Ascites Syndrome and Related Pathologies in Feed Restricted Broilers Raised in a Hypobaric Chamber


*USDA, Agricultural Research Service, Poultry Production and Product Safety Research and †Department of Poultry Science, Center of Excellence for Poultry Science, University of Arkansas, Fayetteville, Arkansas 72701

ABSTRACT

It has been demonstrated that the incidence of ascites can be significantly reduced through feed restriction. This method is thought to have an effect by slowing the growth rate of the birds. Interestingly, when birds are grown in a hypobaric chamber, ascites incidence increases while the overall growth rate of the birds is decreased. Unfortunately, the restriction programs practiced also have a detrimental effect on growth characteristics. An experiment was conducted to determine if the timing and duration of feed restriction can be used to reduce the incidence of ascites for broilers reared under high altitude and local elevation without having a negative impact on growth. A total of 600 commercial broiler males were used. Birds were divided, placing 360 birds in the hypobaric chamber at a simulated 2900 m (9,500 ft) above sea level, and 240 birds were placed at local elevation [390 m (1,260 ft) above sea level]. At each altitude there were four treatments: 1) fully fed controls; 2) feed available for 8 h/d for 6 wk (the duration of the study); 3) feed available for 8 h/d during the first 3 wk, then full feed for the remaining 3 wk; and 4) full feed for the first wk, then 3 wk of 8 h of feed availability, then 2 wk of full feed. Birds and feed were weighed weekly, and mortalities were necropsied to determine the cause of death. At the end of 6 wk, blood samples were taken, and the birds were weighed, necropsied, and scored for ascites, and organ weights were recorded. All feed restriction treatments significantly reduced ascites incidence, when compared with the fully fed controls. Treatment 2 birds were significantly lighter than any other group at both altitudes. The fully fed controls at local elevation were heavier than the fully fed controls at simulated high altitude, as seen in past experiments.

(Key words: ascites, feed restriction, hypobaric chamber, broilers)

INTRODUCTION

Feed restriction is becoming a more common commercial treatment employed to reduce the incidence of ascites in broilers and roasters (Arce et al., 1992; Acar et al., 1995). This method is thought to have its effect by slowing the growth of the birds. One important observation to note is that slowing growth does not always reduce ascites incidence. Interestingly, when birds are grown in a hypobaric chamber in our laboratory, ascites incidence increases, whereas overall growth rate of the birds is decreased. Birds grown at simulated high altitude are always lighter than their counterparts grown in an environmentally matched chamber at local altitude [390 m (1,260 ft) above sea level] (unpublished data). The hypobaric chamber simulates high altitude and induces ascites by reducing oxygen availability.

An important aspect of using feed restriction to prevent ascites is the timing and duration of the restriction. Powerful management tools can be developed by identifying feeding strategies that can reduce ascites and allow for compensatory gain. Research has shown that the timing, duration, and severity of the restriction have an impact on whether a bird is able to obtain BW consistent with unrestricted birds (Ballay et al., 1992; Yu and Robinson, 1992). To lessen the severity of feed restriction while reducing the incidence of ascites would be to the advantage of growers.

Additionally, as birds develop ascites, they begin to exhibit specific hematological changes (Maxwell et al., 1986, 1987). These changes occur prior to gross physical changes and can be used as markers that indicate a bird or group of birds is in the process of developing ascites.
syndrome. Although a specific feed restriction program may reduce ascites mortality in broilers, these birds may continue to show indicators of the early disease process. In this case, this program may not be beneficial for producers of roasters because they are kept on feed for additional weeks and may develop ascites.

This experiment was designed to identify a feed restriction program that could be used to reduce the incidence of ascites syndrome in broilers raised to 6 wk in a hypobaric chamber or at local elevation while allowing for compensatory growth.

**MATERIALS AND METHODS**

Six hundred vaccinated commercial broiler males (Cobb) were placed at 1 d of age. Three hundred sixty birds were placed at simulated 2900 m (9,500 ft) above sea level. These birds were randomly assigned to one of four treatment groups (five replicate pens per treatment group). Treatment 1 (control) received full feed ad libitum. Treatment (Trt) 2 had feed available for 8 h daily for the duration of the experiment (6 wk). Treatment (Trt) 3 had feed available for 8 h daily for the first 3 wk and then received full feed ad libitum for the remaining 3 wk. Treatment (Trt) 4 had full feed ad libitum for the first week, 8 h feed availability for the next 3 wk, and then full feed ad libitum for the remaining 2 wk. The remaining 240 birds were placed at 390 m (1,260 ft) above sea level (local altitude). They were randomly assigned to the same feed treatments as the birds at simulated high altitude. Five replicate pens were used per treatment group.

Birds were housed in stainless-steel battery units equipped with nipple waterers in a control chamber or one that simulated high altitude by operating under a partial vacuum. Both chambers measured 3.7 m × 2.4 m and were matched in terms of temperature and ventilation. Ventilation was set to maintain airflow at 17 m³m⁻¹ (600 cfm). Birds were warm-room brooded with temperatures decreasing weekly. Birds at simulated high altitude remained at high altitude for the duration of the experiment. Daily management tasks and weekly weighings were conducted under the partial vacuum through the use of an airlock that allowed for pressure equilibration.

Pen weights and feed intakes were obtained weekly. The feed was a nutritionally complete starter and grower that met or exceeded the National Research Council (1994) requirements. Water was available ad libitum, even during periods of feed restriction. All mortalities were weighed daily and necropsied to determine the cause of death.

At 6 wk of age, blood samples were taken via heart puncture. Total white blood cell counts, differentials, and hematology values were determined using a Cell-Dyn 3500 blood analysis system³ that had been standardized for analysis of chicken blood. Serum concentrations of calcium, inorganic phosphorus, total protein, albumin, glucose, and triglycerides were determined using a Ciba-Corning Express Plus clinical chemistry analyzer and the reagents and procedures standardized for use with the analyzer.⁴ Birds were weighed, necropsied, scored for ascites (on a scale of 0 to 3, where 0 = no sign of ascites and 3 = severe ascites), and split heart, liver, and spleen weights were obtained. Pectoralis major and minor were removed and weighed. This experimental protocol was approved by the University of Arkansas Institutional Animal Care and Use Committee and complied with university and federal guidelines for research involving animal subjects.

All percentage data were subjected to arc sine transformation. The experimental design employed a 2 × 4 factorial arrangement of treatments. Data were subjected to ANOVA using SAS⁵ (SAS Institute, 1988). When necessary, mean separation was accomplished using Duncan’s multiple range test (Duncan, 1955). A probability value of less than 0.05 was considered significant, unless otherwise noted.

**RESULTS AND DISCUSSION**

From past experiments, we knew that birds grown at simulated high altitude would weigh less than birds grown under environmentally matched local altitude conditions. Figure 1 indicates that this disparity in growth rate is evident after the first week and continues throughout the entire experimental period. There is almost a 300-g difference between local altitude and the hypobaric chamber final average BW. Although local altitude birds grow more rapidly, the hypobaric birds exhibit a significantly greater incidence of ascites (Figure 2). We define the incidence of ascites as the percentage of mortality caused by ascites plus the percentage of birds exhibiting gross anatomical indications of ascites at the 6-wk necropsy. This increase in ascites inci-

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³Abbott Diagnostics, Abbott Park, IL 60064.
⁴Ciba-Corning Diagnostic Corp., Medfield, MA 02052.
Treatments 3 and 4, which birds were restricted for 3 wk and had 3 wk of full feed, exhibited compensatory gain. At both altitudes, the Trt 3 and 4 birds finished the 6-wk trial with BW that were not significantly different from the fully fed controls. There were no significant differences in feed efficiencies between treatment groups at either altitude (data not shown).

Although the Trt 3 and 4 restriction programs did not affect final BW, these restriction programs did affect ascites incidence. Figure 2 shows that all restricted groups had significantly lower ascites incidence when compared with the fully fed controls. At local altitude, ascites incidence was low in all groups, but the birds that had been restricted exhibited an ascites incidence near zero. Birds reared in the hypobaric chamber, as expected, exhibited a much greater ascites incidence. In the hypobaric chamber, fully fed controls had an ascites incidence of 65%. The Trt 2 birds that were restricted for the entire 6 wk had very low ascites incidence (4%). Both groups that received combined restriction and full feed (Trt 3 and 4) had an ascites incidence that was intermediate to fully fed controls and the fully restricted group (Trt 2).

Before this experiment, a preliminary trial was conducted (Balog et al., 1998; Cooper et al., 1998). Because of mechanical failure, birds in the hypobaric chamber were only at simulated high altitude for the first 4 wk of the trial. The final 2 wk were spent at local altitude. The BW and ascites incidence in the preliminary trial were nearly identical to those shown for this experiment in Figures 3 and 4.

The BW at 6 wk of both dietary restriction treatments in the local altitude and the hypobaric chamber. Within altitudes, the Trt 2 birds that were restricted to 8 h of feed availability per day were significantly lighter than any of the other restrictions and the fully fed controls. Both groups that received combined restriction and full feed (Trt 3 and 4) had an ascites incidence that was intermediate to fully fed controls and the fully restricted group (Trt 2).

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Although BW and ascites data indicate that the restriction programs employed by Trt 3 and 4 give us desirable results (high BW and low ascites incidence), the necropsy results indicate a potential problem. Before a bird exhibits gross ascites syndrome lesions, there are hematological and anatomical changes that can be detected (Maxwell et al., 1986, 1987). An increase in the right ventricle:total ventricle weight (RV/TV) ratio indicates the onset of pulmonary hypertension and ascites syndrome (Burton et al., 1968; Cueva et al., 1974; Huchzermeyer and DeRuyck, 1986; Hernandez, 1987). Figure 4 shows the RV/TV weight ratio of birds at 6 wk. 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 (Burton et al., 1974; Huchzermeyer et al., 1988; Wideman et al., 1998). Although ascites incidence was significantly decreased by the feed restriction programs of Trt 3 and 4 at high altitude, the RV/TV ratio of these birds indicates early ascites development. At high altitude, both Trt 3 and 4 had mean RV/TV ratios that were not significantly different from the fully fed controls. Only the completely restricted birds in Trt 2 had lower RV/TV ratios, and these were still above the accepted cutoff of 0.30 for ascites development. It is possible that given more time at high altitude, even the completely restricted birds would have eventually developed ascites. At local altitude, only Trt 4 had RV/TV ratios similar to the fully fed controls; Trt 2 and 3 had significantly lower RV/TV ratios when compared with the controls.

Hematocrit is another common measure of ascites development (Burton and Smith, 1967; Maxwell, 1991; Mirsalimi and Julian, 1991; Yersin et al., 1992; Fedde and Wideman, 1996). Figure 5 shows the hematocrit values for birds at 6 wk of age. Again, in the hypobaric chamber, the birds in Trt 3 and 4 had hematocrit values that did not differ from the fully fed controls. Only the completely restricted birds in Trt 2 had significantly lower hematocrits. It appears that for 6 wk the restricted/full feeding treatments (Trt 3 and 4) were experiencing lower ascites incidence levels, but early preascitic changes may predict an increase in the disease if the birds were to remain on full feed. At local altitude, all restricted groups had significantly lower hematocrits when compared with the fully fed controls. The values obtained for hemoglobin and red blood cell counts of these groups corroborated the hematocrit changes as they showed the same significant differences (data not shown).

No significant differences among dietary treatments were found for serum calcium, phosphorus, total protein, and albumin (data not shown). Some significant differences were found for serum glucose and triglycerides (Table 1). Unfortunately, it is impossible to determine if differences are true treatment effects or are due to sample timing. Blood samples were drawn from the birds in the hypobaric chamber about 1 h after the feed trays had been returned to birds in Trt 2. At that point in the study, all of the other groups had feed available 24 h/d; only Trt 2 was still being restricted. Upon return of their feed trays, these birds would typically consume feed very rapidly. Blood samples were not taken from birds in the local altitude chamber until several hours later.

Although the combinations of restricted feeding and full feeding (Trt 3 and 4) resulted in improvements in weight gain and ascites incidence, these groups were adversely affected in terms of breast meat yield. Figure 6 shows the Pectoralis major and minor/BW ratio at 6 wk. In both chambers, the continuously restricted group (Trt 2) had the lowest relative breast meat yield. In the hypobaric chamber, both of the restricted and full feeding groups (Trt 3 and 4) had significantly lower

### Table 1. Effect of feed restriction at local and simulated high altitude on clinical chemistry values of 6-wk-old broilers

<table>
<thead>
<tr>
<th>Treatments</th>
<th>Local (mg/dL)</th>
<th>Hypobaric (mg/dL)</th>
<th>Local (mg/dL)</th>
<th>Hypobaric (mg/dL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>242 ± 7b</td>
<td>253 ± 4b</td>
<td>37 ± 7</td>
<td>41 ± 6b</td>
</tr>
<tr>
<td>Trt 2</td>
<td>229 ± 4b</td>
<td>266 ± 6a</td>
<td>30 ± 4</td>
<td>59 ± 7b</td>
</tr>
<tr>
<td>Trt 3</td>
<td>247 ± 5a</td>
<td>246 ± 7a</td>
<td>36 ± 5</td>
<td>35 ± 7b</td>
</tr>
<tr>
<td>Trt 4</td>
<td>245 ± 3b</td>
<td>250 ± 5b</td>
<td>35 ± 3</td>
<td>39 ± 3b</td>
</tr>
</tbody>
</table>

*Means within a column with no common superscript differ significantly (P ≤ 0.05).

1Main effect means ± SE.

2Control = full feed ad libitum; Trt 2 = feed available for 8 h/d throughout the 6-wk experiment; Trt 3 = feed available for 8 h/d for the first 3 wk and then full feed ad libitum for the remaining 3 wk; Trt 4 = full feed available for the first week, 8 h feed availability for the next 3 wk, and full feed ad libitum for the remaining 2 wk.
relative breast meat when compared with the fully fed controls. The situation in the local altitude chamber was less bleak. Breast meat yield from birds in Trt 3 was not significantly different from the fully fed controls. In general, any restriction, regardless of compensatory weight gain, appears to decrease the breast meat yield.

In summary, when birds are grown at simulated high altitude their BW are significantly lower than their counterparts at local altitude. This decrease in growth, however, coupled with reduced oxygen is not enough to decrease the ascites incidence in these birds. Further restriction of growth through feed restriction is effective in reducing ascites incidence. Both programs that employed restriction along with return to full feeding (Trt 3 and 4) resulted in BW equal to the fully fed controls without the ascites incidence of the controls. Unfortunately, the restricted and fully fed birds (Trt 3 and 4) did not attain breast meat yields equal to the fully fed controls. At necropsy, the restricted and fully fed birds (Trt 3 and 4) raised at high altitude showed equivalent right ventricular hypertrophy to fully fed controls. They also had significantly higher red blood cells counts, hematocrit, and hemoglobin levels than the birds that were restricted the entire experiment (Trt 2). The timing of restriction appears to be important because birds that were returned to full feed exhibited rapid growth and began to develop ascites. It is probable that if these birds had been allowed to continue full feed for several more weeks, their ascites incidence would have approached that of the fully fed controls.

Development of ascites indices was not observed in the birds grown at local elevation. At local elevation, all restricted birds (Trt 2, 3, and 4) had significantly less right ventricular hypertrophy, lower red blood cells counts, hematocrit, and hemoglobin levels when compared with the fully fed controls. Because the incidence of ascites syndrome at local altitude is low, we would not expect to see the development of ascites after release from restriction.

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REFERENCES


