Progressive Alteration to Core Temperature, Respiration and Blood Acid-Base Balance in Broiler Chickens Exposed to Acute Heat Stress

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Compared to studies on the cockerel and the domestic laying hen, little information is available concerning the progressive alteration to acid-base balance in broiler chickens exposed to acute heat stress. The primary objective of this study was to determine the time course of changes to core temperature, respiratory rate and blood acid-base parameters of broiler chickens during an episode of acute heat exposure (36 or 38°C). Acute exposure of chickens to 36 and 38°C resulted in significant increases in core temperature, with maximum values reaching 44 and 46°C, respectively. In the first 30 min when rectal temperature increased significantly in the 38°C heat-exposed group, a clear and significant concomitant increase in blood pH was also identified. Conversely, pCO₂ and bicarbonate concentration in the heat-exposed animals had decreased significantly 60 min after the onset of exposure to heat, but not at the 30 min time point. In contrast to previous findings of the time course of changes in arterial pH and pCO₂ of hens exposed to acute heat stress, there was evidence here of a time lag for pCO₂ to decrease after the onset of heat exposure and no compensation for the blood alkalosis at the 90- and 120-min time points after heat exposure. These findings imply the absence of a mechanism for pH compensation in broiler chickens.

Key words: acid-base balance, broiler chicken, core temperature, heat stress, respiratory alkalosis

Introduction

Permanent and acute heat exposure affects metabolic processes in humans and animals. Ambient temperatures exceeding the thermoneutral range lead to an elevated core temperature, followed by a number of responses which result in the neutralization of metabolic changes on one hand, and the reduction of body temperature on the other (Bogin et al., 1996). In the poultry industries of the world, tremendous economic losses occur every year because of mortality and decreased production due to high environmental temperatures. Because energy sources for cooling are expensive worldwide and unavailable in much of the undeveloped world, it would be advantageous to not
only develop heat-tolerant strains of poultry (Bowen and Washburn, 1984), but also the nutritional treatments that promote heat tolerance in otherwise heat-intolerant birds.

Birds rely primarily on respiratory evaporative cooling to maintain thermal homeostasis when exposed to high environmental temperatures (Calder and Schmit-Neilsen, 1966, 1967). Many researchers have studied thermal polypneic responses in the domestic laying hen and cockerel: increased ventilation (evaporative cooling) produces an acid-base disturbance (respiratory alkalosis) that is characterized by a dramatic lowering of blood $\text{CO}_2$ and $\text{H}^+$ concentrations in response to increased ambient temperatures (Linsley and Burger, 1964; Darre et al., 1980; Mather et al., 1980; El-Hadi and Sykes, 1982; Odom et al., 1982, 1986). In cases of prolonged exposure to heat, growth rate is hindered (Bottje and Harrison, 1985). Compared to studies of the domestic laying hen and cockerel, little information is available on progressive alterations to acid-base balance in broiler chickens exposed to acute heat stress, even though blood $\text{pH}$, $\text{CO}_2$ and $\text{HCO}_3^-$ of broiler chicks exposed to chronic heat stress have been investigated (Teeter et al., 1985), and many studies on the interrelationship between resistance to acute heat stress and nutritional status including fasting were carried out (Garlich and McCormick, 1981; Raup and Bottje, 1990; Tabiri et al., 2000).

In order to better understand physiological events that take place in heat-stressed broiler chickens, we followed for 2 h the time course of core temperature, respiratory rate and the blood acid-base balance as well as plasma osmolality after exposure of birds to temperatures of 36°C or 38°C. Such information is fundamental for developing the nutritional manipulation to heat-stressed birds. We also examined plasma potassium concentrations to clarify whether an increased $K^+$ concentration was associated with the heat stress-induced death of broiler chickens, given the deleterious effect of elevated potassium concentration on cardiac function (Gutierrez et al., 1996).

**Materials and Methods**

Male chicks (Arbor Acres/Cobb) were obtained from a commercial hatchery (Economic Federation of Agricultural Co-operatives, Miyagi, Japan) at 1 d of age, housed in electrically-heated batteries, and provided with water and commercial starter diet *ad libitum* for 14 d. Then, they were selected from a two fold larger population to obtain uniform body weight, kept in a wirebottomed cage under conditions of controlled temperature (24±1°C) and continuous light for 6–7 d. All experiments were performed in accordance with institutional guidelines concerning animal use.

In the first series of experiments, twenty-seven chickens (21-d-old, 687±35 g) were used for assessing heat-induced changes to core temperature and respiratory rate. Two groups of nine chickens were transferred to either 36 or 38°C for 120 min while the remainders were maintained at 24°C. In the two sets of the second series of experiments, two groups of 4–6 chickens (first set: 20-d-old, 688±37 g and second set: 21-d-old, 679±21 g) were kept at either 24 or 36°C for determination of the time course of the blood acid-base balance as well as plasma osmolality, and potassium and sodium concentrations. In the third series of experiments, two groups of twelve randomly assigned chickens (20-d-old, 634±30 g) were maintained at either 24 or 36°C for 60 or
90 min, and then subjected to hematocrit value and plasma volume measurements. Six chickens were used to determine zero-time baseline. All birds were deprived of feed but given free access to water during the heat exposure. The relative humidity was kept as close as possible to a constant 55%.

Core temperature and respiratory rate were measured at 30 min intervals throughout the 120 min of acute heat challenge. Blood samples were taken via a wing vein at 30 min intervals until 120 min after completion of the heat exposure phase. These samples were evaluated for pH, pCO$_2$ and bicarbonate concentration. As described in our previous paper (Toyomizu et al., 1999), heparinized syringes containing the blood samples were placed on ice for no longer than 5 min prior to analysis using an Acid-Base Laboratory Blood Gas Analyzer (Radiometer Medical A/S, Copenhagen, Denmark), calibrated assuming a body temperature of 42°C (Ruiz-Lopez and Austic, 1993).

For measurements of plasma osmolality, potassium and sodium, blood samples were also taken via a wing vein at the same intervals described above, and centrifuged at 7,000 g for 10 min. Aliquots of plasma were applied to an osmometer (Model L3, Advanced Inc., USA) and flame spectrophotometer (Hitachi, Model 205DT). Blood volume was determined by Gregersen’s method (1944) using T-1824 dye (Evans Blue). Briefly, 10 min after a single injection of dye (20 mg/body weight) via one wing vein, a blood sample was drawn via a wing vein on the opposite wing. The plasma concentration of the dye was measured spectrophotometrically at 620 nm for calculation of the plasma volume. For hematocrit determination, aliquots of blood were obtained in heparinised capillary tubes and centrifuged in a microhematocrit centrifuge for 5 min at 7,000 g. Total blood volume was then calculated from the following equation:

\[
\text{Total blood volume (c.c.)} = \frac{\text{plasma volume (c.c.)}}{(1 - \text{hematocrit})}
\]

Data were analyzed using the Statistical Analysis System (SAS Institute, 1985). Means within a group were compared using Duncan’s least significance multiple-range test for the results of plasma volume and hematocrit value, and Dunnett’s procedure for the results from all the other analyses. Differences between groups, such as comparisons of results from heat-exposed and control groups were analyzed by Student’s t tests for unpaired data. All data are expressed as the mean value ± standard deviation (SD). Differences were considered significant at the level of $P < 0.05$.

**Results**

Figure 1A shows that a significant increase in core temperature occurred in chickens exposed to temperatures of 36 or 38°C. The magnitude of the heat-induced elevation in rectal temperature depended on the chamber temperature, with approximate maximum values of 44 and 46°C measured for chamber temperatures of 36 and 38°C, respectively. The mean respiratory rate increased from a pre-heat exposure value of 61 br/min to a maximum of 261 br/min after 60 min of exposure to 38°C. Chickens exhibited slow and deep breathing after 90 min of heat exposure (Fig. 1B).

Figure 2 shows the progressive alterations to blood pH, pCO$_2$ levels and bicarbonate concentrations in control chickens and chickens exposed to 38°C for 120 min. While values for the respective parameters in control chickens remained near basal levels
throughout the experiment, blood pH (Fig. 2 A) in the heat-exposed group was found to be significantly increased within the first 30 min when rectal temperature also increased significantly. Values for pCO₂ (Fig. 2 B) and bicarbonate concentration (Fig. 2 C) in the heat-exposed animals decreased significantly compared to control at 60 min but not at 30 min, and thereafter gradually stabilized and remained constant during the period from 90- to 120-min.

Further analysis of blood samples showed that plasma osmolality of 38°C-exposed chickens increased in the second hour of heat treatment compared to the control group, and that plasma potassium concentration remained increased throughout the experiment (Fig. 3). The plasma sodium concentration on the other hand remained unchanged in both groups. No differences between control and heat-treated groups were observed in relation to plasma and blood volumes after 60 or 90 min of heat-exposure, while hematocrit was slightly but significantly decreased by the heat treatment (Fig. 4).

Discussion

The results of this study support in general the findings of Odom et al. (1986) concerning the time course of changes in arterial pH and pCO₂ of hens exposed to acute heat stress. This was except for the presence here of a lag time for pCO₂ to decrease in the period immediately after heat exposure, along with the absence here of compensation for the blood alkalosis at the 90- and 120-min time points after heat exposure. Differences between the two studies in relation to pCO₂ in the first period may be due to the rapid enhancement in rectal temperature observed in the broiler chicken used.
here without a concomitant increase in CO₂ exchange in the lung. Then, the following reductive reaction of HbO₂⁻⁻ to Hb⁻⁻ might cause a rise in pH considering that oxygenated blood gives up O₂ to the cells (Valtin and Gennari, 1987). Alternatively, excretion of [HCO₃⁻] might be decreased due to the dysfunction of the kidney, causing a rise in pH according to the Henderson-Hasselbalch equation (pH = pK’ + log ([HCO₃⁻]/constant × pCO₂)). In regard with the compensation for alkalosis, it was reported that respiratory alkalosis in domestic hens occurred 1 h after the onset of heat exposure (35°C), but that approximately 1 h later there was a concomitant decline in blood pH as plasma lactate and pyruvate concentrations increased (Odom et al., 1986). This compensation for the blood alkalosis in the hen is in accordance with the findings of Halles et al. (1970) who showed that the concentration of HCO₃⁻ in the cerebrospinal fluid of sheep decreased markedly during advanced stages of heat stress (rectal temperature >40.5°C). In marked contrast to the compensation for blood alkalosis in hens or sheep, our results from broiler chickens showed that blood pH remained elevated throughout the experimental period since the depression in [HCO₃⁻] disappeared during advanced stages of heat stress as shown by remaining constant values during the period from 90- to 120-min, again implying a lack of pH compensation in the

![Graphs showing pH, pCO₂, and bicarbonate concentrations.](image)
broiler chickens.

This implication does, however, require some discussion. To better understand the sequence of physiological events accompanied by increases in body temperature of broiler chickens, the relative changes in respiratory rate, blood pH, pCO$_2$ and bicarbon-
ate concentration vs. core temperature have been depicted in Figure 5. Acute heat-exposed chickens showed a rapid increase in respiratory rate as core temperature began to rise. The respiratory rate then declined with continuing increases in core temperature. Blood pH rapidly increased and remained elevated. The decrease in blood pCO$_2$ could be described by a sigmoidal curve with inflection point in the range from 42 to 43°C, and a plateau reflecting the decline in respiratory rate at a core temperature of about 43°C. In contrast to our observations, Mather et al. (1980) reported that cockerels exposed to 45°C displayed increased respiratory rates for colon temperatures up to 42°C, and then breathed progressively slower and deeper at 43.5°C. Further, their data demonstrated that pCO$_2$ decreased continuously for increases in colon temperatures up to 43°C, then this was followed by a slight decline in pCO$_2$, while pH rose continuously for colon temperatures up to 42°C and declined slightly thereafter. Taken together, the results from acute heat stressed broiler chickens and leghorn chickens suggest that the core temperatures of the two types of chicken undergo similar changes (given the turning point of respiratory rate), but that pattern of changes in pH and pCO$_2$ are different. Therefore, one could postulate that the rapid increase in blood pH and the lack of pH compensation in broiler chickens may be due to the fact that [HCO$_3^-$] increased slightly at the beginning of heat exposure and thereafter declined slightly, but reached a constant level. Birds are indeed as efficient at conserving HCO$_3^-$ as are mammals, with almost complete reabsorption of filtered bicarbonate during periods of acidosis (Anderson, 1967). Of note here is the observation that plasma potassium concentrations in broiler chickens were enhanced by the heat-exposure. In contrast to this finding, it was reported that plasma potassium levels of Leghorn hens and broiler chickens were not increased when birds were exposed to temperatures of 38°C for 140 min, or to a stepwise
increase in chamber temperature from 29 to 39°C at a rate of 4°C/hr (Ait-Boulahsen et al., 1989; Koelkebeck and Odom, 1995). These discrepancies may be due not only to different ages and strains of birds used, but also to varying conditions of temperature exposure: exposure to very high temperature 45–49°C resulted in an increase in plasma [K⁺] in chickens (Edens, 1977). Plasma osmolality also changed in a similar manner to that of plasma potassium, whereas plasma sodium concentration was not changed during a 2-h exposure to 38°C (result not shown), meaning that the heat-induced changes in plasma electrolytes could not be explained by simply invoking a process of hemoconcentration accompanying heat exposure. In fact, plasma volume, which was calculated using the Evans blue dye method was not changed by acute heat exposure for 60 min or 90 min, though hematocrit values were slightly, but significantly decreased in the heat treatment group compared to control. Thus, changes in plasma volume can not be used to account for the elevated plasma potassium noted in this study. The increase in plasma potassium and reduction in hematocrit value are suggestive of cell damage with subsequent leakage of K⁺ out of cells. These responses will be clarified further in future studies given the deleterious effects of potassium on cardiac function (Gutierrez et al., 1996).

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References


Gregersen MI. A practical method for the determination of blood volume with the dye T-1824. Journal of Laboratory and Clinical Medicine, 29: 1266–1286. 1944.


