



REVIEW

# Insights into the molecular mechanism of glucose metabolism regulation under stress in chicken skeletal muscle tissues



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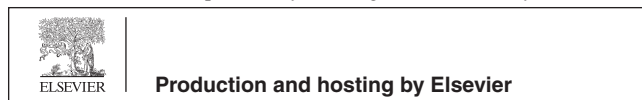
**Abstract** As substantial progress has been achieved in modern poultry production with large-scale and intensive feeding and farming in recent years, stress becomes a vital factor affecting chicken growth, development, and production yield, especially the quality and quantity of skeletal muscle mass. The review was aimed to outline and understand the stress-related genetic regulatory mechanism, which significantly affects glucose metabolism regulation in chicken skeletal muscle tissues. Progress in current studies was summarized relevant to the molecular mechanism and regulatory pathways of glucose metabolism regulation under stress in chicken skeletal muscle tissues. Particularly, the elucidation of those concerned pathways promoted by insulin and insulin receptors would give key clues to the understanding of biological processes of stress response and glucose metabolism regulation under stress, as well as their later effects on chicken muscle development.

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## 1. Introduction

The objective of modern chicken production with intensive breeding and farming is to excessively pursue high growth rate and efficient feed conversion and production yield, which makes broilers and layers more and more sensitive to the external environmental stressors. Many stressors, including temperature, humidity, crowd, immunization, and transportation, are frequently observed during the processes of chicken feeding and farming. Most of these stressors could induce acute stress responses and further reduce the production performance, health and welfare, quality and quantity of meat products in the chicken population. Some stressors could increase their susceptibilities to diseases, resulting in low daily gains and feed conversion rates. Therefore, it is of importance to investigate the production mode of stress response, the behavioral and physiological adaptation, and the mechanism of energy metabolism equilibrium in chicken skeletal muscle tissues under stress.

The concepts of stress were primly proposed by Hans Selye and Harris in the 1940s, originally referring to the sum of non-specific responses of animals to various stimulations (Chrousos and Gold, 1992; Szabo, 1998; McGrowder and Brown, 2007; Tache and Brunnhuber, 2008; Chrousos, 2009; Szabo et al., 2012). Chrousos (2009) suggested that stress comprises a long list of potentially adverse forces acting on animals that could be classified as emotional or physical situations. Stressor constantly challenges the dynamic equilibrium of the living organism (i.e. *homeostasis*), which is maintained in complex adverse states as mentioned by many researchers (Tache and Brunnhuber, 2008; Chrousos, 2009; Szabo et al., 2012). In order to find out the underlying regulatory mechanism or pathway in which stress affects avian growth and development, researchers investigated the possible roles of stressor-simulated glucocorticoid in chicken. A number of studies showed that stress simulated by blocking glucose could regulate the function of broiler chicken corticosteroids, and significantly affect their glucose metabolism and muscle development (Duclos et al., 1993; Kono et al., 2005; McGrowder and Brown, 2007). The increasing of glucose transportation across cellular plasma membrane in chicken skeletal muscle tissues by insulin treatment suggested that there existed an alternative mechanism of insulin-responsive glucose transportation (Chrousos and Gold, 1992; McGrowder and Brown, 2007; Szabo et al., 2012). Insulin at a definite level could stimulate glucose uptake by target tissues through specific glucose transporters (GLUTs) (Duclos et al.,

1993; Kono et al., 2005; McGrowder and Brown, 2007). Insulin-dependent glucose uptake in muscle tissues is largely mediated by the translocation of glucose transporter GLUT4 from the intracellular storage site to the plasma membrane. As a final effector of the hypothalamic–pituitary–adrenal axis, glucocorticoid (GC) participates in controlling the whole body homeostasis and stress responses. Meanwhile, AMP-activated protein kinase (AMPK), another important energy regulator and/or sensor, plays a major role in coordinating the energy metabolism equilibrium, especially glucose metabolism regulation (Hardie et al., 1998, 1999, 2003; Hardie, 2007; Leclerc and Rutter, 2004; Xue and Kahn, 2006; Bungo et al., 2011; Doustar et al., 2012). Coupled with Activating Peroxisome Proliferator-Activated Receptors (mainly gamma subtypes), AMPK could affect the glucose uptake mediated by the plasma membrane translocation of glucose transporter GLUT4 in mice C2C12 cells (Kim et al., 2013). As an established model animals similar to mouse, chicken are relatively highlighted in body temperatures, blood sugar and lipid levels, plastic muscle development, and rapid metabolism regulation, which is important for understanding the processes of diabetes and obesity and other pathogenesis (Ngondi Judith et al., 2007; Shankaraiah et al., 2010; Harisa, 2011). This review focuses on chicken cellular glucose metabolism regulation and adaptation mechanism under stress.

## 2. Stress might block chicken skeletal muscle development via glucose metabolism regulation

Animal muscle fibers can be divided into three categories, i.e. slow contraction oxidized (type I), fast-twitch oxidative (type IIA) and fast-twitch glycolytic (type IIB), according to their metabolic and genetic properties (Carver et al., 2001). It was thought that these three muscle fiber types could be transformed into each other under stress through blood metabolism and glycolytic pathways (Zhang et al., 2009). It was reported that stress on chicken could increase their muscle glycogen reservation, glycolysis capabilities, and induce the issues of defective chicken meat, i.e. PSE (*pale, soft, exudative*) (Carver et al., 2001; Urdaneta-Rincon and Leeson, 2002; Gao et al., 2008; Zhang et al., 2009). Moreover, stress might also promote the deposition of fat and intramuscular fat content by the release of glucocorticoids in chick muscle tissues (Wang et al., 2010). Stress by the regulation of hypothalamic–pituitary–adrenal axis prompted high levels of glucocorticoid release (Urdaneta-Rincon and Leeson, 2002; Hatzivassiliou et al.,

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