Acclimatization of chickens to high and low temperatures

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Abstract

It is well known that ambient temperature has a major impact on poultry production. Heat and cold stress can occur at temperatures above and beneath the upper and lower critical temperatures, respectively. In the case of heat exposure, feed consumption decreases, resulting in highly detrimental effects on growth of broilers and egg production and shell quality in laying. Exposure to low temperatures results in increased feed consumption, but also results in decreased performance in broilers and laying hens. Thus, both may lead to economic losses and alter animal welfare. Different strategies have been studied in order to reduce the detrimental effects of heat and cold stress. Temperature acclimatization during embryogenesis has received experimental attention as an effective way. This phenomenon refers to chickens subjected to higher or lower incubation temperatures that may acquire tolerance to higher or lower temperatures during postnatal life, respectively. This hypothesis is based on the assumptions that: 1. Temperature experienced during embryonic development may influence physiological responses to the environment and 2. It may have positive effects by improving thermotolerance. Previous studies showed that temperatures during critical developmental phases that affect gene expression have persistent effects on thermal acclimatization of chickens. This critical period is linked to the development and maturation of the hypothalamus-pituitary-thyroid axis and hypothalamus-pituitary-adrenal axis. The mechanisms involved in thermotolerance acquisition include changes in pathways regulating energy metabolism resulting in a change in heat production, modifications in stress markers, changes in anti-oxidant pathways and membrane alterations. The aim of this paper is to review 1. Whether and how temperatures experienced during embryogenesis affects temperature tolerance of broilers and laying hens and 2. The physiological mechanisms underlying adaptive temperature response.

Keywords*:* ambient temperature, temperature acclimatization, adaptive response, chickens, incubation

Introduction

It is well known that ambient temperature is one of the major factors influencing growth and egg production in poultry. Chickens have the ability to maintain their body temperature and performance within certain limits. At optimum temperatures, they convert the feed to muscle or egg production

efficiently. Today's genetically selected broiler and layer strains have limited abilities to tolerate extreme ambient temperatures. The effects of high and low ambient temperatures on performance of broilers and laying hens have been extensively studied (Yalcin et al., 1997; Sato et al., 2002; Mashaly et al., 2004; Cahaner et al., 2008; Mack et al., 2013). At high temperatures, chickens decrease feed intake to maintain the balance between heat production and heat loss. Therefore, high temperatures decrease body weight, increase market age in broilers and decrease egg production and reduce shell quality in layers. Chickens increase feed intake at low temperatures to maintain essential body functions and body temperatures resulting in less energy available for growth and egg production.

The exposure of chickens to high and low temperatures induces several physiological adaptive responses. They includes changes in blood acid-base status and thyroid hormones, increases stress parameters such as plasma corticosterone hormone concentration and heterophil to lymphocyte (H:L) ratio, and diminish antioxidant status by increasing malondialdehyde (MDA) decreasing glutathione peroxidase, catalase and superoxide dismutase activities (Yalcin et al., 2003; Lin et al., 2006).

Reducing detrimental effects of high and low temperatures in chickens requires multidisciplinary approaches. There are many housing and equipment systems, management and nutritional programs to improve comfort and performance and to reduce economic losses under high and low temperatures. Acclimatization to high or low temperatures is a promising approach for enhancing temperature tolerance of chickens. This approach is based on the assumption that incubation temperature can be used to affect the development of thermoregulatory mechanisms during prenatal ontogeny by inducing alterations in cellular properties. This review addresses: 1. How thermal acclimatization during embryogenesis affects embryo development 2. Acclimatization to high and low temperatures and temperature tolerance of broilers and laying hens in later life and 3. The physiological mechanisms underlie adaptive temperature response

Embryo development and thermal exposure during incubation

It was hypothesized that exposing embryos to higher or lower incubation temperatures could induce adaptive thermoregulatory reactions to high (Yahav et al., 2004 a, b, Collin et al.., 2007; Yalçın et al., 2008a; Loyau et al., 2013) or low (Tzschentke and Basta, 2002, Shinder et al., 2011; Yalcin et al., 2012) ambient temperatures, respectively, probably via epigenetic adaptation processes. The development of thermoregulatory mechanisms begins during the prenatal stage. Apart from being a metabolic hormone, thyroid hormone is involved in the thermoregulation process with the thyroid gland under the control of the hypothalamus-pituitary axis (McNabb and Darras, 2015). During embryonic development the hypothalamus-pituitary-thyroid axis becomes functionally mature about embryonic d 10.5-11.5, a time in embryonic development when the blood thyroid hormone concentrations begin to increase. Higher thyroid hormones concentrations at embryonic d 13 (T_3) or 14 (T_4) than embryonic d 9 and 11 indicate that circulating thyroid hormones in the embryo are principally derived from the embryonic thyroid

gland, under the control of the hypothalamus-pituitary axis, by the time of somatotroph differentiation (Lui and Porter, 2004). Circulating corticosterone levels also increase around embryonic d 14 (Liu and Porter, 2004). Development of hypothalamus- pituitary -adrenal axis is involved in in stress responses. Therefore, embryonic d 7 to 18 is a critical period for the development of hypothalamus-pituitary-thyroid and hypothalamus-pituitary-adrenal axes (Yalcin et al. 2008a, Piestun et al., 2008). During this sensitive period, embryo temperatures vary with changes in incubation temperature. Loh et al. (2004) measuring allantoic fluid temperatures in embryos at high incubation temperatures found 1-2.5°C higher temperatures than incubation temperature and concluded that allantoic fluid temperatures depended on incubation temperatures. Thus temperature regulation has an influence on the thermoregulatory system via the control of neuronal hypothalamic thermosensitivity and physiological systems to maintain body temperatures.

Acclimatization to high temperatures in broilers

Several studies have investigated the effects of higher incubation temperatures (38.5, 39.5 or 41 °C) for different durations (3, 6, 12 or 24 h per d) during different periods (from 10 to 18 d, from 16 to 18 d or from 7 to 16 d) of incubation. In most cases embryos exposed to cycling higher incubation temperatures resulted similar or higher chick weight and hatchability (Yahav et al, 2004a; Collin et al., 2007; Yalcin et al., 2008a; Loyau et al., 2013). Cyclic higher temperatures between E 10 and 18 at 38.5 °C for 6h/d reduced embryo weight at E 14 and then accelerated growth which was reflected in chick weight being almost 2.2 g heavier. Cyclic higher incubation temperatures increased incubation duration which was associated with lower CO_2 pressure at IP stage and lower blood glucose levels at hatch (Yalcin et al., 2008a). It can be assumed that those embryos used more hepatic glycogen before hatching. Cyclic 38.5 °C from 10 to 18 d of incubation 6 h/d increased blood T_3 concentrations at hatch, however, after exposing 4 h at 32 °C remarkably reduced T_3 levels. Low levels of T_3 concentration persisted up to 42 d under heat stress conditions indicating an alteration of the set-point of the hypothalamus-pituitary-thyroid axis. Moreover, cyclic high incubation temperatures affected blood metabolites. Lower uric acid, triglycerides, and corticosterone levels and up to 8% less abdominal fat with no effect on meat quality were observed for acclimated broilers when exposed to heat stress than their controls (Yalcin et al., 2008b; Piestun et al., 2008; Loyau et al., 2013). Loyau et al. (2013) reported a limited stress response for acclimated broilers during heat challenge compared with control as a sign of improved adaptive capacities. Chickens having already experienced heat exposure during incubation presented a lower stimulation of energy metabolism in the liver and breast muscle (Loyau et al., 2014a), and an amplified response in terms of differential gene expression during a heat challenge, especially for genes regulating energy metabolism, stress response, vascularization and epigenetic mechanisms (Loyau et al., 2016). All these results indicate a carry-over effect of prenatal temperatures on physiology of broilers during postnatal stage.

Acclimatization to low temperatures in broilers

Similar to acclimatization to higher temperatures, lower incubation temperatures during the critical periods of incubation can enhance resistance of broilers to low temperatures. Shinder et al. (2009) tested 15 °C for 30 or 60 min at 18 and 19 d of incubation and observed no effect on hatchability, increased chick weight, and improved ability to withstand low temperatures. Yalcin et al. (2012) exposed embryos to 36.5 °C for 6 h/d from 10 to 18 d of incubation. Slower embryonic growth between E 10 and 14 was compensated with a 4 h delay in hatching process. A chronic decrease of incubation temperature (from E18 to 21 at 34 °C) resulted in 2 day delay in hatch (Loh et al., 2004). Broilers from older breeders exposed to cyclic low incubation temperatures were more resistant to low temperatures from 21 to 42 d than those from young breeders. This result was associated with fatty acid uptakes of embryos that was affected by low incubation temperatures: lower levels of 20:4n-6 and 22:6n-3 yolk fatty acids were transferred to embryo and accumulation of liver18:1n-9 was lower, thus lower levels of liver SFA/PUFA compared to control incubation (Yalcin et al. 2012). An increase in the proportion of unsaturated to saturated fatty acids (SFA) is an indicator of modifications of membrane lipid composition at lower temperatures and may be related to a prenatal adaptation to low temperatures. Moreover, long-term effects of lower incubation temperatures influenced the expression of the avian UCP3 which is involved in fatty acid metabolism and mitochondrial antioxidant defense (Abe et al., 2006; Collin et al., 2009). Our unpublished results showed that broilers exposed to lower incubation temperatures had higher levels of liver oleic acid which upregulates UCP3 gene expression. Also, there was an increase in liver catalase activity which is essential for antioxidant defense systems (Loyau et al., 2014b).

Acclimatization and laying hens

Higher or lower incubation temperatures may have consequences on adaptation to higher or lower ambient temperatures in egg-type poultry. Glatz (1997) reported that constant 0.5–2 °C higher incubation temperatures between E 10 and 21 resulted in lower body weights at 18 wk and smaller eggs. Walstra et al. (2010) reported that 40 °C for 4h/d between E 14 and 18 reduced the incubation duration in Lohmann browns, without affecting hatchability or chick weight. Also those chicks responded to higher environmental temperatures only until 8 d of age. Bovans brown embryos exposed to 1.1 °C higher incubation temperature either between E 0 and 5 d or E 5 and 18 d had a more mature pattern of glucocorticoid release at 23 d when exposed to high ambient temperature (Wilsterman et al., 2015). Kamanlı et al. (2015) exposing brown laying hens embryos (ATAKS) to 36.5 C (low) or 38.5 C (high) temperatures for 6h/d from E 10 to 18 found no effect on the age of sexual maturity, egg production and egg weight. When those hens were exposed to high or low ambient temperatures during the laying period, lower and higher T_4 levels were observed, respectively, suggesting changes in thyroid function. They concluded that not higher but lower incubation temperatures could be protective against the oxidative damage in laying hens under cold ambient temperatures.

In conclusion

Temperature manipulations during critical periods in embryonic development induce various physiological affects including positive impacts in the response of meat and egg type chickens to temperature stress post hatch.

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