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Identification Heat Stress of Control Genomic Regions in Poultry

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Abstract

Heat stress-induced reduction in feed intake in an annoyance of the poultry industry. High environmental temperature is a serious stress affecting economic and biological efficiency of poultry production in tropical and subtropical regions countries that are expected to become more prominent with global climate change. Iowa experienced 3 acute heat waves of 11, 3, and 4 d of heat index above 38◦C in the summer of 2012, which led to production losses and increased bird mortality. An effective tool for improving heat tolerance can be genomic selection based on single nucleotide polymorphisms. In the past few years, new molecular tools have been developed, such as RNA-Seq, Single Nucleotide Polymorphisms (SNPs), and bioinformatics approaches such as Genome-Wide Association Study (GWAS). Based on these genetic tools, many studies have detected the main pathways involved in cellular response mechanisms. This study aims to identify genomic regions controlling survival against heat stress. Genetic studies conducted on experimental lines can therefore be of potential interest for marker-assisted selection in commercial lines.

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Keywords

Heat stress, poultry, higher metabolic rate, poultry, GWAS.

Introduction

Heat stress is one of the most important environmental stress factors in poultry production worldwide. Chronic heat stress can lead to changes in poultry physiology and affect production. Heat stress is a response to an increase in body temperature, and as one of the most important influencing factors, it causes a decrease in growth performance and the immune system, and as a result, increases in casualties (Bottje and Harrison, 1985). All categories of poultry experience heat stress along with relative humidity and temperature range above the comfort point. The increase of these two variables reduces the ability of the bird to dissipate heat (Belay and Teeter, 1996). Due to a higher metabolic rate, they produce more body heat and are prone to heat stress. High stocking density of birds, along with the high ambient temperature, increases the propensity of heat stress (Goo *et al.,* 2019). The normal body temperature of the chicken is around 41–42 $^{\circ}C$, and the thermoneutral temperature to maximize growth is between 18–21 $^{\circ}C$ (Kumari and Nath, 2018). Studies have shown that any environmental temperature higher than 25 $^{\circ}C$ elicits heat stress in poultry (Donkoh, 1989). Understanding the genetic mechanism and the genetic architecture of the trait and the sensitivity and resistance to heat stress makes it possible to produce commercial strains with the special trait of natural adaptation to hot climate and stress (Quinteiro-Filho *et al.,* 2010). The starting point

for understanding heat stress responses is to identify genes whose expression is regulated by high temperature. High-throughput technologies have made it possible to examine gene expression on a large scale.

Sign of heat stress in poultry

The signs of heat stress in poultry are panting with open mouth, elevated their wings and squatting near to the ground, droopy acting, slowness and lethargic closed eyes, lying down, increased water intake, decreased appetite, drop in egg production, reduced egg size, poor egg shell quality, reduced body weight, and increased cannibalism (Nardone *et al.,* 2010; Dayyani and Bakhtiari, 2013). Birds are trying to lose heat by gasping and changing the position of their feathers, losing water in their breath and cooling by evaporation through the surface of the lungs. Birds are facing heat stress conditions, they spend less feeding time during feeding, more time drinking and panting, less time moving or walking and more time resting (Mack *et al.,* 2013).

Genome wide association studies in poultry

In the case of heat stress, the physiological responses of different organisms to the increase in ambient temperature is heat gain of the body and increase in the blood flow of the skin (Sandercock *et al.,* 2001) These can be regarded as indicators of the body's reaction to regulate its production through elimination of excess body heat (Hocking *et al.,* 1994). Genes regulation related to activation of Heat Shock Factors (HSFs) and their transcription activators have been identified as cellular responses to different types of stresses (Sorger, 1991). Heat shock proteins have been suggested to be the key cellular defense mechanisms in chickens during exposure to hot environments (Cedraz *et al.,* 2017).

Two Predisposing factors that affect heat stress include genetics and the environment. In an experiment, muscle damage in fast-growing broilers were reported to be associated with an ambient temperature (Zahoor *et al.,* 2016). Additionally, it has been indicated that much of the variation in response to heat stress has a genetic basis (Lin *et al.,* 2006; Lu *et al.,* 2007). Soleimani *et al.,* (2011) found that commercial broilers were more susceptible to heat stress than their Red Jungle fowl counterparts. Also, Mack *et al.,* (2013) provided evidence that genetic selection is a useful strategy for reducing the heat stress response in laying hens. Tamzil *et al.,* (2013) demonstrated that a specific group of genes and also some proteins, that are called HSP, involve in heat stress. These proteins vary in different strains and breeds and increases the resistance response of birds against the high temperature of the environment (Xie *et al.,* 2014). Actually, in the case of heat stress, genetic factors affect the physiological responses and different birds and populations show different genetic responses to heat stress (Lu *et al.,* 2007). As a result, the selection of individuals for survivability to heat resistance could help greatly to increase the resistance of the population against heat stress Advances in molecular genetics and broad access to molecular polymorphism information have allowed researchers to select directly for genes and genomic regions influencing traits through marker-assisted selection (MAS) (Dekkers and Hospital, 2002; Lande and Thompson, 1990). association studies (Van Goor *et al.,* 2015) and transcriptome comparison (Coble *et al.,* 2014) have been suggested to implement for studying heat stress. Several quantitative trait loci (QTLs) have has been identified to be associated with heat stress tolerance in Holstein cattle (Melchiorre *et al.,* 2013) and catfish (Zhou *et al.,* 2017). For chickens, few results have been found kinds of the literature and several candidate genes were reported to be associated with heat stress tolerance (Van Goor *et al.,* 2015). The managing of new traits, such as Heat stress resistance, has brought about the development of new technologies to investigate genetic relations with these phenotypes. A good approach to identify the functional genes and polymorphisms correlated with HS resilience has been accomplished by virtue of high-throughput screening technologies, such as genome-wide analyses of genetic variations. The genome-wide Association study (GWAS) has been proposed to be a suitable method for identifying genomic regions affecting the resistance of birds to heat shocks (Bjorkquist *et al.,* 2015; Lamont *et al.,* 2014).

Whole genome profiling has allowed researchers to understand the genetic effect that heat stress has on genetic pathways involved in heat stress modulation. (Lei *et al.,* 2013) They found that pathways play a role in modulating heat stress. These researchers found that genes affecting hormones associated with appetite-regulating peptides affect the thermal process. Exposure to heat causes anorexia due to this stress. Therefore, an important factor in the reduction of feed consumption and the reduction of nutrient consumption in birds is affected by heat. These findings show what strategies should be taken to reduce the negative effect of heat stress and increase weight and improve production. One of these factors is improving genetic performance by creating resistant genes in these animals (Lei *et al.,* 2013). Several studies, mostly in plant breeding,

indicated that both the unbalanced and the balanced data sets detect common SNPs markers, suggesting that the unbalanced data could be used to gain knowledge about the genetics of important traits and identify marker-trait associations for molecular breeding (Johnson *et al.,* 2019; Wang *et al.,* 2012). However, there is published literature indicates that the linear mixed model and logistic mixed model produce large type I error rates in the analysis of unbalanced case-control phenotypes (Zhou *et al.,* 2018). A Scalable and Accurate Implementation Generalized mixed model (SAIGE) was proposed by these authors for GWASs, who indicated that SAIGE controls the type I error rates efficiently even when case-control ratios are extremely unbalanced. Poultry, like other animals, has the possibility to develop specific phenotypes advantageous for adaption to the harsh environment where they live. There are several phenotypes that mainly act for the alleviation of Heat stress, mostly related to feather types. Indeed, feathers guarantee a thermal shelter between the animal body and the environment. Plumage delays the process of heat elimination from the skin surface (Wolf and Walsberg, 2000). As an example, the Naked Neck (Na) chicken shows a better fitness under Heat stress conditions (Eberhart and Washburn, 1993). In a recent study, compared the expression levels of HSP70 (used to assess the heat tolerance) in three Egyptian local breeds (Fayoumi, Dandarawi, and Sinai) with and without the Na gene and under normal and Heat stress conditions. As expected, they found higher HSP70 expression levels in crossbreeds with the Na gene, suggesting that the Na gene is responsible for the up-regulation of HSP70 expression and has a positive impact on HS adaptation not only by reducing feather cover (Galal *et al.,* 2019). Another important feature of the plumage is the color of feathers, which can impact the ability of chickens to respond under Heat stress conditions. A recent study demonstrated that dark chicks showed a lower expression rate of genes belonging to pathways of stress (cellular stress: SOD2 and HSPA8; DNA damage repair: ALKBH3) than paler chicks (Diaz-Real *et al.,* 2017).

The significant Gga_rs16111480 SNP, detected in this study, was located inside the CEP78 gene. The MAF of this SNP (0.095), as a rare variant (MAF $\leq$ 0.10), suggests that it could play a more important role in heat stress tolerance than the other two SNPs, detected in this study (Asadollahi *et al.,* 2022). The CEP78 gene regulates protein activity of PLK1 at G2/M transition in relation to the HSP90 (Senju *et al.,* 2006) and HSP70 (Chen *et al.,* 2014). It is also involved in the maturation of Centrosomes and Centrioles (Brunk *et al.,* 2016), and plays an important role in cell survival. The PLK proteins are important regulators of the cell cycle (Casenghi *et al.,* 2003). This gene, which stimulates family enzymes of PLK, particularly PLK1 (www. uniprot.org) and PLK4 (Brunk *et al.,* 2016), is responsible for the activation and regulation of these proteins. The PLK1 substrates consist of multiple proteins, engaged in mitosis, including CDC25C, APC and CLYN B, which are responsible for the regulation of the tyrosine dephosphorylation in CDKs (Kraft *et al.,* 2003).

Previous studies have shown some interactions between PLK1 and HSF-1 proteins. These interactions, which increase during thermal stress, are caused by serine dephosphorylation at 419 serine sites through HSF-1 protein. Mutations that change serine 419 into alanine inhibit heat stress due to nuclear translocation. HSF-1 activity requires phosphorylation by PLK1 protein, and it is a necessary step during heat stress (Kim *et al.,* 2005). In normal cells, HSF-1 greatly exists as an inactive monomer inside the cytoplasm. By heat stress exposure, HSF-1 is begun to transition from a monomer to a homotrimer form and is transferred to the nucleus and binds to DNA, resulting in phosphorylation on several amino acids. Subsequently, HSF-1 is attached to heat shock elements (HSEs) that have been formed from repeated opposite structures of 5 nucleotide motifs in the promoter site of the HSP gene and thereby increase the gene expression of HSP including HSP70 and HSP90 isoforms. In different organisms, HSF-1 is a prime integrator of transcriptional responses during stress., HSF-1 activity increases the cellular level of heat shock proteins (HSP) (Kim *et al.,* 2005), and these proteins act as cell protectors for denaturation of defective proteins and are also used to survival mechanisms in protein-damaging conditions to protect the cell (Zhang *et al.,* 2002). Heat shock proteins, especially HSP70 (Silver and Noble, 2012) and HSP90, have a critical role in cell survival in response to stressful environments (Gupta *et al.,* 2010). As a result, enzymes PLK (PLK1, PLK4) are affected by the CEP78 gene. Due to HSF-1 phosphorylation, these enzymes will activate heat shock proteins, as the activity of these proteins is also a key impact on cell and body resistance against heat stress, so in different organisms, the CEP78 gene plays an important role in heat stress resistance.

Myocyte enhancer factor 2C (MEF2C) was the closest gene to two SNPs, namely GGaluGA354375 and Gga_rs14748694, significantly associated with survivability to heat stress. Usually, two specific cellular response incidents are to a variety of stresses, such as

activation of heat shock factors (HSFs) and activators of transcription of these genes (Sorger, 1991), and the activation of JNKs and P38 MAP kinase, which have been identified as active protein kinases in stressful environments (Koul *et al.*, 2013). Both protein kinases are subgroups of the MAPK family (Koul *et al.,* 2013) and are involved in various complex biological processes in cells and deeply respond to stress signals (Vassalli *et al.,* 2012). Literature studies have shown that the regulation of protein kinases activity of the MAPK family is influenced by the MEF2C gene (www. unipr ot.org), and also phosphorylation and activation of MEF2C are affected by MAPK protein kinases. These protein kinases have special effects on amino acids including serine, threonine and tyrosine (Cleveland, 1923), and they are involved in managing and directing cellular responses to a wide type of stimulants such as mitogen and osmotic stress, heat shock, and proinflammatory cytokines and are also engaged in the regulation of some cell functions including proliferation, gene expression, differentiation, cell survival and apoptosis (Pearson *et al.*, 2001). Chu *et al.*, (1996) indicated that part of the activation of HSF-1 is regulated by MAPK inside the body and one or more members of the MAPK family is involved in phosphorylation and regulation of HSF-1 activity. Furthermore, the MEF2C is well known as GLUT4 enhancer protein and appears to be the pivotal regulator of GLUT4 expression. An increase in expression of GLUT4 was associated with increased MEF2C. It has been reported that pigs under chronic heat stress increase the respiratory frequency and body temperature and modify the higher expression of HSP90 and GLUT4 (Cervantes *et al.,* 2016; Garcia *et al.*, 2012). However, some results indicated that GLUT4 was not identified in chickens and proposed that the hyperglycemia, which might have toxic effects on the liver, and insulin resistance observable in chickens is due to the possible deficiency of GLUT4 (Gochee *et al.*, 2002). For this study, these biological pathways indicate that CEP78 and MEF2C genes plays an important role in enhancing the organism resistance against heat stress through regulating the activity of MAPK protein kinase, activating HSF-1, and increasing levels of heat shock protein.

The Gga_rs16111480 SNP detected in this study was located 243 kb downstream of the VPS13A gene (Asadollahi *et al.,* 2022). In recent years, the VPS13A gene, which encoded Chorein, has been recognized as a key regulator of secretion and aggregation of blood platelet in heat stress conditions and has an important role in the production and regulation of blood platelets (Schmidt *et al.,* 2013). Chorein protein is derived from a wide variety of human tissues and red blood cells as well as the original skin fibroblasts (Dobson-Stone *et al.*, 2004). The heat stress raises the total number of platelets and blood viscosity. This condition may increase the physiological damages and also the risk of cerebral and coronary thrombosis (Keatinge *et al.*, 1986). ARRDC3 gene, which was located 947 kb and 888 kb upstream of the two GGaluGA354375 and Gga_rs14748694 SNPs, respectively, directly involved in the resistance to high temperatures (www.unipr ot.org). A high level of ARRDC3 stimulates the norepinephrine by increasing cAMP signalling that it lowers energy consumption and ultimately will reduce the heat production in the body (Patwari *et al.,* 2011). In recent studies, the regulation of G protein-coupled receptors (GPCRs) degradation and trafficking due to ARRDC3 has been reported (Dores *et al.,* 2015). It has been suggested that the processing of both wild-type and mutant GPCRs is promoted by HSP70 proteins (Meimaridou *et al.,* 2009). Furthermore, it has been indicated that ARRDC3 is linked to the regulation of adrenergic signalling through interaction and regulation of ubiquitination of the β2 adrenergic receptor (Batista *et al.,* 2020). Andersson *et al.,* (2011) indicated that the stimulation of β-adrenergic has resulted in an increase in the mitochondrial reactive oxygen species (ROS) production in cardiomyocytes. While the role of ROS in ageing is inconsistent in the pieces of literature, it has generally been observed that the lifespan decreases as the ROS level increases (Shields *et al.*, 2021). From the above results, it can be concluded that survivability to high temperature might be due to the SNP markers that are located inside or close to the genes such as CEP78, MEF2C, VPS13A and ARRDC3, which had relatively biological pathways in heat shock resistance.

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