Changes of Blood Gases, Internal Organ Weights and Performance of Broiler Chickens with Cold Induced Ascites

M. Daneshyar, H. Kermanshahi and A.G. Golian
Department of Animal Science, Faculty of Agriculture,
Ferdowsi University of Mashhad, Mashhad, Iran, P.O. Box 91775-1163, Iran

Abstract: An experiment with 250 day-old Ross male broilers was conducted to investigate the differences of blood gas parameters, internal organ weights and perfosrmance of cold induced ascitic and healthy broilers in a 6 week period. These chickens were divided to two groups of 5 replicates each. One group of these chickens was bred in Normal Temperature (NT treatment) but other group was set in Low Temperature (LT treatment) to induce ascites. Body Weight Gain (BWG) and Feed Intake (FI) were measured weekly. Blood gases analyses were measured from day 14 afterwards. Mortality was necropsied daily to determine cause of death. At the end of the experiment, 5 chickens from each replicate were randomly selected, weighed, slaughtered, heart was removed and its parts weight determined. Moreover weights of liver, lung, gut (empty weight) and spleen were assayed. The results of blood gases analyses showed that pH, bicarbonate and pCO_2 were similar for two groups, while pO_2 of LT group was lower ($p \le 0.05$) than that of NT group in weeks 3, 5 and 6. Also O_2 saturation of LT group was lower ($p \le 0.05$) than that of NT in weeks 5 and 6. Despite of some differences between two treatments for internal organ weights such as liver, lung, heart, empty gut and spleen weights, these differences were not significant. FI in first two weeks of age was similar for two groups then FI of LT group began to decrease when compared to NT group and at the ages of 3, 4, 5 weeks and all of the study period (6 weeks) FI was lower ($p \le 0.05$) than that of NT treatment. BWG of LT group at 4th week of age and total period of the study was lower ($p \le 0.05$) than that of NT group. Right Ventricle (RV) and RV to Total Ventricle (TV) ratio of LT treatment was greater ($p \le 0.05$) than that of NT group. Mortality caused by ascites in total period of study for LT treatment was significantly ($p \le 0.05$) higher than that of NT group (8.8 vs 2.4%). Under the conditions of this study it was concluded that inducing ascites by decreasing temperature may impact performance and changes pO_2 and O_2 saturation of broiler chickens.

Key words: Ascites, blood gases, internal organ weights, performance

INTRODUCTION

The incidence of ascites in broilers has become of increasing concern to poultry industry in many areas of the world (Silva *et al.*, 1988). Despite investigations of the syndrome for many years, it is still a condition that inflicts financial loss on poultry farmers around the world (Maxwell and Robertson, 1997). It is estimated that 5% of broilers and 20% of roster birds die of ascites (Swire, 1980; Julian *et al.*, 1986); considering that an estimated 40 billion broilers are produced annually around the world, it is evident that losses due to ascites are significant (Soils de los Santos *et al.*, 2005). It is a severe cause of loss to the broiler industry in many countries, not only due to high rate of mortality, but also due to reduced weight gain and increased condemnations at slaughter (Julian, 1993). A recent survey has placed the global cost at around $1 billion per annum (Maxwell and Robertson, 1997).

Ascites is described as a syndrome caused by severe accumulation of fluids in the body cavity (Bolukabasi *et al.*, 2005). This syndrome characterized mainly by hypoemia, followed by a cascade of events including increased cardiac output; development of pulmonary hypertension; right ventricle hypertrophy; right valve insufficiency; central venous congestion; fluid exudation; mainly to the peritoneal cavity and pericardium and finally death (Olkowski and Classen, 1998). In hypoxic situations, it has been postulated that lowered oxygen tensions reduces blood flow capacity and in combination with the polycythemia, ascites development is hastened (Olander *et al.*, 1967). Right Ventricle to Total Ventricle (RV/TV) ratio, hemoglobin, hematocrit, blood gases and specific

Corresponding Author: H. Kermanshahi, Department of Animal Science, Faculty of Agriculture,
Ferdowsi University of Mashhad, Mashhad, Iran, P.O. Box 91775-1163, Iran

clinical chemistries can be used to determine the ascites status of a bird before gross lesions are apparent. An RV/TV ratio of more than 0.27 is considered an accurate measure of the onset of ascites (Huchzermeyer and Deruyck, 1986; Peacock *et al.*, 1988). Therefore heart, lungs and liver are the main internal organ challenged in this syndrome, Spleen weight is a general indicator of stress (Silversides *et al.*, 1997). Moreover, it seems that gut may be affected by this syndrome since Gastrointestinal Tract (GIT) is a metabolically active system that has considerable nutrient and oxygen requirements. Although the total oxygen demand of the gut is not known for chickens, the pig GIT uses 25% of total oxygen consumption and represents only 5% of its total body weight (Yen *et al.*, 1998). The high oxygen demand of the heart and lungs for GIT may explain why feed restriction can reduce ascites incidence in broilers (Balog *et al.*, 2000). So, one aim of this study was evaluate the heart, lungs, liver, gut and spleen weight changes by ascites.

Analysis of pH and blood gases could clarify the acid-base disturbances and help to differentiate between metabolic and respiratory disorders. Gas analysis of venous rather than arterial blood may be more appropriate in determining acid-base disturbances (Hunt and Simkiss, 1967). Scheele *et al.* (2003) observed that ascites-resistant and ascites-sensitive flocks at a young age (11 d), are different in their blood pCO_2 and in pO_2. Balog *et al.* (2003) showed higher blood pCO_2 for ascites susceptible lines than those of resistant lines. Wideman *et al.* (1998) observed the lower percentage saturation of hemoglobin with oxygen for cold induced ascitic males on day 42 in comparison to non ascetic males. Buys *et al.* (1998) demonstrated that progeny of ascites-sensitive broiler lines (incubated under normal conditions) had higher venous pCO_2 at 6 weeks than the progeny of ascites-resistant lines. Olkowski *et al.* (1999) investigated the changes of blood gases of ascitic and healthy birds in 14 and 35 days of age. Since, there are not a comprehensive study about weekly blood gases changes with inducing ascites, another objective of this study is to investigate the weekly and continuously changes of blood gases variables in cold induced ascites in broiler chickens.

MATERIALS AND METHODS

Two hundred fifty day-old Ross male broiler chickens were used in this experiment. Half of the birds were housed in 5 pens ($1*2$ m^2) each 25 and were reared under normal temperature (NT group) up to 4 weeks of age, then were exposed to constant $22 \pm 1C$ until 6 weeks of age (Luger *et al.*, 2001). Another half were housed in a room

Table 1: Composition of experimental diets

Ingredients (%)	Starter (o-21 d)	Grower (21-42 d)
Corn	52.12	59.65
Soybean meal	29.94	27.84
Corn gluten meal	8.87	4.55
Veg. Oil	5.00	4.50
Limestone	1.20	1.32
Dicalcium phosphate	1.74	1.22
Vit. and min. premix[1]	0.50	0.50
Salt	0.47	0.34
DL-methionine	0.10	0.03
L-Lysine	0.06	0.05
Total	100.00	100.00
Calculated analysis		
ME (kcal/kg)	3200.00	3200.00
CP (%)	23.00	20.00
Calcium (%)	1.00	0.90
Ava. Phosphorous (%)	0.45	0.35
Sodium (%)	0.20	0.15
Arg. (%)	1.30	1.19
Met. + Cys. (%)	0.90	0.72
Lys. (%)	1.10	1.00
Try. (%)	0.29	0.26

[1]Supplied per kilogram of diet: Vitamin A, 10000 IU; vitamin D_3,9790 IU; vitamin E, 121 IU; B_{12}, 20 µg; riboflavin, 4.4 mg; calcium pantothenate, 40 mg; niacin, 22 mg; choline, 840 mg; biotin, 30 µg; thiamine, 4 mg; zinc sulphate, 60 mg; manganese oxide, 60 mg

with 5 pens ($1*2$ m^2) each 25 in a low temperature condition (LT group). For inducing ascites, the birds of LT group were raised under 32 and 30°C during weeks 1 and 2, respectively. The temperature lowered to 15°C during week 3 and maintained between 10 and 15 for the rest of study (Igbal *et al.*, 2001). Feed and water provided *ad libitum*. All chickens were supplied with a mash broiler starter diet until 21 d of age and thereafter a mash broiler grower diet (Table 1). BWG and FI were measured weekly. Blood gases analyses from day 14 onwards were performed on pH/Blood Gas Analyzer, ABL50, Radiometer Copenhagen, Denmark. Throughout the study, mortality was recorded daily and all of the chickens died during the experimental period were examined for lesions of heart failure and ascites. At the end of the experiment (week 6), 5 chickens from each replicate (pens) were randomly selected and slaughtered. The heart was removed; the Right Ventricle (RV) was dissected away from the left ventricle and septum. The weights of right and left ventricular were determined separately. Moreover, the weights of liver, lung, gut (empty weight) and spleen were determined. The data were analyzed based on a completely randomized design with two treatments with 5 replicates each using the general linear model procedure of SAS (2002). When treatment means were significant ($p \leq 0.05$), they compared using the Duncan test (Steel and Torrie, 1982).

RESULTS AND DISCUSSION

Performance: FI, BWG and Feed to Gain Ratio (FCR) of NT and LT treatments are shown in Table 2. There were

Table 2: Effect of treatments on feed intake, body weight gain and feed to gain ratio of broiler chickens at different periods

Treatments/ Week	1	2	3	4	5	6	1-6
Feed intake (g)							
NT[1]	107.0	253.0	535.0[a]	826.0[a]	986.0[a]	1305.0	3914.0[a]
LT	108.0	261.0	496.0[b]	746.0[b]	886.0[b]	1185.0	3585.0[b]
±SEM	1.83	2.81	8.98	10.19	25.34	52.41	14.46
P value	0.818	0.071	0.016	0.0005	0.0235	0.144	0.027
Body weight gain (g)							
NT	83.0	159.0	296.0	374.0[a]	452.0	544.0	1908.0[a]
LT	80.0	166.0	308.0	329.0[b]	390.0	484.0	1758.0[b]
±SEM	1.31	3.00	6.00	12.79	20.73	34.98	44.24
P value	0.157	0.127	0.201	0.037	0.068	0.259	0.043
Feed to gain ratio							
NT	1.30	1.6.0	1.81[a]	2.22	2.22	2.43	1.93
LT	1.35	1.58	1.61[b]	2.28	2.31	2.50	1.94
±SEM	0.033	0.037	0.048	0.067	0.16	0.18	0.05
p value	0.280	0.766	0.020	0.532	0.662	0.816	0.882

[1]Treatments are: NT= birds raised in normal temperature, LT= birds raised in low temperature, [a, b] Means in each column with different superscript are significantly different ($p \leq 0.05$)

Table 3: Effect of treatments on relative organ weights (to live body weight, %) in broiler chickens at week 6

Treatments[1]	Liver	Lung	Heart	Empty gut	Spleen
NT	2.24[2]	0.67	0.67	9.10	1.59
LT	2.53	0.66	0.73	9.53	1.85
±SEM	0.980	0.330	0.041	0.388	0.010
p value	0.070	0.880	0.368	0.453	0.119

[1]Treatments are: NT= birds raised in normal temperature, LT= birds raised in low temperature, [2]Means in each column with no superscripts are not significantly different (p>0.05)

Table 4: Effect of treatments on heart parts weight, RV/TV ratio and mortality of broiler chickens at week 6

Treatments[1]	TV (g)	LV (g)	RV (g)	RV/TV	Mortality[2](%)
NT	6.80	5.29	1.06[b]	0.20[b]	2.4[b]
LT	6.36	5.27	1.53[a]	0.29[a]	8.8[a]
±SEM	0.220	0.190	0.040	0.005	1.790
p value	0.068	0.256	0.0005	0.0001	0.035

[a, b] Means in each column with different superscripts are significantly different ($p \leq 0.05$), [1]Treatments are: NT= birds raised in normal temperature, LT= birds raised in low temperature, [2]Mortality due to ascites was diagnosed by post mortem examination.

no significant differences for FI between two treatments at the first two weeks of age, but at weeks 3, 4 and 5, FI of NT group was higher ($p \leq 0.05$) than that of the LT treatment. At the end of week 6, although FI in NT group was higher than that of LT, but this difference was not significant. At the whole period of 6 weeks, FI of NT treatment was also higher ($p \leq 0.05$) than that of LT group. Although BWG for NT treatment was higher than that of LT group, but this variable was significant ($p \leq 0.05$) only at week 4. Also at the whole period of 6 weeks, BWG of NT group was significantly ($p \leq 0.05$) higher than that of LT treatment. FCR was significantly higher ($p \leq 0.05$) at week 3 in NT birds but in other weeks and whole period of study, this variable was not significantly different.

Internal organ weights: Table 3, shows the relative organ weights at week 6. As shown in this table, there were no significant differences between two treatments for the variables. Relative lung in NT treatment and liver, heart, empty gut and spleen weight in LT group were slightly greater, but these differences were not significant between treatments.

Heart part weights, RV/TV ratio and mortality: These variables are show in Table 4. As shown, there were no significant differences for TV and LV for two treatments, but RV and RV/TV ratio of LT treatment was greater ($p \leq 0.05$) than those of NT group. Total mortality due to ascites at the whole period of study for LT treatment was 8.8% when compared to 2.4% for NT treatment and this difference was significant ($p \leq 0.05$).

Blood pH and gases: Blood pH and gases are shown in Table 5. As seen, there were no significant differences between two treatments for pH and HCO_3^-. Although pCO_2 for LT treatment was greater than that of NT group, but these differences were not significant. pO_2 for NT treatment was significantly ($p \leq 0.05$) higher than that of LT group at weeks 3, 5 and 6, but not in other periods. O_2 saturation for NT treatment at all weeks was higher than that of LT group, but this difference was only significant ($p \leq 0.05$) at weeks 5 and 6.

The intensive selection of broilers for maximal body mass has resulted in anatomical and physiological limitations of blood flow through the lungs, with

Table 5: Effect of treatments on mean pH, pCO_2, pO_2, HCO_3^- and O_2 saturation of broiler chickens at different periods

Treatments[1]	Week					
	2	3	4	5	6	2-6
pH						
NT	7.30	7.37	7.42	7.38	7.41	7.37
LT	7.26	7.36	7.40	7.38	7.45	7.37
±SEM	0.032	0.018	0.018	0.015	0.023	0.006
p value	0.356	0.681	0.491	0.877	0.345	0.867
HCO_3^- (nmol/l)						
NT	22.32	23.53	27.70	32.28	27.04	26.57
LT	23.65	24.94	30.14	32.84	30.34	28.38
±SEM	0.995	0.461	0.909	1.750	1.094	0.792
p value	0.372	0.065	0.094	0.826	0.065	0.145
pO_2 (mmHg)						
NT	47.84	69.70[a]	38.80	42.08[a]	49.54[a]	47.32
LT	45.86	58.80[b]	37.22	36.02[b]	35.56[b]	44.87
±SEM	5.170	2.330	3.220	1.670	2.720	2.010
p value	0.793	0.01	0.805	0.033	0.006	0.413
pCO_2 (mmHg)						
NT	51.87	42.08	44.86	55.70	42.72	47.44
LT	55.34	45.12	46.54	56.92	44.38	49.66
±SEM	2.070	2.220	2.370	4.060	3.160	1.970
p value	0.271	0.362	0.63	0.837	0.72	0.451
O_2 saturation (%)						
NT	73.90	73.50	70.12	69.28[a]	64.54[a]	70.27
LT	72.60	72.40	71.24	54.62[b]	50.12[b]	64.20
±SEM	6.440	5.400	5.470	2.450	2.990	3.320
p value	0.89	0.889	0.888	0.002	0.009	0.232

[1]Treatments are: NT= birds raised in normal temperature, LT= birds raised in low temperature, [a, b] Means in each column with different superscripts are significantly different ($p \leq 0.05$)

consequent insufficient oxygenation of the tissues (Julian, 1993). Exposure to cold conditions enhances the imbalance between supply and oxygen needs and changes in the cardiovascular system have been observed to accommodate the needs for oxygen under those conditions (Luger *et al.*, 2001). The mass of the RV primarily reflects the work performed to propel blood flow through the pulmonary vasculature, where the left ventricular mass increases in proportion to body weight and thereby reflects the work performed to deliver a cardiac output sufficient to supply oxygen and nutrients in support of whole body requirements for maintenance, activity and growth (Burton and Smith., 1967; Burton *et al.*, 1968). Finally growth tends to decelerate subsequent to the onset of hypoxemia, presumably reflecting a cardiopulmonary capacity that is inadequate to deliver oxygen in quantities sufficient to sustain maximal growth (Owen *et al.*, 1990; Yersin *et al.*, 1992; Roush *et al.*, 1994; Wideman and Kirby, 1996).

It is known that various organ changes and an increase in the RV/TV ratio indicate the onset of pulmonary hypertension and ascites syndrome (Huchzermeyer and Deruyck, 1986; Hernandez, 1987). An RV/TV ratio of more than 0.27 is considered an accurate measure of the onset of ascites (Huchzermeyer and Deruyck, 1986; Peacock *et al.*, 1988). In recent research, we observed an RV/TV ratio of 0.29 for LT group in

comparison to 0.20 for NT treatment and the difference between these ratios for two groups was significant ($p \leq 0.05$). Balog *et al.* (2003) showed that relative heart weight was significantly higher in the susceptible and relaxed line in comparison to resistant lines but relative weights of liver and spleen were not different between susceptible and resistant lines. It seems that such organ weights can be used as indices of ascites syndrome. Although in our study the relative heart weight in LT birds was higher than that of NT birds but this difference was not significant. Liver and spleen weights were not significant between two treatments too. Due to high oxygen demands of GIT, it concludes that the birds that reveal ascites syndrome have restricted oxygen for developing GIT, consequently they must have different gut weights from healthy birds although we didn't notice any significant difference between empty gut weight of LT and NT birds. It might have been some changes in gut morphology. Solis de los Santos *et al.* (2005) investigated the gut morphology of susceptible and resistant lines to ascites in hypobaric chambers and they observed that under hypoxic conditions, duodenum villus surface area was higher in resistant line compared with the susceptible line. Their results showed the reduced enteric function (Soils de los Santos *et al.*, 2005). Ascites syndrome is a severe cause of loss to the broiler industry in many countries, not only due to high mortality, but also due to

reduced weight gain and increased condemnations at slaughter (Julian, 1993). Ascites syndrome causes a significant deterioration in the performance of the broilers before the death happens (Luger *et al.*, 2001). As observed, weight gain of NT and LT treatments was not different till 3 weeks of age but in week 4 the weight gain of LT birds was significantly lower than that of NT birds. The overall weight gain of LT birds was significantly lower than that of NT birds and this effect might be due to reduction in feed intake that seen after week 3. It was imagined that cause of susceptibility of broilers to ascites is high rate of growth (Dale and Villacres, 1988; Vereijken and Alberts, 1990). But Luger *et al.* (2001) showed that weight gain of ascetic birds was significantly lower than healthy birds. They observed no significant difference in weight gain between ascetic and healthy birds up to 3 weeks of age, but thereafter a significant decline in weight gain was observed in the ascetic chickens prior to death. Wideman (2000) emphasized that broilers susceptible to ascites do not have to be the fastest growing members of the flock as long as their rate of weight gain exceeds the rate at which their pulmonary vascular capacity increases to accommodate cardiac output (Luger *et al.*, 2001). Lubritz and McPherson (1994) investigated the effect of genotype and cold stress on incidence of ascites in cockerels and showed that effects of cold on body weight were more dramatic up to 28 days of age when cold stress significantly decreased body weight. Also, Wideman *et al.* (1998) observed that ascitic males have lower body weights on day 42 in comparison to nonascitic males. Our results were similar to that previously reported in the literature and this reduction in performance is possibly due to decrease in feed intake. It is said that cold stress while limiting the access of birds to feed might put more distress, which speculated in the greater reduction in body weight (Khajali and Qujeq, 2005).

Venous blood parameters provide more information on overall tissue oxygenation, distribution of oxygen and elimination of CO_2 from the circulation. Venous blood closely approximates mean tissue O_2 and CO_2 tension and reflects changes of tissue aerobic metabolism. The mean partial pressure of oxygen in tissue is useful because it is indicative of oxygen availability (Black and Tenney, 1980). Experiments of Besch and Kadono (1977) with domestic chickens showed that under strict hypoxic conditions (13% pO_2), pO_2 in arterial blood was reduced to about 30 mmHg, but pCO_2 was not significantly changed. These results indicate that the birds were able to keep arterial pCO_2 constant under changing circumstances with little variation within flocks. The surveys of other researchers showed the positive results. Scheele *et al.* (2003) used differences in blood pCO_2 between juvenile birds within a Ross broiler cross population to select two different flocks which could be characterized as an ascites-resistant and an ascites-sensitive flock. At a younger age (11 d) of these flocks, pCO_2 and in pO_2 were different. Balog *et al.* (2003) showed higher blood pCO_2 for susceptible lines to ascites than resistant lines. Also Wideman *et al.* (1998) observed the lower percentage saturation of hemoglobin with oxygen for cold induced ascitic males on day 42 in comparison to non ascetic males. Buys *et al.* (1998) demonstrated that the progeny of ascites-sensitive broilers lines (incubated under normal conditions) had higher pCO_2 in venous blood at 6 weeks than the progeny of ascites-resistant lines. They also reported that differences in pCO_2 values between ascites-resistant lines and sensitive lines in venous blood were similar to those in arterial blood. Olkowski *et al.* (1999) reported that at day 14, blood pO_2 and O_2 saturation of blood was significantly different between ascetic and healthy birds. The levels of two variables were lower in chickens developing ascites in comparison to normal chickens and blood pCO_2 at this age was higher for ascetic birds but the difference was not significant. In their work, at day 35, ascetic chickens had lower pO_2 and O_2 saturation and higher pCO_2 and HCO_3^- in both venous and arterial blood. Blood $pO2$ and O_2 saturation tended to decline and pCO_2 tended to increase with the age. Blood pH was not significantly different in 14 and 35 days of age for ascetic and healthy birds. Hassanzadeh *et al.* (1997) observed no significant differences between birds who received T3 in their diet for inducing ascites and normal birds for pCO_2, pO_2 and O_2 saturation at ages 3, 4 and 5 weeks but HCO_3^- and pH for normal birds (control) was statistically higher. Buys *et al.* (1999) showed that high susceptible lines to ascites had low pO_2 and higher pCO_2 in venous blood at low ambient temperature. These lines had a respiratory acidosis and they postulated that the respiratory and/or cardiovascular system of these birds seemed unable to cope with the increased metabolic demands of these stocks at low temperature. Also in all except one stock, venous pCO_2 as well as bicarbonate values were higher at low temperature than at high temperature but the resulting pH did not show any differerence between temperature regimes. Olkowski *et al.* (1999) indicated that hypoxaemia was not the critical symptom leading to ascites, but that could be the result of the pathophysiology of ascites. However they showed that hypercapnia (high pCO_2) was a feature of ascitic broilers. The increased venous pCO_2 and consequently decreased venous pO_2 could indicate a chronic lung insufficiency, compensated by increased renal bicarbonate retention (Beetham, 1982).

CONCLUSION

In our research, although the results of blood analysis in Table 4 showed that differences between ascitic induced birds (LT group) and healthy birds (NT group) was only significant for pO_2 and O_2 saturation in some weeks and differences for other variables were not significant but generally pO_2 and O_2 saturation was lower and pCO_2 and HCO_3^- was higher for ascitic induced birds in comparison to healthy birds. Moreover, pH of ascitic induced and healthy birds were similar. The trend of changes for some blood parameters in our research was similar to the study showed in the literature. However, some of our results in some weeks were not consistent with others and some variables such as pCO_2 and HCO_3^- were not significant between LT and NT treatments. This inconsistency might be related to the low number of birds (5 birds). Mortality caused by ascites (8.8%) and RV/TV ratio (0.29) of ascetic birds were higher and it seems that the format of this study is truly designed. In overall, under the conditions of this study, it was concluded that ascites had some impact on performance of broilers and pO_2 and O_2 saturation of blood that decreased by ascites might be involved in these results.

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