CHANGES ASSOCIATED WITH FASTING AND ACUTE HEAT STRESS IN BODY TEMPERATURE, BLOOD ACID-BASE BALANCE AND ELECTROLYTES OF JAPANESE QUAIL

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ABSTRACT

The study was performed to examine the effect of fasting on quail chicks exposed to acute heat stress. Fasting for 24 and 48 hours significantly reduced rectal temperature (Tr) ($P<0.05$) compared with nonfasted group. The blood pH did not change by fasting but there was a significant fall in blood HCO_3- and pCO2 levels in both the fasted groups for 24 and 48 hr relative to the other groups.

In all groups, pH significantly increased ($P<0.05$) with the increase in Tr induced acute heat exposure but the rectal temperature in nonfasted group was significantly higher than in the fasted group ($P<0.05$). Although acute heat stress caused a sharp decline in blood pCO2 ($P<0.05$) accompained by a fall in blood HCO3 in all groups, the increase in pH and fall in pCO2 and HCO3 were more marked in nonfasted group. Heat induced haemodilution was more pronounced in fasted birds compared with those in nonfasted groups.

By resisting these results, fasting enhances the physiological adaptation of quails in response to acute heat stress, and the manipulations of diet might increase tolerance of quails to heat stress.

INTRODUCTION

Environmental temperature above the thermoneutral level decreases feed intake, weight gain and resistance to diseases, and increases the mortality rate in broiler chicks (Teeter *et al.*, 1985; Kutlu, 1996). These negative effects of heat stress have been attributed to increased plasma cortisol level resulting in excess catabolism (Kutlu, 1996), decreased T3 (triiodothyronine) concentration (Bobek *et al.*, 1980), acid-base imbalance (Bottje and Harrison, 1985) and fall in ascorbic acid level (Kutlu, 1996). Studies in acute heat-stressed young chicks (McCormick *et al.*, 1979; Garlich and Mc-Cormick, 1981) have indicated that fasting markedly increases the survival time and that the metabolic shift from carbohydrate to fat catabolism may in part account for this effect. Ait-Boulahsen *et al.* (1989) showed that the tolerance of fasted chicken to acute heat stress increased and fasting reduced the rates of heat-induced changes in blood acid-base and electrolyte status. On the other hand, manipulation of the electrolyte composition of the diet or drinking water alleviated some of the adverse effects of heat stress on blood acid-base and electrolyte status, and thus enhanced the growth rate and livability (Teeter *et al.*, 1985; Bottje and Harrison 1986; Smith and Teeter, 1987).

Thermal polypnae associated with heat loss in birds reduces blood carbon dioxide partial pressure and H+ ion concentration and results in respiratory alkalosis (Odom, 1982; Teeter *et al.*, 1985). Fasting is also known to alter blood acid-base and electrolyte balance in hens (McCormick *et al.*, 1979; Ait-Boulahsen *et al.*, 1989) as in mammals (Cizek *et al.*, 1977).

The aim of this work was to evaluate the effects of fasting on heat-induced changes in blood acid-base balance and electrolyte status in Japanese quail chicks, because the changes associated with fasting and heat stress in quails may differ from those in hens.

MATERIALS AND METHODS

Sixty male and female Japanese quails, 5-6 week old, were used in this study. All the chicks were weighed and divided equally into three groups in order to minimize the effects of body weight. Twenty chicks in each group were housed in wire cages within a thermostatically and humidistatically controlled chamber under continuous lighting.

Group I was used as a non-fasted group (NF). In this group, water and feed were present during the first stage of the study. Group II (24F) and group III (48F) were fasted for 24 and 48 hr, respectively. At the end of the fasting period, rectal temperature (Tr) was recorded. Then venous blood samples were taken anaerobically and analysed for pH, HCO3, pCO2, Na+, K+ levels and PCV (Packed Cell Volume), Hb (Haemoglobine) amount and RBC (Red Blood Cell)

count from 10 chicks in each group.

At the second stage of the study (acute heat exposure), birds of all groups were exposed to acute heat stress for 150 min. Heat exposure was performed by increasing environmental temperature from 20°C to 42°C at a rate 08°C/hr and heat exposure period lasted for 150 min at 42°C. During this period feed and water were not available for all groups. At the end of the acute heat exposure the same parameters were determined in each group (10 chicks from each group).

Statistical analysis

Differences between group means were evaluated by student's unpaired t-test (Steel and Torrie, 1982).

RESULTS AND DISCUSSION

Fasting for 24 and 48 hr significantly ($P<0.05$) reduced Tr compared to NF group (Table 1). This decrease in Tr may be a result of the fall in plasma chalorigenic hormones such as T3 (Harvey and Klandorf, 1983).

Although the blood pH values were not affected by fasting, there was a significant decrease ($P<0.05$) in HCO3- and pCO2 levels in both 24F and 48F groups (Table 1). Despite the unchanged blood pH values, fasting acidosis was evidenced by the decline in blood HCO3- and possibly by a secondary respiratory compensation. This compensated acidosis may be partially due to the elevation in plasma total ketones by fasting (Brody *et al.*, 1978; Ait-Boulahsen *et al.*, 1989). The plasma Na+ levels in 24F and 48F groups increased slightly but did not differe from those in NF group (Table 1). This finding is similar to the data reported for fasted rabbit (Cizek *et al.*, 1977) and swine (Kornegay *et al.*, 1964) but unlike to that for fasted chicken (Koike *et al.*, 1983; Ait-Boulahsen *et al.*, 1989).

On the other hand, plasma K+ levels in group 48F significantly increased ($P<0.05$) as compared to NF (Table 1). In contrast, the depressed plasma K+ level has been reported in fasted chicken (Koike *et al.*, 1983; Ait-Boulahsen *et al.*, 1989). The above workers have not explained reason of the depression in K+ level in fasted chicken, but it is well known that plasma K+ increase in metabolic acidosis is a result of renal compensation of acidosis which increases excretion of H+ versus K+.

The Hb, RBC and PCV increased in 24F and 48F groups compared to NF (Table 1). The haemoconcentration observed particularly in 48F agrees with the data reported for fasted chicken (Koike *et al.*, 1983; Ait-Boulahsen *et al.*, 1989) and mammals (Kornegay *et al.*, 1964).

Table 1: Effect of fasting before and during acute heat exposure on body temperature, acid-base balance, PCV, RBC, Hb and some plasma electrolytes in Japanese quails (n = 10).

Parameters	Fasting (at 22°C)			Fasting + acute heat stress (150 mn. at 42°C		
	NF	24F	48F	NF	24F	48F
Tr(°C)	41.10 ± 0.07Ab	40.60 ± 0.08Bb	40.20 ± 0.08Bb	45.20 ± 0.08Aa	43.90 ± 0.10Ba	43.60 ± 0.09Ba
pH	7.41 ± 0.011Ab	7.39 ± 0.008Ab	7.40 ± 0.006Ab	7.55 ± 0.019Aa	7.50 ± 0.010Ba	7.47 ± 0.018Ba
HCO_3^- (mEq/L)	19.30 ± 1.11Aa	15.00 ± 0.98Ba	13.40 ± 0.86Ba	12.20 ± 0.90Ab	10.80 ± 1.20Ab	11.30 ± 0.90Ab
pCO_2 (mmHg)	33.30 ± 1.71Aa	26.50 ± 0.98Ba	24.10 ± 0.91	19.10 ± 1.30Ab	16.30 ± 1.40Ab	16.50 ± 1.10Ab
Na^+ (mEq/L)	142.60 ± 0.73Aa	144.50 ± 0.60Aa	145.10 ± 0.59Aa	143.50 ± 0.76Aa	145.90 ± 0.89Aa	145.80 ± 0.62Aa
K^+ (mEq/L)	5.08 ± 0.07Aa	5.24 ± 0.06Aa	5.36 ± 0.08Ba	4.87 ± 0.06Bb	5.01 ± 0.09Ab	5.18 ± 0.08Aa
PCV (%)	34.90 ± 1.02Aa	36.40 ± 1.05Ba	39.10 ± 1.24Ba	33.80 ± 1.31Aa	32.20 ± 1.22Ab	35.60 ± 1.14Aa
RBC ($x10^6/mm^3$)	4.36 ± 0.12Aa	4.48 ± 0.13Ba	4.88 ± 0.19Ba	4.26 ± 0.16Aa	4.09 ± 0.11Ab	4.39 ± 0.12Aa
Hb (g/dl)	11.59 ± 0.51Aa	12.40 ± 0.54Ba	13.50 ± 0.68Ba	11.47 ± 0.51Aa	10.43 ± 0.43Ab	12.04 ± 0.62Aa

^{A,B}A parameter with unlike superscripts differ from those in the identical group which in the other period ($P<0.05$).
^{a,b}A parameter with unlike superscripts differ from those in the other groups within the same period ($P<0.05$)

Acute heat exposure (150 min. at 42°C) resulted in a significant elevation ($P<0.05$) in Tr in all groups compared with thermoneutral period (Table 1). Tr in NF (45.2°C) was significantly higher ($P<0.05$) than that in fasted groups for 24 (43.9°C) and 48 hr (43.6°C). At the end of the heat exposure, pH significantly increased in all groups but pH in 24F and 48F was lower relative to NF ($P<0.05$).

There was a sharp decline in blood pCO2, accompanied by a fall in blood HCO3 with the increase in pH in all groups ($P<0.05$). The fall in pCO2 and HCO3, in NF was higher than in the other groups in response to acute heat stress (Table 1). These data reflect the excessiveness of thermal polypnea in NF compared with fasted groups in agreement with the data reported for fasted chicken under acute heat exposure (Ait-Boulahsen *et al.*, 1989).

Although the PCV, Hb and RBC levels decreased in response to heat stress in all groups, the decrease was significant ($P<0.05$) only in 24F group (Table 1). The plasma Na+ levels showed no significant changes induced by the heat stress in all birds. This observation suggested that haemodilution related to acute heat stress was accompanied by unchanged plasma Na+ level and osmolality unlike hens (Ait-Boulahsen *et al.*, 1989) but similar to mammals (Harrison, 1974). The haemodilution observed in this study can be attributed to peripheral vasodilation (Darre and Harrison, 1986; Ait-Boulahsen *et al.*, 1989).

By the end of the acute heat stress, plasma K+ levels decreased in all groups. The decreases in K+ levels of NF and 24F were significant ($P<0.05$) compared with thermoneutral period. The intense reduction in plasma K+ level of NF suggests that respiratory alkalosis was evident, because the highest rectal temperature occurred in NF in response to acute heat stress. This decrease in K^+ has been explained by its entering into intracellular fluid in response to respiratory alkalosis in relation to acute hyperventilation (Deetz and Ringrose, 1976; Smith and Teeter, 1987; Ait-Boulahsen *et al.*, 1989).

A comparison of fasted and nonfasted quail chicks has indicated that acute heat stress increased pH and rectal temperature in nonfasted birds ($P<0.05$). On the other hand, the comparison of 24F and 48F revealed that heat tolerance was substantially improved as the time of fasting was extended, because rectal temperature and pH in 48F (43.6°C, 7.47) were lesser than in 24F (43.9°C, 7.50). Consequently, it may be possible to enhance heat tolerance of chickens to acute heat stress to manipulate the acid-base status, electrolyte contents and amount of diet without fasting.

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