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Comparison of The Effects of Several Feed Restriction Periods to Control Ascites on Performance, Carcass Characteristics and Hematological Indices of Broiler Chickens

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■ Keywords

Ascites, broiler, feed restriction, hematological indices, performance.

ABSTRACT

This experiment was conducted to evaluate the effect of some feed restriction regimes on the performance, carcass characteristics and hematological values of broiler chickens. A number of 480 one-day-old Cobb broilers were distributed into 24 floor pens and reared for 42 days. A basal diet was formulated according to the NRC (1994) recommendations for starter (1-11 d), grower (12-28 d) and finisher (29-42 d) periods. The restricted birds were fed 8 h per day from 7 to 21 ($R_{7\text{-}21d}$), 14 to 28 d ($R_{14\text{-}28d}$) or from 21 to 35d ($R_{21\text{-}35d}$), while the control birds were fed *ad libitum*. Birds and feed were weekly weighed, and dead birds were submitted to necropsy to determine the cause of death. At the end of experiment, blood samples were taken from 3 birds per pen, birds were weighed, and organ weights were recorded. All birds in the feed restriction treatments had lower body weight gain and feed intake than those fed *ad libitum*. Feed conversion ratio (FCR) was better in birds under $R_{7\text{-}21d}$ as compared to the control group ($p<0.05$). $R_{7\text{-}21d}$ and $R_{14\text{-}28d}$ birds presented lower general mortality and death due to ascites as compared to *ad libitum*-fed broilers. The birds under $R_{21\text{-}35d}$ and $R_{14\text{-}28d}$ treatments had lower breast weight and abdominal fat weight as compared to control birds, respectively ($p<0.05$). A similar trend was observed for the percentage of the mentioned cuts. The right/total ventricular weight ratio (RV/TV) was significantly lower both in $R_{7\text{-}21d}$ and $R_{14\text{-}28d}$ treatments as compared to the control group. Blood constituents, such as glucose, cholesterol, and lactate dehydrogenase were also significantly altered by feeding regimes ($p<0.05$). The results of present study showed that $R_{7\text{-}21d}$ reduced ascites incidence, but had no significant effect on growth performance.

INTRODUCTION

The ascites syndrome (ascites) is the primary cause of death for rapidly growing broiler strains, resulting in economic loss (Hassanzadeh *et al*., 2009). Ascites is a condition in which the body cavity accumulates serous fluid, leading to carcass condemnation or death (Julian, 1993). The causes of the syndrome are multifactorial and mainly induced by exogenous and/or endogenous factors. An imbalance between oxygen supply and the oxygen required to sustain rapid growth rates and high food efficiencies is believed to be the primary cause of ascites in broiler chickens (Decuypere *et al*., 2000 & 2005; Julian, 2005). The housing environment, including factors such as temperature (cold or fluctuating temperatures) and air quality (dust concentration, carbon dioxide levels, and oxygen levels), is known to influence the incidence of ascites in broiler chickens. The incidence of ascites greatly increases at altitudes greater than 1300 meters above sea level, presumably because of the low oxygen partial pressure (Hernandez, 1987). Physiologically, low

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oxygen (O_2) concentration creates an oxygen deficit (hypoxia) and a demand for more oxygen. The increased demand may exceed the cardiopulmonary capacity to supply sufficient oxygen, resulting in pulmonary hypertension and right ventricular failure (Julian, 1993). The peak of ascites incidence occurs in weeks 5-6 of the growing period, but it is thought that the etiology of the syndrome is initiated much earlier, even during the embryonic stage (Coleman & Coleman, 1991). Feed restriction is becoming a common treatment employed to reduce the incidence of ascites in broilers and roasters under commercial conditions (Arce *et al*., 1992; Acar *et al*., 1995). Research has shown that feed restriction timing, duration, and severity have an impact on whether a bird is capable of achieving the same body weight as unrestricted birds (Ballay *et al*., 1992; Yu & Robinson, 1992). Early feed restriction regimes for broiler chickens have been previously researched (Mollison *et al*., 1984; Summers *et al*., 1990; Wall *et al*., 1999; Bhat & Banday, 2000; Lee & Leeson, 2001). The benefits of early feed restriction are the monetary savings obtained by improved feed conversion (Proudfoot *et al*., 1983), reduced sudden death syndrome (Mollison *et al*., 1984; Bhat & Banday, 2000), reduced death losses (Tottori *et al*., 1997), reduced ascites (Arce *et al*., 1992) and reduced skeletal disease (Robinson *et al*., 1992). Not only the early feed restriction regimes, but also the late feed restriction or feed withdrawal regimes before four to five weeks of age may reduce the incidence and severity of ascites. Therefore, the objectives of the study were to evaluate the effects of early and late feed restriction on broiler growth performance, carcass characteristics, ascites syndrome incidence, and changes in blood constituents.

MATERIALS AND METHODS

Birds and housing

One-day-old male broilers of a commercial strain (Cobb), obtained from a commercial hatchery, were kept on floor pens and fed *ad libitum* with a pelleted commercial broiler starter diet until 7 days of age. At this age, 480 broiler chicks were individually weighed to obtain similar pen weights, and randomly assigned to one of four treatments, with six replicates of 20 chicks each. The chicks were placed at simulated 1890 m (6,200 ft) above sea level and housed in 24 floor pens covered with 10-cm deep, clean wood shavings after 7 days of age. In order to increase probability of ascites incidence, a cold stress temperature schedule was applied. All birds were brooded under about 35 °C from 1 to 7 d of age, then 30 °C from 8 to 14 d. Thereafter, birds were subjected to a step-down temperature program of 1 to 2 °C per day down until 12 to 14 °C, where it remained constant until the end of the experiment. A continuous lighting program was provided for all treatment groups during the experiment.

Diets

Birds were fed commercial corn-soybean pelleted broiler starter, grower and finisher diets, formulated to meet or exceed minimum NRC (1994) standards for all ingredients. All birds received a starter diet from 1 to 11 d. The grower and finisher diets were provided from 12 to 28 d and 29 to 42 d of age, respectively. The compositions of the diets are shown in Table 1.

Table 1 - Ingredient composition (as percent of dry matter) and calculated analysis of the basal diets.

Ingredients	Starter	Grower	Finisher
Yellow corn	495.3	530.4	536.4
Soybian meal(44%)	383.1	315.1	300.2
Wheat grain	50.2	80	100
Fish meal (66%)	25	25	0
Vegetable oil	10.3	16	28
Dicalcium phosphate	15.3	14.4	15.2
Oyster shell	9.5	8.4	10.2
Salt	1.5	1.4	1.8
Vitamin-mineral premix[1]	6	5.5	5
Coccidiostat	0. 4	0.4	0.4
DL- Methionine	2.3	2.2	2
L-Lysine	1.1	1.2	0.8
Calculated chemical composition			
ME (MJ/kg)	12.14	12.56	12.97
Crude protein (%)	22.2	20.2	18.2
Calcium (%)	1	0.97	0.92
Available phosphorus (%)	0.5	0.48	0.45

1 - vitamin and mineral provided per kilogram of diet: vitamin A, 360000 IU; vitamin D3, 800000 IU; vitamin E, 7200 IU; vitamin K3, 800 mg; vitamin B1, 720 mg; vitamin B9, 400 mg; vitamin H2, 40 mg; vitamin B2, 2640 mg, vitamin B3, 4000 mg; vitamin B5, 12000 mg; vitamin B6, 1200 mg; vitamin B12, 6 mg; Choline chloraid, 200000 mg, Manganeze, 40000 mg, Iron, 20000 mg; Zinc, 40000 mg, coper, 4000mg; Iodine, 400 mg; Selenium, 80 mg.

Feed restriction program

Three quantitative feed restriction programs were applied at different ages. Broilers on treatment $R_{7\text{-}21d}$ had feed available for 8 h daily for 2 weeks, from d 7 to 21, and then received *ad libitum* feed for the remaining 3 weeks. Broilers on treatment $R_{14\text{-}28d}$ were fed 8 h daily from d 14 to 28, then returned to *ad libitum* feeding. Treatment $R_{21\text{-}35d}$ were fed *ad libitum* for the first 3 weeks, had 8 h feed availability for the next 2 weeks, and then fed *ad libitum* for the remaining 1

week. The controls were given *ad libitum* access to feed throughout rearing.

Variables measured

Body weight and feed consumption were weekly measured per pen. Feed conversion ratio was calculated on pen basis. The incidence of ascites, death due to ascites, and overall mortality rate were determined at 7 and 42 days of age and calculated as percentage of total birds in each treatment. Incidence of ascites (non-fatal) was determined by physical examination. However, all dead birds were autopsied to determine if the cause of death was ascites. Birds that exhibited marked hypertrophy in the right ventricle and the pericardium filled with fluid were identified as dead due to ascites. Birds with severe or less severe ascites syndrome were identified to calculate the incidence of ascites. At the end of the experimental period, 4 ml of blood was collected from wing vein from 18 birds in each treatment. In order to prevent clotting, blood was collected in heparinized test tubes and centrifuged (at 2,000 rpm for 10 min), and the serum was separated, then stored at -20°C until assayed to measure blood parameters (glucose, total protein, albumin, cholesterol, and lactate dehydrogenase) using appropriate laboratory kits (Gordon *et al*., 1977; Gowenlock *et al*, 1988). Hematocrit was measured using microhaematocrit (Safamehr, 2008) and activity of serum lactate dehydrogenase was measured by a spectrophotometric method (Mc Comb *et al*., 1976). Serum globulin was calculated by subtracting serum albumin from total serum protein levels. Birds were then weighed and slaughtered for carcass evaluation (Perreault & Leeson, 1992). Carcass, breast, heart, and abdominal fat were individually weighed and expressed as a percentage of live body weight. Hearts were dissected and, after careful removal of the atria, right ventricle (RV) and total ventricle (TV) were weighed to calculate RV/TV weight ratio, which is considered as an index of ascites (Julian, 1993; Lubritz *et al*., 1995).

Statistical analysis

All data were submitted to analysis of variance using the One-Way Anova procedure of SAS® (SAS, 1998). Differences among treatments were determined as significant at 5% level by Duncan's multiple range tests (Duncan, 1955).

RESULTS AND DISCUSSION

Growth performance and mortality

The effects of feed restriction regimes on performance and mortality are presented in Table 2. The control group presented significantly higher performance than the $R_{14\text{-}28d}$ and $R_{21\text{-}35d}$ groups. Feed consumption was significantly reduced by feed restriction regimes ($p>0.05$). Feed conversion was improved in all restricted-fed groups, although this differences was significant ($p<0.05$) only when the control group was compared to the $R_{7\text{-}21d}$ treatment. Both $R_{7\text{-}21d}$ and $R_{14\text{-}28d}$ regimes significantly decreased the incidence of ascites, mortality, and death due to ascites. Also, there was a significant difference between $R_{7\text{-}21d}$ and $R_{14\text{-}28d}$ in death due to ascites rate.

Similar observations were reported for body weight gain of birds under $R_{7\text{-}8d}$ and $R_{21\text{-}35d}$ by Balog *et al*, (2000) and Camacho *et al*, (2004), respectively. On the other hand, our findings for body weight gain of birds under $R_{7\text{-}21d}$ contrast with those of Pan *et al*. (2005). It seems that the birds restricted-fed from 14 to 28 d or 21 to 35 d of age did not present sufficient compensatory growth to achieve the same final body weight of the control group. As a result, the age of birds at the beginning of the restriction period had an important effect on compensatory growth rate. Plavnik & Hurwitz (1990) tested the effect of age on the response of male chicks to feed restriction. Restriction started at 3, 5, or 7 d of age, and feed intake was significantly reduced by feed restriction, without any difference due to age. However, the present study shown that feed intake following feed restriction from 21 to 35 d was significantly reduced. Also, our findings for FCR of R7-21d group are consistent with those of Pan *et al*, (2005),

Table 2 - The main effects of treatments on performance and mortality of broiler chickens at 42d.

Variables	Treatments				SEM
	Control	$R_{7\text{-}21d}$	$R_{14\text{-}28d}$	$R_{21\text{-}35d}$	
Body weight gain (g)	2286.14 a	2224.09 ab	2171.73 b	2161.42 b	36.49
Feed consumption (g)	4558.34 a	4302.42 b	4254.93 b	4237.56 b	43.13
Feed conversion ratio (g g-1)	1.99 a	1.93 b	1.95 ab	1.96 ab	0.01
Mortality (%)	12.50 a	4.16 b	5.83 b	9.16 ab	1.82
Death due to ascites (%)	10.83 a	4.16 b	5.00 b	7.50 ab	1.77
Incidence of ascites (%)	17.50 a	8.33 bc	6.66 c	13.33 ab	1.79

a,b - means in each column with different superscripts are significantly different ($p<0.05$).

whi reported that cumulative FCR between d 7 and d 49 was not significantly affected by feed restriction.

It is known that early feed restriction reduces the incidence of ascites, mortality ,and death due to ascites (Leeson *et al*., 1992; Acar *et al*., 1995; Balog *et al*., 2000; Camacho *et al*., 2004; Pan *et al*., 2005). A similar result was obtained in the present study. Although the birds in the $R_{7\text{-}21d}$ group followed a similar trend, there were no significant differences as compared to the control group. In the present study, out of the 480 broilers used, 38 died between 7 and 42 days of age. A total of 33 broilers died due to ascites in all treatment groups.

Carcass composition

The effect of the experimental treatments on the carcass weight (both in terms of absolute value and percentage of body weight) and RV/TV ratio are given in Table 3. Carcass weight and breast weight and yield of birds under $R_{21\text{-}35d}$ treatment were significantly lower as compared to those of control treatment ($p<0.05$). Mean abdominal fat weight and yield of $R_{21\text{-}35d}$ birds was lower as compared to the control treatment ($p<0.05$). RV weight and RV/TV values obtained with $R_{7\text{-}21d}$ and $R_{14\text{-}28d}$ treatments were lower than those of birds fed *ad libitum* ($p<0.05$). Carcass weight and heart weight and yield did not differ among treatments ($p>0.05$).

However, our carcass weight findings in birds under the $R_{21\text{-}35d}$ treatment was in contrast to those of Camacho *et al*. (2004). The results abdominal fat content of treatments $R_{7\text{-}21d}$ and $R_{21\text{-}35d}$ support the recent findings of Summers *et al*. (1990), Yu *et al*. (1990), and Fontana *et al*. (1993), who observed no differences in abdominal fat pad weight between *ad libitum* and feed-restricted broilers during the restriction period. Contrary to these findings, Plavnik *et al*. (1986), Rosebrough *et al*. (1986), Acar *et al*. (1995), and Saleh *et al*. (1996) determined that feed restriction reduced abdominal fat pad weight, which is in agreement with our finding for the $R_{14\text{-}28d}$ group. According to the study of Rosebrough *et al*. (1986), the activities of lipogenic enzymes were depressed during the period of feed restriction, peaked in the first week of refeeding, and gradually declined to below those of the control birds in the subsequent weeks. The differences in these findings may be due to the differences in treatments, time of feed restriction, sex, strains, or duration of feed restriction. The percentage of total heart relative to body weight at 42 days of age was also not statistically affected by the feeding restriction regimes. This is consistent with the results of Acar *et al*. (1995). Also in the present study, the negative effect of feed restriction on breast yield was in contrast with those reported by Camacho *et al*. (2004). Before a bird exhibits gross ascites syndrome lesions, hematological and anatomical changes can be detected (Maxwell *et al*., 1986; 1987). An increase in RV/TV ratio indicates the onset of pulmonary hypertension and ascites syndrome (Burton *et al*., 1968; Cueva *et al*., 1974; Huchzermeyer & DeRuyck, 1986; Hernandez, 1987). It is generally accepted that an RV/TV index greater than 0.30 is indicative of right ventricular hypertrophy, pulmonary hypertension and, ultimately, ascites syndrome (Cueva *et al*., 1974; Hernandez, 1987; Huchzermeyer *et al*., 1988; Wideman *et al*., 1998). In contrast to our findings, Balog *et al*., (2000) reported that the RV/TV ratio was not increased in restricted-fed birds at hypobaric altitude.

Table 3 - The effect of feed restriction on carcass composition and heart characteristics of broiler chickens at 42 d.

Variables	Treatments				SEM
	Control	$R_{7\text{-}21d}$	$R_{14\text{-}28d}$	$R_{21\text{-}35d}$	
As gram					
Carcass	1994.22 a	1996.74 a	1928.62 ab	1895.40 b	30.95
Breast	645.39 a	609.92 ab	589.39 ab	562.65 b	19.85
Abdominal fat	44.68 a	39.94 ab	35.45 b	37.33 ab	2.49
Heart	9.51 a	9.30 a	8.77 a	8.89 a	0.46
TV[1]	7.56 a	7.39 a	7.38 a	7.38 a	0.13
RV[2]	1.99 a	1.82 b	1.80 b	1.87 ab	0.03
As %					
Carcass	79.67 a	80.33 a	79.45 a	77.89 a	1.14
Breast	25.77 a	24.50 ab	24.28 ab	23.145 b	0.76
Abdominal fat	1.78 a	1.60 ab	1.45 b	1.53 ab	0.09
Heart	0.38 a	0.37 a	0.36 a	0.36 a	0.01
TV	0.303 a	0.298 a	0.305 a	0.303 a	0.01
RV	0.080 a	0.073 b	0.075 ab	0.076 ab	0.01
RV/TV	26.35 a	24.65 b	24.46 b	25.32 ab	0.74

a,b - means in each column with different superscripts are significantly different ($p<0.05$). 1 TV= Total Ventricular, 2RV= Right Ventricular.

Blood constituents

The effect of feed restriction on blood constituents is presented in Table 4. No significant differences were observed in total protein, albumin, or globulin levels among treatments. Broilers under the $R_{21\text{-}35d}$ treatment had significantly higher blood glucose levels than broilers in group $R_{7\text{-}21d}$ and control group ($p<0.05$). Lactate dehydrogenase levels for $R_{21\text{-}35d}$ and $R_{14\text{-}28d}$ broilers were significantly higher than in control broilers. $R_{21\text{-}35d}$ regime caused a significant decrease in hematocrit value as compared to full-fed broilers. Serum cholesterol concentrations of $R_{14\text{-}28d}$ and $R_{21\text{-}35d}$ treatments were lower than those of control birds ($p<0.05$).

Blood parameters findings in this experiment, except for cholesterol and albumin, support previous findings (Balog *et al*., 2004; Demir *et al*., 2004). Also, serum cholesterol data are not in agreement with the data of Church & Pond (1988), who indicated that meal-fed or starved chickens have been shown to have higher plasma cholesterol than control birds. The higher levels of serum glucose in restricted-fed birds may be due to better FCR and nutrient absorption. However, Riesenfeld *et al*. (1982) and Klasing (1998) have reported that chickens maintain a steady glucose level during fasting. Hypoglycemia during fasting is prevented by glucose synthesis via gluconeogenesis. Lactate is produced from glucose by the intestine and up to 37% of the glucose taken up from the intestinal lumen may be converted into lactate before being transferred to the circulation (Riesenfeld *et al*., 1982). Lactate dehydrogenase is responsible for converting pyruvate into lactate, and therefore, the high levels of glucose in $R_{21\text{-}35d}$ and $R_{14\text{-}28d}$ broilers may have caused an increase in the blood lactate dehydrogenase levels. Hematocrit is another common measure of ascites development (Burton & Smith, 1967; Maxwell, 1991; Mirsalimi & Julian, 1991; Yersin *et al*., 1992; Fedde & Wideman, 1996). As expected, broilers fed *ad libitum* had a higher hematocrit values due to their higher growth rate and oxygen requirement. According to the results, it is probable that if the restricted-fed birds had been allowed to continue full feed for several more weeks, their hematocrit values would have approached those of the fully fed controls.

CONCLUSION

The results of this study showed that the age at which feed restriction regime is applied is an important factor to control mortality from ascites and to allow compensatory growth of the restricted-fed chickens. According to the results of performance and ascites indexes the $R_{7\text{-}21d}$ and $R_{14\text{-}28d}$ treatments were more effective to reduce ascites problems.

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Table 4 - The effect of feed restriction on blood constituentd of broiler chickens at 42 d.

	Treatments				
Parameters	**Control**	**$R_{7\text{-}21d}$**	**$R_{14\text{-}28d}$**	**$R_{21\text{-}35d}$**	**SEM**
Glucose (mg dl-1)	213.66 b	213.50 b	217.50 ab	224.83 a	2.70
Total protein (g dl-1)	3.89 a	3.76 a	3.80 a	3.84 a	0.18
Albumin (g dl-1)	1.78 a	1.74 a	1.80 a	1.74 a	0.14
Globulin (g dl-1)	2.10 a	2.02 a	1.99 a	2.09 a	0.12
Cholesterol (g dl-1)	165.73 a	159.13 ab	143.55 b	146.10 b	6.03
Hematocrit (%)	31.10 a	30.67 ab	30.07 ab	29.76 b	0.37
lactate dehydrogenase (IU l-1)	487.06 b	540.70 ab	553.20 a	556.08 a	18.80

a,b - means in each column with different superscripts are significantly different ($p<0.05$).

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