J. Cancer 105, 226–231.
[24] Enomoto Y., Bharti A., Khaleque A. A., Song B., Liu C., Apostolopoulos V., Xing P. X., Calderwood S. K. and Gong J. 2006. Enhanced immunogenicity of heat shock protein 70 peptide complexes from dendritic cell-tumor fusion cells. J. Immunol. 177, 5946–5955.
[25] Gong J., Zhang Y., Durfee J., Weng D., Liu C., Koido S., Song B., Apostolopoulos V. and Calderwood S. K. 2010. A heat shock protein 70-based vaccine with enhanced immunogenicity for clinical use. J. Immunol. 184, 488–496.
[26] Heath W. R. and Carbone F. R. 2009. Dendritic cell subsets in primary and secondary T cell responses at body surfaces. Nat. Immunol. 10, 1237–1244.
[27] Neefjes J. J. and Momburg F. 1993. Cell biology of antigen presentation. Curr. Opin. Immunol. 5, 27–34.
[28] Cresswell P. 1994. Assembly, transport, and function of MHC class II molecules. Annu. Rev. Immunol. 12, 259–293.

[29] Norbury C. C., Basta S., Donohue K. B., Tscharke D. C., Princiotta M. F., Berglund P., Gibbs J., Bennink J. R. and Yewdell J. W. 2004. CD8+ T cell cross-priming via transfer of proteasome substrates. Science 304, 1318–1321.

[30] 30-Bao,E.D.,Gong,Y.Y.,Hartung,J.,Fu,X.B.,Wang,X.J.,Zhang,H.T. and Wang,Z. L.2004. Relation between pathologic damages and HSP70, changes in acute heat-stressed broilers.Sci. Agric. Sinica37:301–305.

[31] Sun,P. M.,Liu,Y. T.,Zhao,Y. G.,Bao,E. D. and Wang,Z. L.2007. Relationship between heart damages and HSP mRNA in persistent heat-stressed broilers.Agric. Sci. China6:227–233.

[32] Beck,S. C.,Paidas,C. N.,Mooney,M. L.,DeitchE. A. and De MaioA.1995. Presence of the stress-inducible form of hsp-70 (hsp-72) in normal rat colon.Shock3:398–402.

[33] Tsukimi,Y. and Okabe,S.2001. Recent advances in gastrointestinal pathophysiology: Role of heat shock proteins in mucosal defense and ulcer healing.Biol. Pharm. Bull.24:1–9.

[34] Ryder,A. A.,Feddes,J. J. R. and Zuidhof,M. J.2004. Field study to relate heat stress index to broiler performance.J. Appl. Poult. Res.13:493–499.

[35] Ning,Z. Y.,Liu,S. D.,Zhao,D. M.,Tan,X. and Yu,S. M.2003. The influence of heat stress on morphological and ultrastructure change of respiratory, digestive, and endocrine tissues in broilers.Acta Vet. Zootech. Sinica 34:558–561.

[36] Liu,F.,Yin,J.,Du,M.,Yan,P.,Xu,J.,Zhu,X. and Yu,J.2009. Heat-stress-induced damage to porcine small intestinal epithelium associated with downregulation of epithelial growth factor signaling.J. Anim. Sci. 87:1941–1949.

[37] Yu,J.,Yin,P.,Liu,F. H.,Cheng,G.L.,Guo,K. J.,Lu,A.,Zhu,X.Y.,Luan,W.L. and Xu,J.Q. ,2010 . Effect of heat stress on the porcine small intestine: A morphological and gene expression study.Comp. Biochem. Physiol. A Mol. Integr. Physiol.156:119–128.

[38] Alpers,D. H.,Zhang,Y. and Ahnen,D. J.1995. Synthesis and parallel secretion of rat intestinal alkaline phosphatase and a surfactant-like particle protein.Am. J. Physiol.268:E1205–E1214.

[39] Bates,J. M.,Mittge,E.,Kuhlman,J.Baden,K. N.,Cheesman,S. E. and Guillemin,K.2006. Distinct signals from the microbiota promote different aspects of zebrafish gut differentiation.Dev. Biol.297:374–386.

[40] Ruan,H. and Niu,D.,2001. Study on hot stress reducing activities of digestive enzymes in broiler intestine.Chinese J. Anim. Sci.37:16–17.

[41] Routman,K.S.,Yoshida,L.,Frizzas de Lima,A. C.,Macari,M. and Pizauro,J.M. 2003. Intestinal and pacreas enzyme activity of broilers exposed to thermal stress.Braz. J. Poult. Sci.5:23–27.