CHAPTER VI
OVERALL CONCLUSION AND IMPLICATION

6.1 Overall conclusion

Heat stress (HS) in poultry causes significant economic losses and threatens poultry health and welfare globally. This study used transcriptomic techniques to identify mechanisms and gene markers in jejunal mucosal tissue of heat-adapted and heat-sensitive breeder hens to enhance chicken thermotolerance. Moreover, the study evaluated the effectiveness of either synthetic (a combination of vitamin E, vitamin C, Se, and L-carnitine) or phytogenic (a combination of clove, green tea pomace, and Vietnamese coriander) antioxidants in heat-sensitive breeder hens to determine their potential in alleviating HS. The main results are summarized as follows:

6.1.1 In heat-sensitive breeder hens under acute HS, 138 DEGs were identified, enriched in pathways including steroid biosynthesis, protein processing in endoplasmic reticulum, PPAR signaling, and adipocytokine signaling. Acute HS affected energy metabolism, fat metabolism, and glucose transport in the jejunal mucosa. The different expressions of HSPB9, HSPA2, IL-18BP, and CD36 genes have the potential to serve as gene markers indicative of HS effects in the jejunal mucosal tissue of heat-sensitive breeder hens. Furthermore, supplementation with either synthetic or phytogenic antioxidants induced upregulation of CD36 and downregulation of HSPB9, HSPA2, and IL18BP, which improved intestinal health by enhancing immune response, lipid and energy metabolism in breeder hens, thereby repairing HS damage in breeder hens.

6.1.2 Comparative analysis between heat-adapted and heat-sensitive breeds revealed 284 differentially expressed genes (DEGs) associated with response to heat, cell division, and transport of glucose and amino acids. Key pathways included VEGF signaling, MAPK signaling, steroid biosynthesis, cell adhesion molecules, neuroactive ligand-receptor interaction, and cell cycle. KEGG pathway and PPI analyses showed that acute HS may affect the cell cycle (CDK1, PLK1, CDC7, and CDC20), immunity (HSPA2, IL6), and organic acid (SLC22A13L), glucose, and fatty acids transport (LBFABP,

SLC2A2) in the jejunal mucosa of breeder hens. Heat-stressed hens increase the expression of HSPs as a protective mechanism for their cells. These changes might indicate that jejunal mucosal tissue was more damaged by HS in heat-sensitive breeder hens than in heat-adapted breeder hens. Nine candidate genes, including HSPB9, HSPA2, RAG2, CD36, CLDN15, LBFABP, SLC22A19A, SLC2A2, and IL18BP, may play key roles in the regulation of the jejunal mucosa of breeder hens with acute HS.

6.1.3 Antioxidant supplementation effectively alleviated HS negative effects by enhancing antioxidant status (SOD, GSH-Px), modulating the expression of immune-related and tight junction genes (IL-10, IL-6, TNF-α, NF-κB, TLR4, and CLDN1), increasing cecal SCFAs concentrations, reducing ammonia production, and enhancing beneficial bacteria of cecal microbiota. In addition, both antioxidant sources also changed the expression of marker genes (HSPB9, HSPA2, RAG2, CLDN15, IL-18BP, and CD36) identified in heat-sensitive breeder hens, contributing to improving intestinal integrity and function under thermal challenge.

6.2 Implication

6.2.1 The identified gene markers (HSPB9, HSPA2, IL-18BP, and CD36) provide molecular targets for selective breeding programs focused on heat tolerance. However, validation of these markers across different commercial breeds and production systems is required to confirm their universal applicability.

6.2.2 Both synthetic and phytogenic antioxidants effectively mitigate HS, presenting viable dietary strategies for the commercial poultry industry. Future research should focus on developing practical on-farm tests for assessing HS susceptibility based on identified biomarkers. Additionally, large-scale field trials are necessary to validate the effectiveness of these strategies across diverse management systems and environmental conditions.