ABSTRACT  

β-Adrenergic agonists have been shown to be capable of improving growth performance in poultry when added to the feed at 1.0 ppm. However, no reference has been made concerning the cardiovascular responses when one of these agents is added to the feed at a lower concentration during the whole production cycle. The aim of this paper was to assess the effects on the ascites syndrome of 0.25 ppm clenbuterol in the feed, throughout 52 d, in broiler chicks. Results showed a lack of difference in growth and feed conversion rate between the untreated control groups and the experimental group. There were differences in mortality due to the ascites syndrome, abdominal fat:body weight ratio, and ventricular index. A statistically significant positive correlation was also found between ventricular index and mortality rate ($r = 0.98$). If adequate withdrawal times are ensured, the use of clenbuterol at 0.25 ppm is suggested to reduce mortality due to the ascites syndrome in broilers.

(Key words: broiler, ascites syndrome, weight gain, mortality, clenbuterol)

INTRODUCTION

Various β-adrenergic agonists have been shown to be capable of improving weight gain when added to the feed of various domestic species (Ricks et al., 1981; Ornelas, 1990; Buyse et al., 1991; Muramatsu et al., 1991; Alpizar et al., 1993; Macri et al., 1993; Takahashi et al., 1993; Malucelli et al., 1994). One of the key features observed when β-adrenergic agonists are added to the feed is a reduction in the proportion of fat in tissue (Brian and Lefkowitz, 1991; Takahashi et al., 1993; Bobowiec et al., 1994), an increase in nitrogen accretion, and a reduction in the saturated fatty acid concentration within the fat associated with muscular fibers (Takahashi et al., 1993; Bobowiec et al., 1994; Foglia et al., 1994; Hamano et al., 1994). It has been suggested that the ascites syndrome is a multifactorial disease with a common triggering factor: reduced cardiovascular reserve (Machorro and Paasch, 1981). It has also been shown that β-adrenergic agonists can increase cardiac performance and regulate the number and bioavailability of β-receptors in target tissue (Brian and Lefkowitz, 1991). However, it is also well known that therapeutic concentrations of bioactive catecholamines induce first an inotropic effect that is gradually reduced with chronic administration (Brian and Lefkowitz, 1991). Yet, minimal quantities of a β-adrenergic agonist may stimulate performance and induce an increment in cardiovascular reserve without the expected reduction in physiological response (Brian and Lefkowitz, 1991; Foglia et al., 1994; Hamano et al., 1994). Based on this hypothesis, it was considered useful to evaluate whether or not the feeding of poultry with clenbuterol-medicated feed throughout the complete fattening cycle could reduce the incidence or severity of the ascites syndrome.

MATERIALS AND METHODS

Three hundred and twenty 1-d-old Arbor-Acres broiler chicks from a commercial hatchery were housed in groups of 40 animals each under controlled standard conditions. The temperature of the room with natural light (13 h) was maintained at 34°C initially, and then reduced by 3°C/wk until it reached 21°C, at which temperature the room was maintained for the rest of the feeding period. Humidity was held at 65%, and bird density at the end of the trial was 27 kg/m². Animals were fed throughout the production cycle as per the recommendations of Cuca et al. (1996) (Table 1): the diet fed from 0 to 3 wk of age contained 22% CP and 3,000 kcal ME/kg, and the second diet, fed from 3 to 7 wk of age, contained 20% CP and 3,020 kcal ME/kg. In this trial, diets were supplemented with 0.25 ppm of clenbuterol (using a clenbuterol premix 0.6%, contain-
ing; clenbuterol hydrochloride and calcium carbonate as excipient) in the experimental group throughout the production cycle (Ricks et al., 1981; Ornelas, 1990; Muramatsu et al., 1991). Chicks were allowed to have free access to a starter diet during the first 3 wk and then to a finisher diet during the second 5 wk (Table 1). They also had free access to water. The vaccination program included infectious bursal disease (three times) and Newcastle, and infectious bronchitis (two times). They also had free access to a starter diet during the first 3 wk and then to a finisher diet during the second 5 wk (Table 1).

The following variables were assessed: weight gain, feed conversion rate, mortality rate, abdominal fat:body weight ratio, and ventricular index, measured according to the following formula: \( VI = RV/LV \); where \( VI \) = ventricular index; \( RV \) = right ventricle; and \( LV \) = left ventricle.

Data were processed using an analysis of variance for weight gain and conversion rate and Man-Whitney “U” test for ascites mortality, abdominal fat:body weight ratio, and ventricular index (Siegel, 1980).

**RESULTS**

Table 2 shows the mean and standard deviation increments in body weight, and conversion rate values at Days 21 and 52. Table 3 summarizes data for mortality due to the ascites syndrome and the abdominal fat:body weight ratio. Table 4 summarizes values for cardiac weight, weight of the right and left ventricles, and ventricular index. There was a statistically significant increase in the weight of the right ventricle in the clenbuterol-treated birds, relative to that of the untreated control group (\( P < 0.05 \)). A larger ventricular index (\( P < 0.08 \)) was obtained for the clenbuterol-treated chicks.

Statistical analysis revealed no significant differences in body weight (\( P > 0.1 \)), ventricular index (\( P > 0.08 \)), or conversion rate (\( P > 0.1 \)). However, mortality due to the ascites syndrome and abdominal fat:body weight ratio differed significantly (\( P < 0.05 \), and \( P < 0.01 \), respectively). Also, a significant negative correlation was found between ventricular index and cardiac weight and mortality rate (\( r = 0.98 \)).

**DISCUSSION**

In contrast with other studies (Muramatsu et al., 1991; Bobowiec et al., 1994) the experimental model used in this trial failed to show a growth-promoting effect with clenbuterol. As in other studies (Macri et al., 1993), this lack of effect may be due to the well-balanced diet used in this trial, and to the comparatively low concentrations of clenbuterol added to the diet. Muramatsu et al. (1991), Ornelas (1990), and Ricks et al. (1981) found a similar lack of effect using the same dose; however no mention was made about the effects of clenbuterol on ascesis.

As in many other studies (Brian and Lefkowitz, 1991), the use of clenbuterol as a feed additive significantly reduced the abdominal fat:body ratio. It appears that there is a universal consensus to ascribe this effect to the lipolytic effects of the \( \beta \)-adrenergic agonists (Bobowiec et al., 1994).

**TABLE 3. Mean and standard deviation data: for mortality due to ascites syndrome, and for abdominal fat:body weight ratio, at 52 d in the control and clenbuterol-treated group**

<table>
<thead>
<tr>
<th>Group</th>
<th>Mortality (%)</th>
<th>Abdominal fat:body weight (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>5.6 ± 1.3</td>
<td>23.58 ± 6.4 (1.37%)</td>
</tr>
<tr>
<td>Treated</td>
<td>1.8 ± 0.7</td>
<td>21.47 ± 5.2 (1.15%)</td>
</tr>
</tbody>
</table>

*\( P < 0.05 \).
TABLE 4. Mean values of total cardiac weight, weights of the right and left ventricle, and ventricular index

<table>
<thead>
<tr>
<th>Group</th>
<th>Cardiac weight (g)</th>
<th>Weight of the right ventricle (g)</th>
<th>Weight of the left ventricle (g)</th>
<th>Ventricular index</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>7.8</td>
<td>1.83*</td>
<td>6.2</td>
<td>0.30</td>
</tr>
<tr>
<td>Clenbuterol-treated</td>
<td>8.82</td>
<td>2.33*</td>
<td>6.6</td>
<td>0.34</td>
</tr>
</tbody>
</table>

*P < 0.05.

Perhaps the most outstanding feature of this trial is the significant reduction in mortality from the ascites syndrome. A computer-assisted search of the corresponding literature showed that no similar observation has been published to date and therefore it is not possible to offer a sound explanation of this effect or to propose a mechanism of action. However, considering the greater values for the ventricular and cardiac indices, it is possible to speculate that these variables may have contributed to the reduced mortality due to ascites. As implied by Wideman et al. (1996, 1997), a more balanced distribution of intraventricular pressures and volumes may be achieved in the clenbuterol-treated animals, which, in turn, allows the birds to withstand the cardiovascular changes ascribed to the ascites syndrome. This proposal is currently being investigated.

These results could benefit the poultry industry if adequate withdrawal times are established and enforced. Malucelli et al. (1994), recommended a 2-wk withdrawal period. Therefore, it will be necessary to repeat this trial on a larger scale allowing the withdrawal period above mentioned to assess the incidence of mortality due to the ascites syndrome, the tissue persistence of residues, and the cost: benefit ratio of adding clenbuterol as described.

REFERENCES


